Handbook of Developmental Psychopathology

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Edited by

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Plenum Press • New York and London

Library of Congress Cataloging-in-Publication Data

Handbook of developmental psychopathology / edited by Michael Lewis and Suzanne M. Miller.
p. cm.
Includes bibliographical references.
ISBN-13: 978-1-4615-7144-5 e-ISBN-13: 978-1-4615-7142-1
DOI: 10.1007/978-1-4615-7142-1
1. Child psychopathology. 2. Child development. I. Lewis, Michael, 1937 Jan. 10- II. Miller Suzanne M.
[DNLM: 1. Child Development Disorders. 2. Mental Disorders--in infancy & childhood. WS 350 H2359]
RJ499.H332 1990
618.92'89--dc20
DNLM/DLC
for Library of Congress 89-23243
CIP

Softcover reprint of the hardcover 1st edition 1990
A Division of Plenum Publishing Corporation
233 Spring Street, New York, N.Y. 10013

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Foreword

Developmental psychopathology is the new child on the block. As yet not an overly sturdy child, but one clearly out of the cradle, an active toddler and an enterprising explorer of the boundaries of its province. It wasn't always so.

Only 15 years ago Thomas Achenbach in publishing the first edition of his book used a recently coined title, *Developmental Psychopathology*, and began the volume with a provoking first sentence: "This is a book about a field that hardly exists yet." Seven years later when the second edition appeared, that sentence had been deleted. In place of the original 13-page chapter, on the "Developmental Approach to Psychopathology in Children," there was a 40-page chapter focused on the biological, cognitive, social-emotional, and educational perspectives in development, together with a lengthy account of developmental periods and an integrative statement on the constituents of a developmental framework.

Other signs and symptoms began to appear. *Child Development*, a doyen for developmentalists, devoted a special issue, under the guest editorship of Dante Cicchetti, to an emergent developmental psychopathology. This year saw the publication of a new journal, *Development and Psychopathology* (1989), edited by Cicchetti and Nurcombe. And attendees at recent meetings of the Society for Research in Child Development can attest to the growing interest of the membership in the linkage of development and psychopathology as seen and heard via posters, symposia, and guest speakers.

Now there comes another milestone, this *Handbook of Developmental Psychopathology*, which readers will quickly identify as a gateway volume to a new interdisciplinary basic and clinical science. The discipline is sufficiently youthful that definitions as to its contents and course may vary even as promulgated by several of the contributing authors to this volume. Some examples:

> . . . a general approach to understanding relations between development and its maladaptive deviations (Achenbach).
>
> Developmental psychopathology is the study of the prediction of the development of maladaptive behaviors and processes (Lewis).
>
> . . . normal development gone awry . . . the search for understanding is conducted at the interface between normal and abnormal development (Wenar).
>
> A developmental perspective on the study of psychopathology takes into account the continuities and discontinuities between normal growth and psychological disorder, age-related changes in modes of adaptation and symptom expression, behavioral reorganizations that occur around salient developmental challenges, internal and external sources of competence and vulnerability, and the effects of development on pathology and of pathology on development (Attie, Brooks-Gunn, and Petersen).

Actually, these and other authors in their chapters provide case and research examples that reflect a commonality of definition not unlike the editorial commentary written by editor Cicchetti on the first page of the first issue of the first volume of *Development and Psychopathology*.

The emphasis is thus placed on the developmental course of disorder, but always in relation to non-disordered behavior as well. There is interest too in the origins of disordered behavior that may not appear clinically until adulthood. Differential diagnoses, treatment, and prognosis, while important, are to be viewed contextually against the background of development. "The developmental psychopathologist," these authors note, "is concerned with the origins and time course of a given disorder, its varying manifestations with development, its precursors and sequelae, and its relation to non-disordered patterns of behavior." This last phrase is particularly important, for as Sroufe and Rutter (1984) earlier noted, developmental psychopathologists are equally interested in children who may evidence precursors of a disordered behavior pattern, yet fail to develop the disorder proper, as well as those who in time manifest the complete pattern of pathology.

This orientation to two groups, those who actualize their risk potential and others who, despite equivalent risk status, escape disorder, accords centrality to the concepts of risk and protective factors, elements which reflect the province and contributions of epidemiology. For Sroufe and Rutter, risk research is in many ways "the paradigmatic developmental psychopathology" with its emphasis on longitudinal study and differentials in the developmental course of at-risk subjects, control, and comparison groups. The significant contrasts are with those who go on to develop the disorder as opposed to those who do not. Those who escape disorder spur the search for so-called "protective" factors that operate to contain risk. Such exemplars can provide a deepened understanding of development, and in time, an empirical basis for primary prevention efforts. Thus the significance of contrasts of clinically disordered children and adults with their more adaptive counterparts lies in providing new insights into variants that reflect individualized biogenetic, familial, environmental, and personality elements. Thus, one can anticipate research programs of the future that will reflect the multiplicity of sciences that contribute to the role of development in psychopathology.

This handbook, in part, demonstrates this hoped-for catholicity of viewpoints. In terms of coverage, the 37 chapters of this volume attend to the major disorders set forth in DSM-III-R under the heading of "Disorders Usually First Evident in Infancy, Childhood, or Adolescence." In many instances the effort has been made (with varying degrees of success) to provide a developmental emphasis in relation to these diverse disorders. But the constraints when these are evident are less author-imposed than they are field-imposed, reflecting the status of a classification system often devoid of a developmental focus. To bolster the effort the editors have wisely organized multiple chapters around a given disorder to provide a needed diversity that typically is absent in psychiatric texts. For example, attention deficit disorder is discussed in chapters by Barkley ("Attention Deficit Disorders: History, Definition, and Diagnosis"), Campbell ("The Socialization and Social Development of Hyperactive Children"), and Deutsch and Kinsbourne ("Genetics and Biochemistry in Attention Deficit Disorder").

Conduct Disorder provides even more diversity, via six chapters that brook across the disorder and provide research reviews on "Behavioral Genetics and Aggressive Behavior in Childhood" (Plomin, Nitz, & Rowe), "The Learning of Aggression" (Perry, Perry, & Boldizar), "The Stability of Aggressive Behavior" (Eron & Huesmann), "Morality and Conduct Disorders" (Smetana), and "The Development of Prosocial Behavior versus Nonprosocial Behavior in Children" (Eisenberg & Miller).

Anxiety disorders are given a similar outreach in chapters on their "Nature and Development" (Miller), "Separation Anxiety" (Crowell & Waters), "Test Anxiety in the School Setting" (Wigfield & Eccles), "Posttraumatic Stress in Children following Natural and Human-Made Trauma" (Davidson & Baum). To this array I would add the

significant chapter by Kagan on the "Temperamental Qualities of Inhibition and Lack of Inhibition," particularly in light of recent accounts of behavioral inhibition in children of parents manifesting panic disorder and agoraphobia (see Rosenbaum *et al.* in the May, 1988 issue of the *Archives of General Psychiatry*).

Depression too is given a broad canvas with superb chapters by Cantwell ("Depression across the Early Life Span"), Miller and Birnbaum ("Etiological Perspectives on Depression in Childhood"), Burke and Puig-Antich ("Psychobiology of Childhood Depression") and Rehm and Carter ("Cognitive Components of Depression").

I hesitate to single out any individual chapters, given the breadth and depth of this excellent volume, but to the reader interested in the efforts to link contexts of development to institutional settings and to treatment potential for behavior disorder in children, I strongly recommend Landesman's chapter "Institutionalization Revisited: Expanding Views on Early and Cumulative Life Experiences," as well as Meyers and Cohen's "Cognitive Behavioral Approaches to Child Psychopathology: Present Status and Future Directions."

In several ways Landesman exemplifies the power of a developmental focus in relation to individual adaptation to institutional experiences and environments. I find a tie to the critical attributes of a developmental psychopathology contained in her commentary on the predictive features of institutions. Landesman emphasizes the *functional characteristics* of the child's care-providing environment, such as the quality and amount of social interaction, the presence of developmentally appropriate activities, and the variety of choices available to the children. These she asserts are more important in terms of consequences for the child than are the usual structural characteristics such as the size of the facility, the ratio of adult staff to children, etc. In this regard I commend to the reader similar observations by Trudy Festinger in her volume on the consequences of foster care for disadvantaged minority youth (Festinger, 1983).

This emphasis on institutional climate has its parallel in renewed efforts to study the impact of school climate on the development of competence in children from disadvantaged environments (see Comer, 1980; Mortimer, Sammons, Stoll, Lewis, & Ecob, 1988; Rutter, Maughan, Mortimer, & Ouston, 1979).

Here the joint power of environmental and individual variation serve as co-influencing factors in the patterns of children's adaptation. Although not an uncommon event it is an understudied one now being redeemed by the efforts of developmental researchers to look both at the "haves" and the "have-nots" under conditions of equivalent disadvantage.

Another chapter in this superb volume that is required reading is Cicchetti and Olsen's "Developmental Psychopathology of Child Maltreatment." If one wishes to see how the developmental sciences can contribute to needed knowledge about the origins of psychopathology, it is contained in this chapter on child abuse. Example after example of developmental research on maltreated children is provided that produces insights into the consequences of such stressful family pathology on the behavioral functioning of victimized children. An added bonus is the authors' listing of 12 recommendations for future research directions.

To these 12 I would add several others:

- Pursuit of the underlying mechanisms for factors identified as "risk" or "protective" in their action
- The search for the substrates of defined competencies in different behavioral domains
- An increased emphasis on the biology and psychology of temperament and their phenotypic transformations over time
- Short-term longitudinal studies of children's adaptation particularly at critical transition points in development
- Rigorous efforts to define more adequately a typology of coping responses, the

systematization of their measurement with particular attention to rigorous psychometric development, and the testing of such instruments for their predictive rather than their postdictive power

I have kept you too long from this substantial volume. To editors and authors go my congratulations for a task well done, one that should move forward the scientific bases of an emergent developmental psychopathology. To the editors go my appreciation for their invitation to write the Foreword to this contributory volume.

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Preface

The study of developmental psychopathology must combine questions about development on the one hand, and maladaptive behaviors on the other. Therefore, we offer, as have others, the definition of developmental psychopathology as *the study of the prediction of development of maladaptive behaviors and processes.*

The thrust of the definition of developmental psychopathology forces something more than a simple combination of two sets of interests. First and foremost, it is the study of change and development in maladaptive behaviors and processes. This goal stands as the basic definition of this domain. Second, however, is a much more important and interactive level of mutual informing. Combining the study of development with the study of psychopathology broadens the scope of each domain in unique and exciting ways. From a developmental perspective, it introduces the problems of change and transformation, and from a psychopathological perspective, it introduces the concept of individual difference, and with it the variety of possible processes and outcomes.

The study of developmental psychopathology begins with the realization that maladaptive behaviors often have an historical cause. Although most broad theories of maladaptive behavior adhere to such a view, it is the psychoanalytic position which most clearly expresses the developmental perspective that disorders in adulthood have their roots in historical events, some of which occur in infancy and some in early childhood. Erikson's model of stages of development offers a viable framework for the factors which might be related to subsequently different forms of psychopathology.

The view that maladaptive developmental factors are the cause of subsequent psychopathology is so widely held that to argue its necessity seems redundant. Nevertheless, there is relatively little study, either in development or in clinical psychology, which touches upon this point. Thus, for example, in the study of development, most researchers tend to emphasize developmental processes and the normal course of growth. While there are studies on the development of pathological conditions, these remain few relative to the large number of normative studies. The literature in clinical psychology, on the other hand, is full of studies of pathological conditions, yet has concerned itself relatively less with explorations of their initial causes or the stability and consistency of these conditions over time.

In part, the study of developmental psychopathology is hampered by at least two major factors. The first is that it is inherently difficult to conduct longitudinal investigations which could serve as a source for the understanding, not only of normal development, but of the development of psychopathology. There are few longitudinal studies currently available. Not surprisingly, therefore, this lack contributes to the dearth of information on the early causes of maladaptive behavior. This is due to the cost of such studies, both in terms of

monetary expenditure and in terms of time an investigator must spend before there is sufficient payoff to facilitate a career.

The second major difficulty confronting the study of developmental psychopathology has to do with the problem of meaning. While we understand, at least in part, what we mean when we talk about a specific pathological condition in adults, we are often at a loss to understand what the same condition might mean for a young child. That is, it is not clear that the meaning of maladaptive behavior is consistent over age.

This problem of consistency of meaning over age is one that has plagued developmental science and psychopathology for some time. A basic tenet of developmental psychology is that the behaviors that underlie a given construct can change as a function of age. This is but one example of the way in which developmental principles can help elucidate critical issues in psychopathology.

At the same time, psychopathology, the study of individual differences, or deviant behavior, can inform developmental theory. Consider, for example, the problems that occur in the study of development when a normative approach is taken. A normative approach tends to force us into thinking that the developmental process has a single path leading to a specific outcome. A clinical perspective allows for a reconsideration of such a view. It draws us into understanding that multiple models of development are possible, where process as well as outcome can be variable.

It should be clear that the importance of the problem of developmental psychopathology necessitates continued investigation and we see this handbook serving as an important reference for those trying to study this domain. This volume should help to educate and inform those scholars interested in development and in problems related to psychopathology. Therefore, the scope of the handbook is extensive, especially since it captures the intellectual excitement and growth of two areas of study and application. The handbook not only addresses the academic community, but those who, during the course of their day, devote their time and energy to the treatment of maladaptive behavior. As articulated in individual chapters as well as in the volume as a whole, the multiple issues of theory and practice are considered. The book is also intended to provide a framework around which undergraduate and graduate courses can be structured.

Only a handbook broad in scope, bringing together experts across a wide area of problems and domains could hope to capture the challenge of the study of developmental psychopathology. We have undertaken this volume, in part, to meet this challenge. We have done so because there exists at the moment no comprehensive and single volume source in which these issues have been addressed. This is surprising, given the large and rapidly growing literature in developmental psychology and psychopathology. We therefore have attempted to fill this gap by including experts in developmental psychology who focus on maladaptive behaviors and processes. Likewise, we have found those scholars and practitioners of the study of psychopathology who are interested in a developmental perspective and have joined them together in this volume. It is, therefore, a unique effort aimed at addressing an important problem in the study and understanding of maladaptive development.

The integration of developmental and clinical psychology is an important step toward greater understanding, prediction, and management of normal and abnormal behavior. Accordingly, the present volume aims to provide a broad and comprehensive perspective on psychopathology in childhood, within a developmental context. It targets conceptual, diagnostic, and treatment issues as they relate to the field at large, and with respect to specific disorders and conditions. While we follow the broad outlines of current taxonomy, as reflected in the Diagnostic and Statistical Manual of Mental Disorders (DSM), we also go beyond the limitations of this classification system.

In the current version (DSM-III-R), and in the ongoing revision, only certain problems are specifically identified as being relevant to childhood and early development. These include various disruptive behaviors (e.g., attention deficit disorder, conduct disorder), various forms of anxiety disorder (e.g., separation anxiety, avoidant disorder), eating

disorders (e.g., anorexia nervosa, bulimia nervosa), gender identity disorders, tic and elimination disorders, mental retardation and autism. All of these problems play a central role in the growing field of developmental psychopathology and receive broad attention here. While these disorders can occur in adulthood, they usually have their origins in infancy, childhood, or adolescence.

The remainder of the diagnostic system includes disorders that have typically been associated with the psychopathology of adulthood. However, many of these problems—such as depression and schizophrenia—are relevant to the nature and course of early development. The current volume provides a focus on these phenomena, and explores the extent to which separate childhood diagnostic categories may be warranted. In future work, it will be exciting to examine if, and how, distinct adult classification diagnoses should be applied to the evolution or late appearance of disorders that usually have their first onset in the beginnings of life.

The book is divided into six major sections. The first section deals with key conceptual and theoretical issues in the study of developmental psychopathology, and provides an overview of the constructs, models, challenges, and cultural factors that are central to the field. Developmental psychopathology is conceptualized as a "macroparadigm," which can coordinate diverse theoretical models (e.g., biological, behavioral, psychoanalytic) and thereby serve to integrate the study of psychopathology from birth to maturity. A model is proposed that argues for the adoption of a "transformational" perspective, which not only emphasizes the way in which the child and environment interact, but also how each is transformed and changed by the other. Methodological strategies for predicting the cause and nature of pathology over time are highlighted. The application of cultural approaches to psychopathology is also considered, which helps alert us to how the culture defines notions of sickness and deviance and, in part, shapes the course and outcome of outlying behaviors. Important diagnostic and classification considerations are also highlighted. In particular, attention is given to the validity and usefulness of the diagnostic categories and to the need to specify the source from which diagnostic information is obtained.

Sections II through V explore the major specific clinical disorders that are relevant to childhood and development. The first section focuses on "undercontrolled" or disruptive disorders, specifically attention deficit disorder and conduct disorder. It begins with a focus on attention deficit disorder, which is one of the most common reasons children are referred for treatment. Conceptually, a model is proposed in which the deficits in sustained attention, impulse control, and poor regulation of activity level are seen as a result of more basic deficits in rule-governed behavior or in the way in which consequences regulate sustained responding. The importance of socialization influences to the etiology of the disorder, as well as of genetic and biomedical contributions, is also reviewed. Several chapters are devoted to conduct disorder and related problems of aggression. A range of issues is considered, including evidence for two main types of aggressive disorder: group (socialized) and solitary (unsocialized). While the role of genetics in the development of aggression is not well established, there is considerable evidence for the role of social cognitive deficits and for the influence of the family, the peer group, and the mass media. Furthermore, there is an impressive stability of aggressive behavior across time and generations. Finally, conduct-disturbed children appear to reason at lower levels of moral maturity than do nondisturbed children, which seems to be an important barrier to the development of prosocial behavior.

Section III deals with "overcontrolled" or anxiety-related conditions. Little research is currently available on the major clinical anxiety disorders that are specifically applied to children, such as separation anxiety, avoidant disorder, and overanxious disorder. The agenda for future research includes the development of multi-method assessment batteries and the broadening of psychological theories. In addition, temperamental characteristics appear to be important in some cases of anxiety disorder. For example, children who have a low threshold for fear and threat tend to react by displaying protective inhibitory behaviors. Peer relations are also central to and predictive of adaptive function in children. Among

various anxiety conditions that are not specifically identified with but are relevant to childhood (e.g., test anxiety, posttraumatic stress, child maltreatment, and fears and phobias) important advances in conceptualization, treatment, and prevention are reviewed.

In Section IV, we present a perspective on depression as it relates to early development, and review its etiology, psychobiology, and cognitive components. It is suggested that the manifestation of depression in childhood must be evaluated in terms of age-appropriate signs and symptoms. Future research is needed to establish criteria and assessment instruments for the diagnosis and subtyping of depressive episodes. Evidence which suggests that exposure to early loss and other adverse familial and personal experiences are risk factors for the development of depression in young children. The biological parameters of childhood depression appear to be more complex, with age and puberty exerting an important influence. Finally, as with adults, a variety of cognitive deficits have been identified in youngsters. However, these need to be considered and explored with respect to the child's stage of cognitive development.

Specific and pervasive disorders are presented in Section V. These range from more generalized conditions (e.g., borderline disorders, schizophrenia, autism, and mental retardation) to more delimited conditions (e.g., eating disorders, elimination disorders, physical disorders, and sexual disorders). Early childhood autism is the most common of the severe and pervasive childhood disorders and appears to be related to underlying biological and physiological disturbances. With respect to borderline disorders, the current literature needs to be supplemented by prospective longitudinal research, studies of high risk subjects, and more sophisticated etiological models. The study and management of mental retardation is particularly challenging, given the serious nature of the disorder and the fact that it is so frequently linked with other severe behavioral and psychological problems.

Among the more specific disorders, eating problems are prevalent and appear to be most likely to emerge in adolescence. This seems to be due, in part, to the convergence of particular physical changes and psychosocial stresses during this period, particularly in the lives of females. With regard to elimination problems, enuresis (bedwetting) is the most common form. Since these problems can be persistent, it is important to develop useful theoretical models and applied treatment modes. A number of other physical disorders are also considered in this section, including tics (which appear to have a genetic component) as well as sleep disorders and stuttering. Lastly, the development of a range of normal and pathological sexual patterns is explored, including masturbation, effeminate behavior, sexual identity, homosexuality, and paraphilias.

Finally, Section VI concludes with a focus on the management of psychopathology, and provides an evaluation of the effects of institutionalization as well as of the efficacy of a variety of psychotherapeutic intervention approaches. The effects of institutionalization on young children are considered first and it is concluded that the negative consequences can be at least partly offset by providing some stability in at least one close human contact. While treatment considerations are tackled throughout the volume with respect to specific disorders, the final chapters provide an overarching appraisal of critical aspects of the therapies themselves, as they are applied to children. Treatment approaches explored include dynamic, family, community and cognitive-behavioral modalities. These techniques need to consider the child in a developmental context in which social, emotional, and environmental influences are highlighted.

In conclusion, the volume provides a broad and in-depth coverage of the impact of developmental influences on clinical disorders in childhood and explores their linkages with subclinical and normal behaviors. For each disorder, its nature, origins, and evolution over the life cycle are explored, paying close attention to the confluence of behavioral, psychological, genetic, and social factors that are involved. As such, the volume presents a state-of-the-field compendium of our current level of understanding of who is most vulnerable to disorder, the factors that predispose toward disorder, and the conditions that activate, exacerbate, or reverse disorder during the beginnings of life.

Michael Lewis
Suzanne M. Miller

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PART I

Issues and Theories of Developmental Psychopathology

CHAPTER 1

Conceptualization of Developmental Psychopathology

Thomas M. Achenbach

In this chapter, I will consider developmental psychopathology as a general approach to understanding relations between development and its maladaptive deviations. This approach highlights the value of viewing psychopathology in relation to the major changes that typically occur across the life cycle. It does not prescribe particular theoretical explanations for disorders but provides a framework for organizing the study of psychopathology around milestones and sequences in physical, cognitive, social-emotional, and educational development. This framework is especially pertinent to maladaptive behavior occurring during the period of rapid development from birth to maturity. It is also pertinent, however, to the less dramatic changes occurring later in life.

A developmental approach to understanding psychopathology does not supplant particular theories but is intended to sharpen our awareness of connections among phenomena that may otherwise seem haphazard and unrelated. This book, for example, deals with a variety of phenomena, theories, and methods that may seem only remotely related to each other. Although they can all be viewed as potentially pertinent to relations between development and psychopathology, none of them by itself epitomizes the concept. Instead, the concept of developmental psychopathology is broader than can be exemplified by any single study, theory, or explanation.

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Why Do We Need Developmental Psychopathology?

Before elaborating on the concept of developmental psychopathology, I must address a prior question: Why is such a concept needed? The primary reason is that the developmental aspects of maladaptive functioning are often obscured by a focus on behavioral/emotional disorders as if they were encapsulated entities that people either have or do not have. This focus had its origins in nineteenth-century efforts to apply biomedical science to the study and treatment of psychopathology. The success of these efforts in differentiating general paresis from other major mental disorders and in identifying syphilitic infection as the cause fostered a medical model for psychopathology. This model, in turn, has shaped the twentieth-century "mental health" system in which "patients" are "treated" by "therapists." Although psychodynamic and behavioral approaches originating in the early twentieth century emphasized the childhood psychological roots of disorders, they did not really document the developmental course of disorders. Instead,

they extrapolated backward from adult disorders to their hypothesized roots in childhood.

The tendency to view maladaptive behavior in terms of encapsulated disorders has been reinforced lately by efforts to make diagnostic criteria more explicit and precise, as in the third edition of the American Psychiatric Association's (1980, 1987) *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III and DSM-III-R). It has also been reinforced by criteria for special school services, such as those required by Public Law 94–142, the Education of All Handicapped Children Act of 1975. These efforts have been prompted by a need for greater specificity in the definition of disorders for purposes of dispensing and funding services. Yet they have been based more on assumptions about children and on administrative exigencies than on direct assessment of children. (For brevity, I will use the term *children* to span the age range from birth to about 20 years.)

As a result of definitions that are imposed by fiat, children are often categorized according to diagnostic concepts or service distinctions that are not congruent with their actual needs. For example, a child may manifest problems that qualify for a diagnosis of Conduct Disorder according to the DSM-III-R (American Psychiatric Association, 1987). Yet the child may also have problems in other areas that are equally serious but do not happen to qualify for a categorical diagnosis. The child would be viewed diagnostically as having a conduct disorder and thus as belonging to a class of children defined by present-versus-absent judgments of the requisite number of items from the DSM-III-R's definitional list.

Similarly, to be entitled to special school services, a child must meet the criteria for an administratively defined category, such as Learning Disabled or Severely Emotionally Disturbed. If the child has problems related to more than one category, as is often the case, it must nevertheless be decided (a) which disorder is primary, and (b) whether the one that is primary meets the criteria of severity for service within that particular category.

The need to fit children into such categories results in haggling over services, shifting of children from one service to another, and biasing of assessment reports to match children to the categories. It also leads to denial of services when children's problems fail to fit neatly into any of the prescribed categories, even when it is clear that the child needs help that could be rendered. We are all familiar with children who, for example, are denied services for the mentally retarded because they are emotionally disturbed but are denied services for the emotionally disturbed because they are mentally retarded.

Developmental Psychopathology as a Conceptual Framework

To improve our understanding of maladaptive behavior, it is helpful to view it in relation to normative sequences and achievements for particular ages. When this is done, it is evident that many behavioral/emotional problems for which professional help is sought are not qualitatively different from those that most individuals display to some degree at some time in their lives. Instead, many problems for which help is sought are quantitative variations on characteristics that may normally be evident at other developmental periods, in less intense degree, in fewer situations, or in ways that do not impair developmental progress. A fundamental challenge, then, is to distinguish those developmental variations that are within the normal range from those that are more ominous, either because they disrupt development or because they reflect pathological processes that bode ill for the future.

No one theory or type of research can meet this challenge alone, because developmental variations and pathological outcomes can involve so many different kinds of variables. Not only is the range of variables enormous, but those that are most relevant and the ways in which they are assessable vary with the individual's developmental level.

Childhood Depression

An illustrative example is childhood depression. From the 1930s through the 1960s, the prevailing psychoanalytic theory held that true depressive disorders were not possible before the superego was fully established in adolescence (see Kashani, Husain, Shekim, Hodges, Cytryn, & McKnew, 1981). In a swing to the opposite extreme during the 1970s, however, childhood depression was widely inferred from a variety of symptoms that were viewed as "depressive equivalents" or manifestations of "masked depression." The symptoms of childhood depression were said to include abdominal pains, headaches, fears, temper tantrums, aggression, changes in school performance, hyperactivity, truancy, running away, sexual promiscuity, and firesetting (Cytryn & McKnew, 1979; Frommer, 1967; Kovacs & Beck,

1977; Weinberg, Rutman, Sullivan, Penick, & Dietz, 1973). From a general denial of depression in children, there was thus a radical turnabout, whereby almost every problem of childhood was interpreted as reflecting a depressive disorder.

Despite the diversity of symptoms attributed to childhood depression, the official nosology held that "the essential features of a major depressive disorder are similar in infants, children, adolescents, and adults" (American Psychiatric Association, DSM-III, 1980, p. 211). The nosology therefore provided no separate categories for childhood depression but merely offered two slight modifications of adult criteria for diagnosing depressive episodes in children under the age of 6. One modification was that dysphoric mood did not have to be reported by the child but could be inferred from a persistently sad facial expression. The second modification was the requirement that three symptoms out of a particular subset of four from an overall list of eight be present, rather than the adult criterion of any four of the eight symptoms.

We can summarize these approaches to childhood depression as follows:

1. The psychoanalytic approach denied the possibility of true depressive disorders in childhood, because of its assumptions about what depression is and the level of personality development that is required before it can occur.
2. When interest in childhood depression blossomed forth in the 1970s, depressive disorders were inferred from a great many different characteristics of children.
3. When the DSM-III addressed the issue, it held that major depressive episodes were essentially similar from infancy through old age and that minor modifications of adult criteria were needed only below the age of 6.

Note that all three approaches were based on *assumptions* about children, rather than on direct assessment of them. Furthermore, the assumptions were generalized across broad age ranges, without much differentiation within those age ranges. Psychoanalytic theory, for example, ruled out depression for all children below the point in adolescence where the superego was assumed to mature. The clinical theorists of the 1970s did not distinguish between ages at which particular symptoms, such as headaches and hyperactivity, were or were not signs of depression. The DSM did not differentiate between features of depression from birth to age 6 or from age 6 to old age. Finally, none of the three approaches assessed representative samples of children to determine the *prevalence* rates for the hypothesized features of depression at particular ages, whether such features actually occur together to form a *syndrome*, or whether the features reflect *disorders* involving impairment.

A Developmental Approach to Childhood Depression

If we address the question of childhood depression from the perspective of developmental psychopathology, what would we do differently? Rather than start from assumptions about depression in children in general, a developmental approach would start by empirically assessing children according to procedures appropriate for their developmental level, using multiple sources of data on their functioning in different contexts. The aim would be to determine what actually characterizes children at each age. Even if we were interested only in depression, we should assess a wide range of characteristics besides those that might be viewed *a priori* as signs of depression. Otherwise, our assumptions about signs of depression might unduly distort findings by preventing us from discovering relations among variables that would be ignored by focusing exclusively on depression. A higher rate of apparent depressive symptoms at age 4 than at age 11, for example, might merely reflect the fact that our assessment procedures reveal more problems of all sorts among 4-year-olds than among 11-year-olds, rather than that depressive disorders are more common at age 4 than at age 11.

To determine what is atypical and potentially pathological at each age, we first need to know what is typical. This involves standardized assessment of large representative samples of children to obtain norms by age, as well as by other demographic variables that may be associated with the target characteristics, such as sex, socioeconomic status (SES), and ethnic group. This *normative-developmental* approach to determining what is typical for children at each age is well established as a basis for judging cognitive performance and academic achievement. It is also applicable to assessing children's behavioral/emotional problems and social competencies on the basis of standardized reports by parents, teachers, and children themselves, as detailed elsewhere (Achenbach & Edelbrock, 1983, 1986, 1987).

Once normative base rates are obtained for each age, comparisons with demographically

matched clinical samples can be used to determine which characteristics actually differentiate children considered to need professional help from typical children of the same age. Empirical comparisons of this sort can yield surprising results, as problems considered to be of clinical significance on the basis of prevailing theories may not actually differ much between clinical and normative samples.

As an example, comparisons between 1300 demographically matched referred and nonreferred children showed little association between referral status and parents' ratings of the item *Fears certain animals, situations, or places, other than school*, despite the prominence given fears in both psychodynamic and behavioral theories (Achenbach & Edelbrock, 1981). Comparisons between the same referred and nonreferred children showed that parents' ratings of the item *Unhappy, sad, or depressed*, however, discriminated between referred and nonreferred children better than any of 117 other problems, although depressed affect was seldom a reason for referral when the data were collected during the mid-1970s.

Beside developmental norms for the rates of specific problems and their association with clinical status, a developmental approach takes account of differences in patterns and syndromes of problems that may occur at different ages or developmental levels. Factor analyses of problems reported for clinically referred children, for example, show that some syndromes are evident only at particular ages, whereas others are generally similar from one age to another but differ with respect to certain details (Achenbach & Edelbrock, 1983, 1986).

Components of the Developmental Study of Psychopathology

Normative-developmental studies of prevalence rates, detection of clinically significant discriminations, and identification of syndromal patterns are cornerstones of a developmental approach to psychopathology. Although cross-sectional research is needed to lay these cornerstones, they are prerequisites for longitudinal research on continuities and discontinuities between problems across developmental periods. To determine whether particular problems, competencies, or other individual difference characteristics have good or poor outcomes, normative baselines are needed to identify the initial characteristics and to compare particular outcomes with those considered normal. Developmental research on psychopathology ultimately aims to detect relations between particular characteristics of particular individuals across extended periods of development, but a strong foundation of cross-sectional knowledge is required to achieve this aim. Furthermore, because longitudinal research is so costly and slow, cross-sectional research is needed to explore many of the developmental aspects of psychopathology.

In my view, the developmental study of psychopathology includes the following components:

1. Normative-developmental research on the prevalence of particular problems in the general population at each age level.
2. Identification of variables that discriminate between individuals who are considered normal for their age and individuals who are considered deviant enough to need special help from mental health or educational professionals.
3. Identification of syndrome patterns of problems requiring special help in each age range.
4. Longitudinal research on relations between characteristics evident in particular developmental periods and the favorable versus unfavorable outcomes of those characteristics. Such longitudinal research includes treatment studies and others in which outcomes are compared following different intervening conditions to test the effects of the intervening conditions.

Relations between Developmental Psychopathology and Other Approaches

The four components listed above are by no means the only types of research applicable to the developmental study of psychopathology. They mainly emphasize the need to look at the actual characteristics of children in relation to their agemates, to avoid prejudging what is pathological versus normal, to identify the patterns of problems actually characterizing children at different ages, and to pinpoint developmental continuities and discontinuities in functioning. Rather than being restricted to particular kinds of research, however, the concept of developmental psychopathology highlights potential relations between different approaches, variables, and levels of analysis. The four components listed in the preceding section are intended to facilitate a convergence of diverse ap-

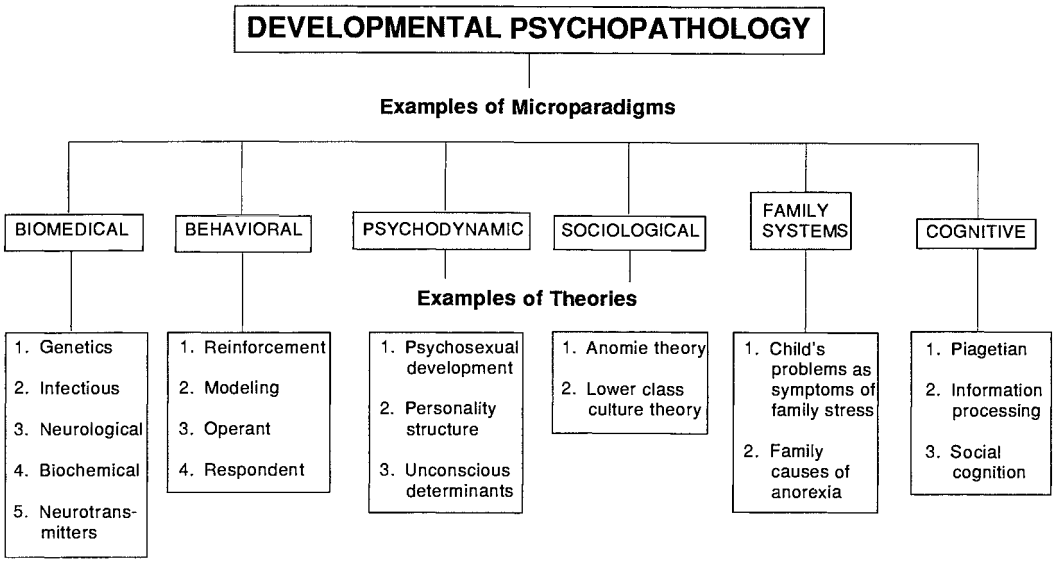

Figure 1. Schematic overview of developmental psychopathology as a macroparadigm in relation to other conceptual levels. From Achenbach (1990).

proaches on a common set of phenotypes but not to replace the other approaches.

If we view developmental psychopathology as a way of integrating different approaches around a common core of phenomena and questions, it can be thought of as a "macroparadigm" within which to coordinate paradigms that deal with particular subsets of variables, methods, and explanations. Figure 1 illustrates relations between developmental psychopathology viewed as a macroparadigm and a variety of relevant paradigms and theories.

The biomedical, behavioral, psychodynamic, and other paradigms are designated in Figure 1 as "microparadigms," because they each deal with only a portion of the phenomena relevant to developmental psychopathology. Most of them cover large domains in their own right, but their contributions to the developmental study of psychopathology can be enhanced by integrating them within a central framework of questions and concepts. Even though researchers inevitably specialize within particular microparadigms and theories, reference to an overarching macroparadigm of developmental psychopathology can also stimulate hypotheses that converge on common issues.

The chapters of this book illustrate both the diversity of topics potentially related to developmental psychopathology and the need for an overarching perspective from which to view the relations between them. Part I focuses on issues that cut across specific disorders, developmental periods, and etiological factors. Part II concerns disorders that involve overt conflicts with standards of decorum and the rights of others. These problems may also have certain underlying characteristics in common, such as low anxiety, lack of internalized social standards, or a weakness in inhibition of impulses. Yet these disorders are apt to have diverse and complex etiologies, as indicated by the chapters dealing with the possible contributions of learning, socialization, genetics, neurochemistry, temperament, abuse, and moral development.

Analogously, Parts III and IV include problems of overinhibition, affective distress, and internal conflict that may share underlying characteristics, such as high anxiety, social introversion, or excessively harsh internalized standards. Here, too, however, the etiologies are apt to be diverse and complex, involving factors such as genetics, temperament, socialization, trauma, and debilitating self-attributions.

Most features of the undercontrolled and the overcontrolled disorders are not intrinsically pathognomonic but are extreme versions of characteristics that many children show at some point in their development. The versions that are extreme enough to warrant professional help may not reflect a specific disease state so much as relatively extreme standing on multiple variables that collectively impair adaptive development.

As an example, a boy whose genetically influenced capacity for attention is at the low end of the

population distribution and whose genetically influenced activity level is at the high end may be at greater risk for being considered hyperactive than a boy with the opposite genotype. Yet, whether the first boy is ever considered deviant at all and, if so, whether his deviance is designated as hyperactivity depend on a host of other factors. If he had exceptionally high cognitive ability, he might achieve so well in school or might work out adaptive strategies that were effective enough that his short attention span and high activity neither stunted his adaptive development nor brought him into conflict with others. Or, if he were socially attractive or had an environment compatible with his attentional and activity levels, he might also adapt effectively.

Alternatively, deviance in attention and activity levels might raise the risk of other types of problems, such as delinquent behavior, if such behavior were reinforced while efforts at more constructive adaptation were not reinforced. Even if genetic risk factors in attentional capacities and activity level were involved, they would thus not necessarily produce a phenotypic disorder that was uniform among all children who had the risk factors, nor would the disorder necessarily remain uniform throughout the course of development. Instead, extremes of attention or activity level might be manifest in different ways during the preschool, elementary school, and adolescent periods.

The presence of particular risk factors can also raise the risk of other problems that are then perceived as primary disorders. There is evidence, for example, that slow perceptual maturation at ages 5 and 6 can interfere with learning to read, resulting in diagnoses of learning disabilities at a later age (Arnold *et al.*, 1977. Yet although the perceptual lags are usually overcome by the age of 8, children who have failed to learn to read at the usual time are subsequently burdened with maladaptive behaviors that then hinder reading, even when the children become perceptually and cognitively able to acquire reading.

Several of the disorders considered in Part V, such as autism, mental retardation, and severe gender identity problems, involve more qualitatively distinctive deviance than most of the problems considered in Parts II to IV. Although mental retardation obviously has a variety of causes—as the other disorders may, too—such disorders usually have more uniform, profound, and pervasive impacts on children's development than most of the problems discussed in Parts II to IV. The range of individual and developmental variations in such disorders is nevertheless enormous, making it essential to view whatever is specific to the disorder in relation to the child's overall developmental progress, the developmental tasks to be accomplished, and the possibilities for maximizing long-term adaptation despite a handicapping condition.

The Relevance of Developmental Periods

Besides emphasizing particular research strategies, the developmental study of psychopathology must take account of existing research and theory on development in general. Although developmental theories often imply stages and levels, these are relatively arbitrary segmentations of sequences that are basically continuous. Nevertheless, massive developmental changes occur in many aspects of functioning that affect children's adaptive possibilities, judgments of normality versus pathology, and the possibilities for research and intervention. It is therefore helpful to have guidelines for the typical sequence of development in multiple spheres of functioning.

Without reifying the stage constructs of any particular theory, it is convenient to summarize the period from birth to maturity in terms of ages at which major changes typically occur. One of the most conspicuous transitional periods occurs between the ages of 18 and 24 months, when children become highly mobile and begin mastering language. According to Piaget's (1983) cognitive theory, the emergence of mental representation underlies the linguistic advances of this period. Whatever the reason for the major advances in language and thought, they not only bring new status and new ways of relating to others, but also the potential for new problems and conflicts. The child's capacity for fantasy, for example, spawns unrealistic fears that cannot yet be tamed by the reality-testing logic of later years. Children's curiousity can also get them into dangerous situations that they cannot cope with. And adult expectations for increasingly socialized behavior create innumerable new conflicts.

Another important transitional period occurs at around the age of 5. According to Piaget's theory, major cognitive advances enable the child to begin applying logical principles to phenomena that were previously grasped only in terms of appearances, figurative representations, and mental associations. At the same time, the onset of formal schooling in most cultures requires children to conform to a social system that emphasizes age-graded

Table 1. A Developmental Overview

Approximate age	Cognitive period	Psychosexual phase	Psychosocial conflict	Normal achievements	Common behavior problems[a]	Clinical disorders
0–2	Sensory-motor	Oral	Basic trust vs. mistrust	Eating, digestion, sleeping, social responsiveness, attachment, motility, sensory-motor organization	Stubbornness, temper, toileting	Organically based dysfunctions, anaclitic depression, autism, failure to thrive
2–5	Preoperational	Anal	Autonomy vs. shame and doubt	Language, toileting, self-care skills, safety rules, self-control, peer relationships	Argues, brags, demands attention, disobedient, jealous, fears,[b] prefers older children, overactive, resists bedtime, shows off, shy,[b] stubborn, talks too much, temper, whines	Speech and hearing problems, phobias, unsocialized behavior
		Phallic-Oedipal	Initiative vs. guilt			
6–11	Concrete operational	Latency	Industry vs. inferiority	Academic skills, school rules, rule-governed games, hobbies, monetary exchange, simple responsibilities	Argues, brags,[c] can't concentrate,[c] self-conscious, shows off, talks too much[b]	Hyperactivity, learning problems, school phobia, aggression, withdrawal
12–20	Formal operational	Genital	Identity vs. role confusion	Relations with opposite sex, vocational preparation, personal identity, separation from family, adult responsibilities	Argues, brags[c]	Anorexia, delinquency, suicide attempts, drug and alcohol abuse, schizophrenia, depression

[a]Problems reported for at least 45% of children in nonclinical samples.
[b]Indicates problem reported for ≥45% of girls only.
[c]Indicates ≥45% of boys only.
Note. From *Developmental Psychopathology* (p. 67) by T. M. Achenbach, 1982, New York: Wiley. Copyright 1982 by J. Wiley & Sons. Reprinted by permission.

acquisition of complex new skills. This opens up great opportunities for developing new modes of adaptation and feelings of competence. Yet it also raises new risks of frustration, maladaptive behaviors, and feelings of incompetence in those who fail to meet the age-graded expectations.

The next important transition is marked most conspicuously by the physical changes of puberty and also by major changes in educational and social status. The relatively protected environment of self-contained classrooms is typically replaced by a more differentiated educational environment requiring greater independence on the part of the child. New privileges and activities offer new opportunities for success and failure. In the cognitive sphere, the advance to formal operational thought not only brings new possibilities for cognitive mastery but also new sources of conflict as adolescents become capable of questioning the values they have been taught and even the meaning of life itself. In addition, behaviors that were relatively harmless at younger ages can now threaten the well-being of others and of the adolescents themselves. Furthermore, the need to prepare for adult roles and the onset of early versions of major adult disorders makes deviance in adolescence seem more ominous than in earlier periods.

Table 1 presents an overview of developmental periods in terms of the major cognitive changes hypothesized by Piaget, psychosexual changes hypothesized by psychodynamic theory, and psychosocial conflicts hypothesized by Erikson (1980). Table 1 also lists the normal achievements of each period, as well as the behavior problems reported most often in general population samples and the clinical disorders that typically become evident at each period.

Applications of Developmental Psychopathology

Many different disciplines are concerned with children's problems in one way or another. Because classroom teachers have to deal with a wide range of problems every day, problems that exceed their expertise may often be referred for more specialized help within the educational system, involving school psychologists and special educators. Pediatricians may be among the first professionals consulted by parents about their children's problems, especially in the early years. Psychologists, psychiatrists, and other mental health professionals are usually sought only when the problems are deemed too serious to be handled by the school or the pediatrician alone. Those that involve antisocial behavior often come to the attention of law-enforcement agencies whose reactions may determine whether the child is viewed primarily as a criminal to be punished or someone needing special help.

The variety of disciplines involved with children's problems and the variety of pertinent circumstances and mandates inevitably create barriers to communication and coordination. Furthermore, within each discipline, there is considerable variation in theoretical orientations and interpretations of particular problems. This makes it difficult to bring research findings to bear on services and to insure that help for each child is based firmly on state-of-the-art knowledge.

If developmental psychopathology is viewed as a macroparadigm within which to integrate the study of psychopathology from birth to maturity, it can also help to integrate practical applications of research knowledge to such activities as training, prevention, services, and the planning of services. Figure 2 illustrates the ways in which these different activities can be viewed in relation to the macroparadigm of developmental psychopathology. As with the research paradigms shown in Figure 1, the activities are not viewed as subordinate to a specific theory of developmental psychopathology but as coordinated around a central set of concepts and cumulative findings on relations between development and its deviations. Thus, for example, in the box labeled "Training," the goals, common core of knowledge, professional models, subject matter, and practical experience for the professions concerned with troubled children would focus on maximizing the awareness of relations between development and its deviations. Similarly, services should strive for standardization of assessment procedures geared to the developmental levels of the children they serve. Services should also strive to coordinate developmentally appropriate procedures for identifying cases, for applying research findings to prevention and intervention, and for evaluating outcome and follow-up status in relation to norms for the children's age.

An Assessment Model for Developmental Psychopathology

Throughout this chapter, I have emphasized the multiplicity of variables and concepts that are

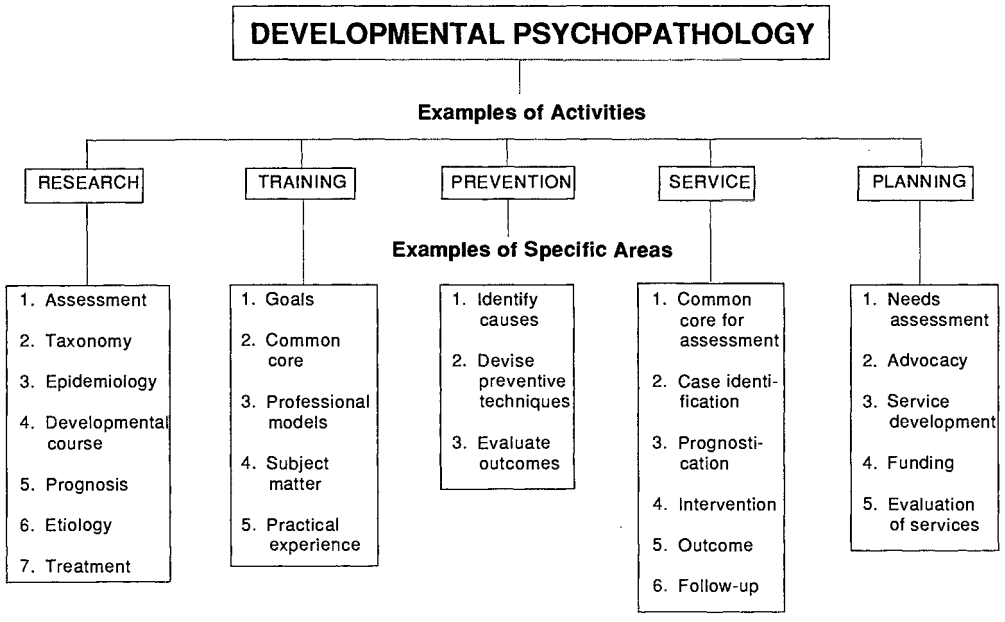

Figure 2. Potential applications of developmental psychopathology. From Achenbach (1990).

relevant to developmental psychopathology. I have also stressed the need for coordination of different activities around a common core of concepts, procedures, and data. Assessment of children's problems and competencies is central to virtually all other activities, including research, training, services, evaluation of outcomes, epidemiology, and planning. It is via assessment procedures that concepts of disorders are operationalized, the problems of individual children are identified, interventions are chosen, and outcomes are judged.

Because children's behavior varies from one situation and interaction partner to another, no single assessment procedure or source of data can provide a comprehensive picture of the child's functioning. Meta-analyses of correlations between reports of children's behavioral/emotional problems thus show only modest agreement between reports by different informants. These correlations average about .60 between similar informants seeing children under generally similar conditions (e.g., pairs of teachers, pairs of parents); .28 between different types of informants seeing children under different conditions (e.g., parents vs. teachers, teachers vs. mental health workers); and .22 between children's self-reports and reports by others, including parents, teachers, and mental health workers (Achenbach, McConaughy, & Howell, 1987).

Although many of the rating instruments showed high reliability, the modest correlations between informants indicate that no single informant can substitute for all the others. It is therefore necessary to obtain data from multiple informants who interact with children under different conditions. Furthermore, it is important to understand assessment as intrinsically multiaxial, aimed at identifying the strengths and weaknesses of children's functioning in each of several important areas. Because the child's functioning may really differ from one area to another, the goal is not to determine which assessment procedure yields a singular truth about the child but to use what each one reveals about needs for help in particular areas. In some cases, multiaxial assessment may reveal that certain interaction partners, such as a parent or teacher, need changing more than the child does. In other cases, multiaxial assessment may show that one type of intervention is needed for one context but a different type is needed for another context.

Table 2 outlines five axes that are relevant to the assessment of most children. Examples of assessment procedures are listed that have promising reliability, validity, and/or normative data, or for

Table 2. A Model of Multiaxial Assessment

Age range	Axis I Parent reports	Axis II Teacher reports	Axis III Cognitive assessment	Axis IV Physical assessment	Axis V Direct assessment of child
0–2	Developmental history; Minnesota Child Development Inventory (Ireton & Thwing, 1974)	—	Bayley (1969) Infant Scales	Height, weight; neurological and medical exam	Observations during developmental testing
2–5	Developmental history; Child Behavior Checklist (Achenbach, 1986; Achenbach & Edelbrock, 1983); Louisville Behavior Checklist (Miller, 1981)	Kohn (1977) Symptom Checklist; Preschool Behavior Checklist (Behar & Stringfield, 1974)	McCarthy (1972) Scales	Height; weight; neurological and medical exam	Observations during play interview
6–11	Developmental history; Child Behavior Checklist; Louisville Behavior Checklist	CBCL-Teacher's Report Form (Achenbach & Edelbrock, 1986); School Behavior Checklist (Miller, 1972)	Achievement tests; Kaufman & Kaufman (1983) Assessment Battery; Koppitz (1975) Bender Gestalt; WISC-R (Wechsler, 1974)	Height; weight; neurological and medical exam	Child Assessment Schedule (Hodges, McKnew, Cytryn, Stern, & Kline, 1982); Semistructured Clinical Interview for Children (Achenbach & McConaughy, 1989)
12–18	Developmental history; Child Behavior Checklist; Louisville Behavior Checklist	CBCL-Teacher's Report Form; School Behavior Checklist	Achievement tests; WISC-R WAIS-R (Wechsler, 1981)	Height; weight; neurological and medical exam	DISC (Costello, Edelbrock, Dulcan, Kalas, & Klaric 1984); Youth Self-Report (Achenbach & Edelbrock, 1987)

Note. Where multiple instruments are available, those with the most promising reliability, validity, and normative data are listed. Adapted from *Assessment and Taxonomy of Child and Adolescent Psychopathology* (p. 173) by T. M. Achenbach, 1985, Newbury Park, CA: Sage. Copyright by Sage. Reprinted by permission.

which such data are potentially obtainable. Not all axes or procedures are equally relevant for all developmental periods. Axis II, Teacher Reports, for example, would be relevant only for children of school age. Similarly, the self-report procedures listed on Axis V, Direct Assessment of the Child, are not usable with infants. At later ages, Axis V procedures vary from play interviews in the preschool period, to semistructured interviews in the elementary school period, and self-ratings in adolescence. Detailed illustrations of multiaxial assessment of a wide variety of problems in children of different ages have been presented elsewhere (Achenbach & McConaughy, 1987; McConaughy & Achenbach, 1988).

The multiaxial model outlined in Table 2 emphasizes the diversity of data relevant to assessing children and the importance of a standardized normative-developmental approach for determining whether and to what degree a child's behavior deviates from that of agemates. This model is applicable to most children who are assessed for purposes of research, services, epidemiology, and planning, regardless of the theoretical predilections of the users. Because Axes I, II, and V can be assessed via standardized procedures for obtaining data from informants, they can be economically applied under many different circumstances and at different ages and different points in contacts with children, such as referral, termination of an intervention, and follow-up. They can thus provide a common descriptive language with which members of different professions can communicate about children's problems, as well as operational procedures for constructing a knowledge base for developmental psychopathology.

Summary and Conclusions

A developmental approach to understanding psychopathology does not supplant particular theories but focuses on connections between development and its deviations. This approach counteracts the tendency to view behavioral/emotional disorders as encapsulated entities existing in all-or-none fashion unrelated to the individual's developmental level.

The developmental study of psychopathology includes the following components: normative-developmental research on the prevalence of particular problems; identification of characteristics that discriminate between individuals who are considered normal and their agemates who need special help from mental health or educational professionals; identification of syndromal patterns; and longitudinal research on the outcomes of particular characteristics.

Developmental psychopathology can function as a macroparadigm within which to coordinate research and theories that deal with particular subsets of variables, methods, and explanations. Different kinds of disorders may require different theoretical models and research strategies, but the range of variation in each disorder makes it essential to view whatever is generic to the disorder in relation to the child's developmental progress, the developmental tasks to be faced, and the possibilities for long-term adaptation.

The developmental study of psychopathology must take account of research and theory on development in general. Major transitions in cognitive, physical, social-emotional, and educational development make it convenient to view relations between development and its deviations in terms of periods from birth to 2 years, 2 to 5 years, 6 to 11 years, and 12 to 20 years.

Developmental psychopathology can help to integrate practical applications of research knowledge to training, prevention, service, and the planning of services. Such activities should be coordinated around central concepts and cumulative findings on relations between development and its deviations. A multiaxial assessment model for developmental psychopathology was outlined in terms of standardized procedures for obtaining data from parents, teachers, cognitive tests, medical exams, and direct assessment of the child. Agreement between different assessment procedures tends to be low, and children's behavior often varies from one situation and interaction partner to another. Consequently, the goal of assessment is not to determine which procedure reveals a singular truth but to use multiple procedures that can identify needs for help in different areas. Standardized normative-developmental assessment can provide a common descriptive language and operational procedures on which to build a knowledge base for developmental psychopathology.

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CHAPTER 2

Models of Developmental Psychopathology

Michael Lewis

Models of development represent world views about human nature and environments that create a human life course (Lewis, 1972; Reese & Overton, 1970; Riegel, 1976). Models of abnormal development also reflect these views, and the data from normal and abnormal lives inform our theories of development. So, for example, the trait notion of personality (Block & Block, 1980) and the invulnerable child (Anthony, 1970; Garmezy, 1974; Rutter, 1981) both share the view that some fixed pattern of behavior may be unaffected by environmental factors. Likewise, information about the regression to old behavior patterns requires that we reconsider the notion that all developmental processes are transformational; that all old behavior patterns are changed into new ones. Clearly, models of development must be applicable for normal and for abnormal development.

Models of development have been considered by many writers, and the interested reader is referred to Reese and Overton (1970). Riegel (1978) developed a scheme for considering models that involve the child and the environment. In this model, each of these elements can be active or passive agents. The passive child–passive environment model is of relatively less interest because it arose from John Locke and David Hume and now receives little attention. In such a model, the environment does not try to affect behavior, and the child is a passive "blank tablet" upon which is received information from the world around it. Such models originally had some use, for example, in our understanding of short-term memory. These memories were likened to a small box that was sequentially filled. When a new memory was entered and there was no more room, the first (or oldest) memory dropped out. Although such a view of memory is no longer held, other views, especially in perception, share many of the features of this model. The notion of affordance of Gibson (1969), for example, suggests such a model because innate features of the child extract the given features of the environment. Such models are by their nature mechanistic.

The second, the passive child with an active environment model, is an environmental control view, because here the environment actively controls, by reward and punishment, the child's behavior. The characteristics of this environment may differ as may the nature of the different reinforcers, but the child's behavior is determined by its environment. We are most familiar with this model in operant conditioning (Skinner, 1953). It is a model much favored by many therapists and is used in diverse areas, such as behavior modification treat-

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ment to alter maladaptive behavior, as well as in theories that explain normal sex role learning by parental or peer reinforcement (Bem, 1987; Fagot & Patterson, 1969).

In the third model, we are confronted with the view of an active person and a passive environment. These models have in common an active child extracting and constructing its world from the material of the environment. Piaget's theory fits well within this framework (Piaget, 1952), although some have argued that Piaget may be a preformationalist (passive child-passive environment) in that all the structures children create are identical (Bellin, 1971). Given the active organismic view of Piaget, it is easy to see that although the child needs the environment to construct knowledge, the environment itself plays little role (Lewis, 1983). Linguistic theorists, such as Chomsky (1957, 1965) and Lennenberg (1967), believe that biological linguistic structures are available for children to use in their construction of language in particular environments. Whether such views are better placed in the passive child–passive environment model can be questioned, although the critical feature of this model should not be lost. In psychopathology and therapy, we often employ such a model when we attempt to help patients alter their behavior (active person) but discount the role of the environment.

The last model is most familiar to developmentalists because of its interactive nature. An active person and an active environment are postulated as creating, modifying, and changing behavior. These interactive models take many forms, varying from the interactional approach of Lewis (Lewis, 1972; Lewis & Feiring, in press) to the transactional models of Sameroff and Chandler (1975). They also include Chess and Thomas (1984) and Lerner's (1984) goodness-of-fit model and from a developmental psychopathology point of view, the notion of vulnerability and risk status (Garmezy, Masten, & Tellegen, 1984; Rutter, 1979). In his attempt to understand cognitive development, Luria (1976) argued that cognitive structures themselves are the consequence of an interaction between the nature of the environment and active child. Such a view of this interaction is often found in research on cultural differences (Cole, Gay, Glick, & Sharp, 1971).

Even though Riegel's (1978) approach is useful, other systems of classification are available. For example, both passive child and passive and active environment models are mechanistic in that either biological givens within the organism or environmental structures outside the organism act on the child. On the other hand, both active child models must be interactive because organisms almost always interact in some way with their environment, which given its structure (whether active or passive) affects the ongoing interaction. In the models of development, as they are related to maladaptive and abnormal behavior, we will use a combination of approaches.

Developmental models of psychopathology have only recently been considered. It is important when trying to understand the etiology of pathology that we utilize the kinds of models of development that have been addressed within developmental research. Three models of development will be articulated: these include a *trait model,* an *environmental model,* and an *interactional model.* Although each of these models has variations, the interactional model is the most variable. Because attachment theory remains central to normal and maladaptive development, it will be used often as an exemplar in our discussion. These three models, which are prototypes of the various views of development, make clear how such models diverge and how they can be used to understand the etiology of psychopathology. Unfortunately, by describing sharp distinctions, we may draw too tight an image and, as such, may make them caricatures. Nevertheless, it is important to consider them in this fashion in order to reach our goal.

Trait or Status Model

The trait or status model is often called the *medical model.* It is characterized by its simplicity and holds to the view that a trait, or the status of the child at one point in time, is likely to predict a trait or status at a later point in time. A trait model is not interactive and does not provide for the effects of the environment. In fact, in the most extreme form, the environment is thought to play no role either in effecting its display or in transforming its characteristics. A particular trait may interact with the environment, but the trait is not changed by that interaction.

Traits are not easily open to transformation and can be processes, coping skills, attributes, or tendencies to respond in certain ways. Traits can be innate features, such as temperament or particular genetic codes, and can also be acquired through learning or through more interactive processes. However, once a trait is acquired, it remains relatively unaffected by subsequent interactions. The trait model is most useful in many instances, for

example, when considering potential genetic or biological causes of subsequent psychopathology. A child who is born with a certain gene or a set of genes is likely to display psychopathology at some later time. This model characterizes some of the research in the genetics of mental illness. Here the environment, or its interaction with the genes, plays little role in the potential outcome. The work of Kallman (1946) on heritability of schizophrenia supports the use of such a model. The same model is useful in the analysis of the lack or presence of certain chemicals on depression (see Puig-Antich, 1982). In each of these cases, the presence of particular features is hypothesized as likely to effect a particular type of pathology. Although a trait model is appealing in its simplicity, there are any number of problems with it; for example, not all people who possess a trait or have a particular status at one point in time are *all* likely to show subsequent psychopathology. That all children of schizophrenic parents do not themselves become schizophrenic or that not all monozygotic twins show concordance vis à vis schizophrenia suggests that other variables need to be considered (Gottesman & Shields, 1982; Kringlon, 1968). We will return to this point again; however, it is important to note that the failure to find a high incidence of schizophrenic children of schizophrenic parents leads to the postulation of such notions as resistance to stress, coping styles, and invulnerability. Each of these terms has a trait-like feature to them.

This model is also useful when considering traits that are not genetically or biologically based. For example, consider the attachment model as proposed by Bowlby (1969) and Ainsworth (1973). Bowlby holds that the child's early relationship with its mother, in the first year of life, will determine the child's adjustment throughout life. The security of attachment that the child shows at the end of the first year of life is the result of the early interaction between the mother and the child. Once the attachment is established, it acts as a trait affecting the child's subsequent behavior. The following is an example of a trait that is established through the interaction of the child with its environment, but, once established, acts like any other trait; that is, it may interact with the environment but is not altered by it.

Figure 1 presents the trait model using the attachment construct. Notice that the interaction of the mother and child at T_1 produces the intra-organism trait, C_{t_1}, in this case, a secure or an insecure attachment. Although attachment is the consequence of an interaction, once established, it is the

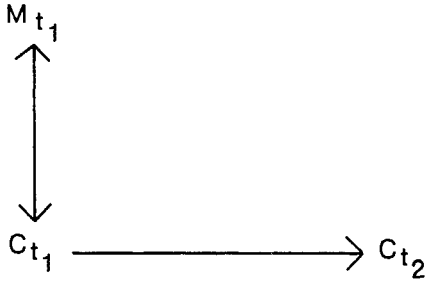

Figure 1. Trait model using the attachment construct.

trait (C_{t_1}) residing in the child that leads to C_{t_2}. There is no need to posit a role of the environment ($M_{t_1} \rightarrow C_{t_2}$) except as it initially produces the attachment. The problems with this attachment model have been addressed by many (Lamb, Thompson, Gardner, & Charnov, 1985; Lewis, Feiring, McGuffog, & Jaskir, 1984; Weinraub, Brooks, & Lewis, 1977); nevertheless, it is a widely held view that the mother–child relationship in the first year of life can affect the child's subsequent social-emotional life as well as impact on its mental health.

Attachment may play a special role in another regard; it may serve as a buffer against stress. Lewis *et al.* (1984) found that securely attached infants were unaffected by subsequent stress. On the other hand, insecurely attached infants who experienced negative environmental factors were more likely to develop psychopathology than were insecurely attached children who did not experience these factors. Although for insecurely attached infants there is an interaction between their status and their environments, none was found for securely attached children. These secure children did not develop psychopathology even though some of them experienced quite stressful environments. Thus, the presence of a secure attachment acts as an invulnerability factor.

In this regard, it is apparent that the concept of invulnerability is similar to a trait model; that is, there are attributes of children which appear to protect them from subsequent environment stress. These attributes (or traits) serve to make the child stress-resistant. Such a mechanism is used to explain why not all at-risk children develop psychopathology (Garmezy *et al.*, 1984; Rutter, 1979). Garmezy (1989) and Rutter (1979) have focused on the factors that can protect the child against stress and, therefore, psychopathology.

Figure 2 presents the invulnerability model

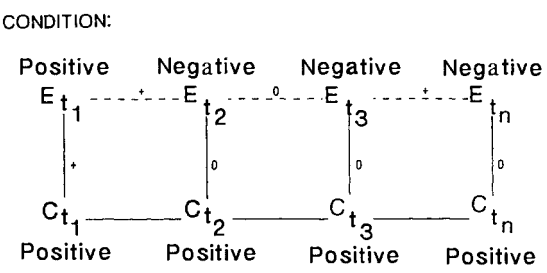

Figure 2. Invulnerability model from point of view of an acquired trait.

from the point of view of an acquired trait, and as such, it reflects an attachment model. Notice that at t_1 the environment is positive so the child acquires a protective attribute. At t_2 the environment becomes negative (stress appears); however, the attribute acquired at t_1 protects the child (the child remains positive). At each additional point in time (t_3, t_4, . . . t_n), the environment may change; however, it has little effect on the child because the intra-organism trait is maintained. Of some question is the prolonged impact of a stress given the protective factor. It is possible to consider such a factor in several ways. In the first place, a protective factor can act to increase the threshold before a stress can affect the child. Stress will have an effect, but it will do so only after a certain level is past. A threshold concept applies not only for intensity but also for duration; that is, invulnerability may represent the ability to sustain one or two stress events, but not prolong stress, or, alternatively, it may protect the child against long-term stress. Specific to secure attachment, we do not know how much stress, over what time frame, is likely to overcome the protective factor of an early positive mother–child relationship.

Certain protective factors may operate more efficiently if they are acquired early in life rather than later. Critical or sensitive periods may exist; for example, a good mother–infant relationship in the first year of life leads to secure attachment and thus a protective factor. However, a good mother–infant relationship in the second year of life may not produce a protective factor or may produce a less efficient factor. These variations suggest that trait theories and models of invulnerability can be quite complex.

Trait models in personality theory are not new (Allport & Allport, 1921), and the problems identified in personality research apply here as well. The major problem related to trait models is the recognition that individual traits are likely to be situation specific (Mischel, 1965). As such, they can only partially characterize the organism. For example, a child may be securely attached to its mother but insecurely attached to its father or its older sibling. It would therefore be hard to characterize the child as insecurely attached simply because it was insecurely attached to one family member but not to the others. Prediction from an insecure attachment trait to subsequent psychopathology would be difficult without knowing the child's total attachment pattern. This would dilute attachment from a trait located within the individual to a set of specific relationships. Thus, to characterize the child in a simple way, such as secure or insecure, may miss the complex nature of traits, especially those likely to be related to subsequent psychopathology.

Equally problematic with the trait notion is the fact that such models leave little room for the impact of environment on subsequent developmental growth or dysfunction. There is no reason to believe that environments do not play a role in children's development past the opening years of life.

The Environmental Model

The prototypic environmental model holds that exogenous factors influence development the most. In the simplest model, behavior, normal or maladaptive, is primarily a function of the environmental forces acting on the organism at any point in time. In such a model, for example, a child does behavior x but not behavior y, because this behavior is positively rewarded by his parents and y is punished. Notice that, in this model, the environmental forces act continuously on the organism, and the behavior emitted is a direct function of this action. Although this model may apply for some behavior, it is more likely the case that environmental forces act on the child, directly at that point in time and indirectly at later points in time. Our hypothetical child may do behavior x, not because of the immediate reward value, but because the child remembers that x is a rewarded behavior. Clearly, much of our behavior is controlled by this indirect form of environmental pressure. Many other forms of indirect reward and punishment have been observed. For example, consider the situation in which a child is present when the mother scolds the older sibling for writing on the walls of the house. The younger child, although not directly punished, does learn that writing on walls is not an action to be

performed. These indirect forms of learning are beginning to receive attention (Lewis & Feiring, 1981).

There are many different types of environmental pressures. For example, we see an advertisement for a product "which will make other people love us." We purchase such a product in the hopes that others will indeed love us. We can be rewarded or punished in many direct and indirect ways; however, it is important to note that the more the organism has to construct the nature or purpose of the environmental forces, the more we move from the passive child-active environment to the active child-active environment model.

Our knowledge of the features of the environment and their various effects are poorly understood. Although we have focused on the taxonomy of organism behaviors, little attention has been paid to situations (see Clarke-Stewart, 1974; Fredricksen, 1972; Lewis, 1978; Parke & O'Leary, 1975). Many years ago, Barker (1965) argued for a greater understanding of the nature of environments, but our knowledge still remains sparse. If we wish to focus on the effects of environments, then more effort is needed to understand their structure.

Because other people make up one important aspect of our environment, the work on the structure of the *social* environment is particularly relevant. From a developmental perspective, some work exists on this topic, and an attempt has been made to expand the numbers of potentially important people in the child's environment (Lewis, 1984; Lewis & Rosenblum, 1975) as well as to create an analysis of the structure of the social environment itself (Lewis, 1987b). Although considerable effort has been focused on the importance of the mother on the child, other persons also play a significant role, even from birth (Lewis, 1984). Fathers (Lamb, 1980), siblings (Dunn & Kendrick, 1982), grandparents (Tinsley & Parke, 1984), and peers (Lewis & Rosenblum, 1975) clearly have importance in shaping the child's life even within the first years of life. The increasing role of peers has been observed (Hartup, 1979), and we have substantial evidence to suggest that once children enter school, peers rather than adults become increasingly influential in their lives.

Given these diverse features of environments and the important roles attributed to them, it is surprising that so little systematic work has gone into their study. For the most part, mothers and families have received the most attention, and we will therefore use them in our examples.

The role of environments in the developmental process has been underplayed because most investigators seek to find the structure and change within the organism itself. Likewise in the study of psychopathology, even though we recognize that environments can cause disturbance and abnormal behavior, we prefer to treat the person—to increase their coping skills or to alter specific behaviors—rather than change the environment. Yet we can imagine the difficulties that are raised when we attempt to alter specific maladaptive behaviors in environments in which such behaviors are adaptive—a point well taken by Szasz (1961). In terms of specific behaviors, like sexual dysfunction, it is possible that changing the environment (as well as the patient) might alleviate the problem. Such a suggestion and the use of surrogate sexual partners was raised by Masters, Johnson, and Kolodny (1977) and created considerable upset.

Our belief that the thrust of development resides in the organism rather than in the environment, in large part, raises many problems. At cultural levels, we assume that violence (and its cure) must be met in the individual—a trait model—rather than in the structure of the environment. The murder rate using handguns in the United States is many times higher than in any other Western society. We seek responsibility in the nature of the individual (e.g., XYY males, or the genetics of antisocial behavior), when the alternative of environmental structure is available. In this case, murders may be due more to the culture's nonpunishment or nonrestriction of handguns. The solution to the high murder rate in the United States might be the elimination, through punishment, for the possession of weapons. Thus, we either conclude that Americans are by their nature more violent than Europeans, or that other Western societies do not allow handguns and therefore have lower murder rates.

Many examples of environmental control of developmental change can be observed. In one school district in New Jersey, for example, children were moved from the primary school to the middle school at the end of the fifth grade. Fifth graders were "seniors" in their school and, as such, were more social than the others; for example, they dated and went to parties. When the district altered the policy and sent the fifth graders to secondary school, the fourth graders became the "seniors" and, as a result, their social behavior changed, becoming more like the fifth graders. Such a case study reveals that social behavior in schools can be

influenced by the perception that one is the oldest and therefore should behave in a particular fashion quite independent of age, the individual trait variable most associated with development.

A general environmental model suggests that children's behavior is a function of the environment in which the behavior occurs. As long as the environment appears consistent, the child's behavior will be consistent; if the environment changes, so too will the child's behavior. If a more active organism model is used, it is still the case that maladaptive environments produce abnormal behavior; however, the abnormal behavior is produced by the child's perception and construction of its reality. From a developmental psychopathology point of view, maladaptive behavior is caused by maladaptive environments; if we change those environments, we alter the behavior.

Figure 3 presents this model. The environment at t_1, t_2, and t_3 all impact on the child's behavior at each point in time. The child's behavior at C_{t_1}, C_{t_2}, and C_{t_3}appears consistent, and it is as long as E remains consistent. In other words, the continuity in C is an epiphenomenon of the continuity of E across time. Likewise, the lack of consistency in C reflects the lack of consistency in the environment. The child's behavior changes over t_1 to t_3 as the environment produces change. Even though it appears that C is consistent, it is so because E is consistent. Consistency and change in C are supported by exogenous rather than by endogenous factors.

Such a model of change as a function of the environment can be readily tested but rarely is it done. This failure reflects the bias of the trait model. Consider the case of the attachment model. Although it is recognized that the environment affects the attachment at t_1, the child's status or trait at t_1 (C_{t_1}) is hypothesized to determine the child's other outcomes, C_{t_2}, C_{t_3}, and so forth. Rarely is the environment, and the consistency of the environment, factored into the model as a possible cause of subsequent child behavior. Consider that it is poor parenting which produces an insecure child at C_{t_1} and this parenting remains poor at t_2, t_3. For example, a nonresponsive mother at t_1 also is a mother that gives her child no peer contact at t_2 or does not teach her child school-related tasks at t_3. The attachment model, as an example of a trait model, would have us understand that the secure attachment at t_1 alone leads to poor peer relations at t_2 and poor school performance at t_3. However, without removing the continuing effects of poor parenting, it is not possible to make such a conclusion. That most research in this area fails in this regard constitutes evidence for the lack of interest in the environmental model.

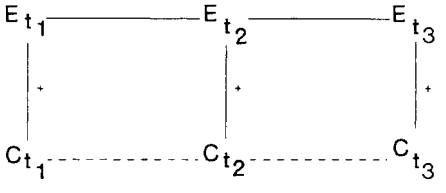

Figure 3. Model of change as a function of the environment.

Lewis and Feiring (in press) explored this problem longitudinally, observing children from 3 months to 6 years. They reported that attachment at 1 year and the family environment at 6 years equally predict psychopathology at 6 years. Moreover, attachment at 1 year (a measure of the environment as well as the child) also predicts the family environment at 6 years. Logically, it cannot be concluded that the characteristics of the child at 1 year are responsible for subsequent maladaptive behavior unless the environmental effects are removed. When they are, the trait effects become less important. Such proof could be gathered from other developmental studies, and it might be possible to assign a more appropriate distribution of effects to environments as well as to child characteristics or traits.

Other forms of maladaptive behavior development have a similar problem. Depressed women are assumed to cause concurrent as well as subsequent depression in children (Zahn-Waxler, Cummings, McKnew, & Radke-Yarrow, 1984). What is not considered is the fact that depressed mothers at t_1 are also likely to be depressed at t_2 or t_3. What role does the mother's depression at these points play in the child's subsequent condition? We can only infer the answer given the limited data available. The question that needs to be asked is: What would happen to the child if the mother was depressed at t_1 but was not depressed at t_2, t_3? This type of question suggests that one way to observe the effect of the environment on the child's subsequent behavior is to observe those situations in which the environment changes.

The environmental change can occur in two ways: a positive environment can become negative or a negative environment can become positive. In each case, the change in the child's behavior should inform us as to the role of the environment in affecting behavior. In the former case, we would expect

an increase in the child's maladaptive behavior, whereas in the latter, we would expect to see a decrease. There are several studies that can be of help in answering this question. Thompson, Lamb, and Estes (1982), for example, examined children's attachment between 1 year and 18 months. They found that the change in the child was related to the mother's going back to work. When the child's environment changed by going from less to more stress, there was an increase in the negative behavior of the child. When there was no change in the stress environment, there was little change in the children's behavior. Unfortunately, there were no cases of the change from high to low stress, but a study by Lewis and Schaeffer (1981) bears on this issue.

Children who are abused are found to be insecurely attached (Schneider-Rosen & Cicchetti, 1984) and also have poor peer relationship. The trait model states that it is the insecure attachment that produces the subsequent poor peer relationship. Alternatively, an environmental model would state that abusive parents are also likely not to encourage or promote good peer relationship; thus, both insecure attachment and poor peer relationships are due to poor parenting at both points in time. Moreover, if peer relationships could be encouraged by placing these children in supervised day care, then peer relationships should improve even though the attachment characteristic did not change. Such findings would support an environmental model. Lewis and Schaeffer (1981) reported that although initial peer behavior in these abused children was different than in a nonabused group, after one month the behavior of the two groups could not be distinguished. Even so, the child–mother relationship remained poor. Such findings as these require that concurrent environmental influences be given more attention.

Although the environmental model can be made more complex, this model suggests, in all cases, that the child's concurrent health status is determined by the environment. Should the environment change, then the child's status will change. To the degree to which the environment remains consistent, and in our case psychopathogenic, is the degree to which psychopathology will be consistently found within the subject. Therefore, the environmental model is characterized by the view that holds that the constraints, changes, and consistencies in children's psychopathology rests not so much with intrinsic structures located in the child as in the nature, structure, and environment of the child.

Degree of Prior Experience

The environmental model also raises the issue of the nature and degree of prior experience; that is, the notion of a critical period. Certain environmental influences may have a greater effect at some points in time, but not others. For example, a responsive environment in the first year and a less-responsive environment in the second year should lead to better consequences than a nonresponsive environment in the first and a responsive environment in the second year. Although critical periods suggest some organismic characteristics, the effects of the environment as a function of past experience remains relevant here. In its simplest form, it is important to know that if a series of positive events is followed by a negative event, does the impact of the negative event depend on the number or the timing of the preceding positive ones? In similar fashion, the same question applies for a series of negative events.

In Figure 4, a model relating to past experience is suggested. In Case A, there are four positive environmental events prior to the negative one, whereas in Case B there are only two. All other variables being equal, does the negative event for Case B produce more of a negative event than for Case A? The simplest environmental model would suggest no difference because such models argue for a passive child and, given such, past experiences have little effect. On the other hand, memory systems are likely to be ones in which past experiences are registered and summed. Given this fact, the four positive past experiences for Case A might dilute the effects of the negative event. A more complex model provides for a more active child, and here the child's memory of all the past positive events allows for a reconstruction of the negative one. The effect of the past events might serve to buffer the effect of the next event.

Besides the effects of past experiences on the behavior of the child, particular time periods may be critical for some environmental events (Bornstein, 1987). For example, a limited number of negative events in early life may have a greater impact than the same number of events later in life. Attachment theory suggests that the failure of the child to securely attach in the first year may predispose this child to serious maladaptive behavior, even though the environment thereafter is altered in the positive direction. The issue of successful adoption after this critical period may be difficult if such a model is correct. Although the data are controversial (Singer, Brodzinsky, Ramsay, Steir, & Waters, 1985),

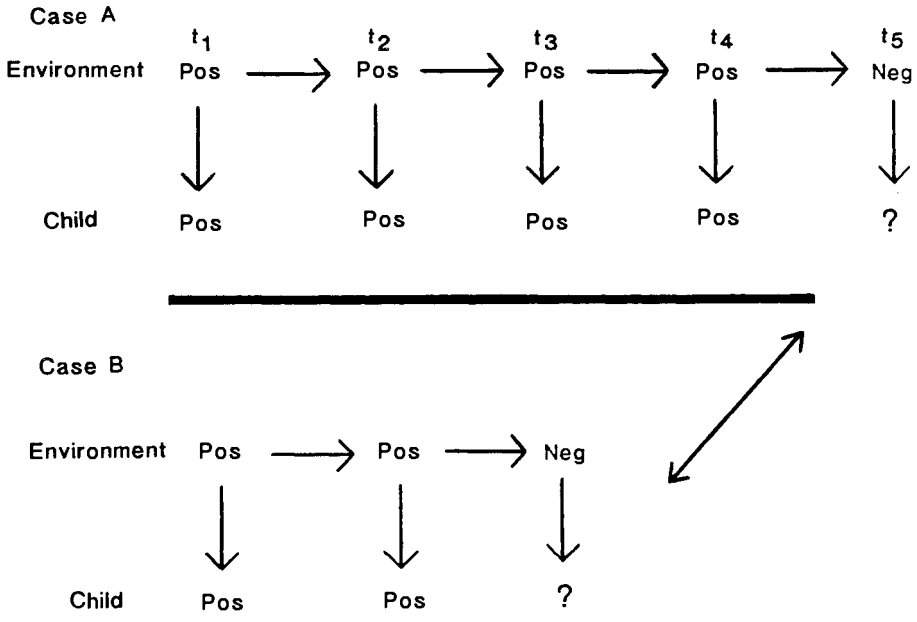

Figure 4. Model of degree of prior experience.

there is some reason to suspect that this may be the case. The models of the effect of past experience and of critical period are in need of serious testing if we are to hold to the importance of an environmental model of developmental psychopathology.

Whatever model we choose, it is clear that the study and treatment of maladaptive behavior require that the environment be considered. Although some maladaptive behavior of the child may be altered within the therapeutic situation, the child usually returns to the same environment in which these maladaptive behaviors were formed. If such behavior is to be modified, we have to modify the environment. Strong environmental models suggest that in some cases this may be sufficient.

The Interactional Model

Interactional models vary; some researchers prefer to call them "interactional" and others "transactional" (Lewis, 1972; Sameroff & Chandler, 1975). As we shall see, all these models have in common the role of both child and environment in determining the course of development. In these models, the nature of the environment and the characteristics or traits of the child are needed to explain concurrent as well as subsequent behavior and adjustment. Such models usually require an active child and an active environment; however, they need not be so. What they do require is the notion that behavior is shaped by its adaptive ability and that this ability is related to environments. Maladaptive behavior may be misnamed because the behavior may be adaptive to a maladaptive environment. The stability and change in the child need to be viewed as a function of both factors, and, as such, the task of any interactive model is to draw us to the study of both features. In our attachment example, the infant who is securely attached, as a function of the responsive environment in the first year, will show competence at a later age as a function of the earlier events as well as the nature of the environment at that later age.

One of the central issues of the developmental theories that are interactive in nature is the question of transformation. Two models of concurrent behavior as a function of traits and environment can be drawn. In the first, both trait and environment interact and produce a new set of behaviors. However, neither the traits nor the environment is altered by the interaction. From a developmental perspective, this is an additive model because new behaviors are derived from old behaviors and their interaction with the environment, but these new behaviors are added to the repertoire of the set of old

behaviors. For example, an insecurely attached child ($-ATT$) can interact with a positive environment ($+E$) so that a positive outcome ($+O$) occurs:

$$(-ATT) \times (+E) \rightarrow +O$$

In this case the trait of ($-ATT$) remains unaffected by the interaction and ($+O$) is added to the set of behaviors including ($-ATT$). Likewise ($+E$) is not altered by the interaction. As we have found, this model is very useful for explaining such diverse phenomena as regression, vulnerability, and goodness of fit.

In the second model, both trait and environment interact producing a new set of behaviors which transform themselves. From a developmental perspective, this is a transformational model because the old behaviors give rise to new behaviors and the environment itself is altered by the exchange. In our example, the insecure attached child ($-ATT$) interacts with the positive environment ($+E$) which results in a positive attribute ($+ATT$) that leads to positive outcome ($+O$):

$$(-ATT) \times (+E) \rightarrow (+ATT) \rightarrow (+O)$$

In this case, the trait of ($-ATT$) is altered, and in like fashion $+E$ might be altered as in the case $(-ATT) \times (+E) \rightarrow (-E) \rightarrow (-O)$. In fact, both might be possible:

$$(-ATT) \times (+E) \rightarrow [+ATT, -E] \rightarrow \text{outcome}$$

and the result would be unclear, depending on the strength of change in each feature. Nevertheless, a transformation of structure has taken place.

The variety of interactional models is considerable. We will focus on two of them in order to demonstrate their importance for our theories of developmental psychopathology.

Goodness-of-Fit Model

The goodness-of-fit model was proposed by Thomas and Chess (1977) with regard to individual differences in children's temperament and more recently by Lerner (1984). The major feature of this model is that discord arises when the child's characteristics do not match the environmental demand, or, stated in another way, the environmental demand does not match the child's characteristic. Notice that maladjustment is the consequence of the *mismatch*. It is not located in the nature of the child's characteristic nor in the environmental demand. Because of this, such a model can be accused of "relativism." Some researchers would argue that certain environmental demands, by their nature, will cause pathology in the same way that certain child characteristics, by their nature, will cause them. Although this may be the case in extremes, the goodness-of-fit model suggests that psychopathology is the consequence of the mismatch between trait and environment, and, as such, it is an interactive model.

Consider the case of the temperamentally active child. If such a child is raised in a household where activity and noise are valued—where there is a match between the active child and the environment—no maladaptive behavior results. However, if this same child is raised in a household where quiet behavior and inhibition are valued, we would expect to see more adjustment problems. Similarly, for the quiet lethargic child, again dependent on the match between the behavior and the environment, different degrees of maladjustment would occur.

In terms of transformation, such a model is relatively silent. Even so, it would seem reasonable to imagine that new behaviors arise due either to the match or mismatch, but these new behaviors do not require the old behaviors to be transformed. The active child may learn to move more slowly, but the trait of activity is not lost or transferred. The environment, too, may change, because less is required of the child, but the values or goals underlying the requirement remain and are not changed.

In exploring the role of early sex role behavior in children and maternal attributes about sex role on subsequent adjustment, a goodness-of-fit model appeared to best explain the data (Lewis, 1987a) and Figure 5.

Sex role behavior at 2 years was observed in terms of how much the children played with male and female toys. There were large individual differences; some boys played more with boy toys than girl toys and some boys played more with girl toys than boy toys. The same was true for the girls. Mothers were given the Bem Scales (1987), and we were able to determine their sex role orientation. Some mothers were traditional, whereas others were more androgenous in their sex role beliefs. We found that school adjustment, as rated by the teacher, was neither dependent on the mother's belief or the child's sex role play. Rather adjustment was dependent upon the goodness of fit between the child's play and the mother's belief. For example, boys showed subsequently better adjustment if their mothers were androgenous in belief and they played equally with boy and girl toys as well as if mothers

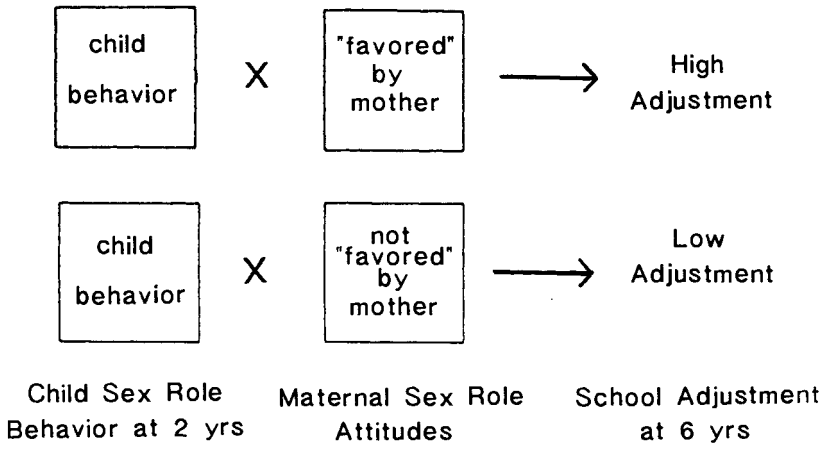

Figure 5. Goodness-of-fit model explaining the role of early sex-role behavior and maternal attributes about sex role on subsequent adjustment.

were traditional and the boys played more with boy than girl toys. Adjustment at 6 years was worse if there was no fit; for example, the mothers were traditional and the boys were androgenous or if the mothers were androgenous and the boys were more male toy oriented. The same was true for the girls. The goodness of fit between individuals and their environments rather than the nature of the child's behavior itself may be more important for the development of maladaptive behavior. One therapeutic solution, then, is to alter the maladaptive behavior of the individual; the other is to alter the nature of the fit. Matching children by their characteristics to teachers' traits reduces educational mismatch and increases academic achievement.

The nontransformational feature of the goodness-of-fit model is particularly relevant for the development of psychopathology in two ways: the phenomenon of regression and the vulnerable child. Regression is a problem for any interactional model in which old behaviors are transformed and become new behaviors (Piaget, 1952). If old behaviors are transformed, they should disappear from the child's repertoire and should be unavailable for use once the new behaviors appear. This should be the case for the growth of intellectual or social behaviors. Nevertheless, it is clear that regression is a common occurrence in all domains and, as such, challenges the transformational model. It is not possible to use old behaviors if they were transformed. That regression appears requires that old behaviors do not disappear but are retained when new behaviors develop. New behaviors may have a greater likelihood of being elicited; however, old behaviors will occur, especially under stress.

The vulnerable child is another example of the usefulness of a nontransformational interactive model. A vulnerable child possesses some characteristics that places it at-risk. If the environment is positive, the at-risk features are not expressed and the child appears to be adjusted. Over repeated exposure to the positive environment, the child appears adjusted; however, if given an instant or two of a negative environment, the child will appear maladjusted, showing abnormal behavior. It is obvious from this example that the positive environmental experiences were unable to transform the at-risk features that remained independent of their interaction with the environment. If the at-risk features remain independent of the environment and are displayed as positive or negative adjustment only as the environment changes, then a goodness-of-fit model, rather than a transformational model, best explains the data. It is possible that at-risk features are influenced by the environment such that repeated positive exposures make the response to a negative event less severe—a type of threshold view. Under such conditions, we approach a transformational model.

Transformational Model

The last model to be described often is captured in the contrast with earlier models and so a discussion of its unique features can be redundant. We shall try to avoid this here by our brevity. These types of models, which require that all features that make up an interaction are themselves comprised of all features and are transformed by their interaction, are called *transactional models* (Sameroff, 1975).

For example, if we believe that the child's characteristics at C_{t_1} interact with the environment E_{t_1} to produce a transformed C_{t_2} and E_{t_2}, then it is likely that C_{t_1} and E_{t_1} also were transformed from some earlier time $t_{(n-1)}$ and that therefore each feature is never independent of the other. The general expression of this, then is:

$$(C_{t_1} \times (E_{t_1}) \rightarrow C_{t_2}, E_{t_2}$$

where

$$C_{t_1} = \int(C_{tn-1} \times E_{tn-1})$$

and

$$E_{t_1} = \int(E_{tn-1} \times C_{tn-1})$$

Such models reject the idea that child or environment characteristics are ever independent or exist as "pure" forms; there is here an ultimate regression of effects. Moreover, from a future perspective of development, these features interact and transform themselves at each point in development. The linear functions that characterize the other models are inadequate for the transformational view. The parent's behavior affects the child's behavior; however, the parent's behavior was affected by the child's earlier behavior.

Lewis and Feiring (1989), for example, have found that intrusive mothers of three-month-olds are likely to have insecurely attached children at 1 year. However, their overstimulation appears to be related to their children's behavior. Children who appear to be not socially oriented at 3 months prefer to play with and look at toys rather than people, and are insecurely attached. These children have mothers who are overstimulating. Any of the models we have suggested fit these data; however, we suspect a transformational interactive model will best explain the findings.

Insecure attachment at 1 year can be transformed given the proper environment. In like fashion, an insecure attachment can transform a positive environment into a negative one. Consider the irritable child who interacts with a positive environment and produces a negative environment that subsequently produces a negative irritable child. The causal chain does not simply pass in a continuous fashion either through the environment or through the irritable child as a trait or environmental model would have it. In fact, it is a circular pattern of child causes affecting the environment and the environmental causes affecting the child. Such models have intrinsic appeal, but by their nature are difficult to test. Nonlinearity requires a mathematics that still eludes us. Moreover, it is difficult not to treat a child or an environmental characteristic as a "pure" quantity even though we might know better. As such, we tend to test the interactive models that require less transformation.

As we have indicated earlier, other less extreme transformational models are possible. Transformational models rest on principles related to critical periods, threshold levels, and past experiences. Nevertheless, these models are transformational in nature and belong in any discussion of these types of effects.

Summary

As we can now see, the distinction between the various models becomes more dim as our characterization of them moves from their extreme to their less-extreme forms. The sharp characterization was intended to make clear the differences and underline implicit assumptions that exist. Whatever their final form, a combination of models most likely will be needed to explain the complex forms of maladjustment. The articulation of normal developmental models continues and should be helpful in understanding developmental psychopathology. In much the same fashion, the interest in the development of maladjustment should provide important data for model building. The interface is an interactive one, perhaps even transformational.

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CHAPTER 3

Challenges to the Study of Developmental Psychopathology

Michael Lewis

Developmental psychopathology can be defined in a variety of ways, all having to do with development on the one hand and the resulting set of maladaptive behaviors on the other. We, too, define developmental psychopathology as focusing on and integrating these two traditional areas; however, a third feature needs to be added, namely, maladaptive processes as well as behaviors. Thus, developmental psychopathology is the study of the development of maladaptive behaviors (Sroufe & Rutter, 1984) and also the study of maladaptive processes themselves. Underlying the study of developmental psychopathology is the principle of predictability. Because of the research of Kohlberg, LaCrosse, and Ricks (1972), the prediction of maladaptive behavior has been viewed not only as possible but as an important feature in the study of developmental psychopathology. With this added feature, we now have a more complete definition: *developmental psychopathology is the study of the prediction of the development of maladaptive behaviors and processes.*

If prediction, development, and maladaptive behavior make up the important features of our inquiry, it is necessary that we consider each of them in turn. In this chapter, the focus will be upon the feature of prediction, although any inquiry into this topic naturally involves the others. Developmental models are presented in another chapter (see Lewis, Chapter 2 in this volume). There are at least four challenges to the issue of prediction that will be considered here: (1) prospective versus retrospective studies, (2) the nature of maladaptive behavior, (3) diagnostic screening and evaluation, and (4) catastrophe. By focusing on these topics, we intend to show that the search for prediction in developmental psychopathology may be a difficult challenge and perhaps not necessary in the study of maladaptive behavior.

Prospective and Retrospective Prediction

The differences between prospective and retrospective analysis are obvious and their advantages and disadvantages are well known. Even so, the error of base rate, a likely consequence of retrospective analysis, often escapes us (Tversky & Kahneman, 1974). Base-rate errors are very common in retrospective studies, so it is necessary that we clarify its meaning. One common example is the case of dreams. An individual dreams every night but on one occasion the dream happens to correspond to daytime activities. If we use this informa-

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tion to say that dreams come true, we commit a base-rate error because we do not ask how many times do we dream (the denominator) and how many times does the dream come true (the nominator). This percentage is very low, certainly not better than chance. However, if we focus only on positive instances, we have at least one example of its occurrence. If a second instance is experienced, the occurrences increase by a factor of two even though the ratio remains quite low.

The gambler's fallacy, that is, the belief that a run of the same side in the coin toss increases the likelihood of the alternate side turning up, also is a base-rate problem. There are many such examples of this type of error, and we must be alert to its occurrence even within scientific study. Errors of base rate are more likely to occur when retrospective analyses are conducted. A good example of this type of error can be seen in the study of child abuse. One of the strongest beliefs (or facts) known about child abuse is that if the mother was abused as a child, the mother is likely to abuse her child. Unfortunately, this finding is derived from a retrospective analysis. Let us consider how this finding was achieved. A group of children are identified as being abused and the mothers of these children are asked if they were abused as children. When the problem is studied in this fashion, it is reported that the great majority of mothers state that they were abused as children. From this evidence, we conclude that if the parent was abused as a child, the parent is likely to abuse. Such a conclusion, though perhaps correct, cannot logically be assumed given this method of study.

Consider Figure 1a in which the data on abuse are tabulated. Note that, on the top of the figure, there are two headings, "Abused Child" and "Not Abused Child." Since we have located abused children, we have data for this column. For this group of children, we ask the mothers if they were abused. (It is a retrospective analysis.) Let us assume that 70% replied they were abused and 30% claim they were not abused. What we know is that, of the abused children, 70% have mothers who were abused. From this analysis it is concluded that being abused is related to abusing one's child.

What is important to remember here is that there are no data available for those children who are not abused. We do not know for this column of nonabused children how many mothers were or were not abused. The percentage of abusing mothers who were abused needs to be compared to the percentage of nonabusing mothers who were abused. This is the appropriate base rate. Unfortunately, this rate is missing and limits the conclusions to be drawn.

	ABUSED CHILD	NOT ABUSED CHILD
ABUSED MOTHER		
NOT ABUSED MOTHER		

Figure 1a. Child abuse outcome by maternal history of abuse.

A retrospective analysis has caused this problem because such an analysis is predicated on the following assumptions: given the condition (that you abuse your child or that you are depressed or maladaptive in some fashion), what were the conditions that came before? The problem in prediction is that from a developmental point of view, all we have are children who have presented themselves as a clinical problem. Retrospective studies can be dangerous because of the likelihood of this error. It is necessary to know how many people were abused as children but do not abuse their children, and this sample is not readily available in a clinical setting. Such a sample is possible to obtain and, in part, would alleviate our problems.

Another example of base-rate error and prospective and retrospective analysis pertains to data collected prospectively, that is, longitudinally. Recently, we reported on a developmental study having to do with attachment differences in the first year and psychopathology in the sixth year of life (Lewis, Feiring, McGuffog, & Jaskir, 1984). Figure 1b indicates that, at 1 year, 38 children were rated as securely attached, whereas 20 were rated as insecurely attached. At 6 years, 10 children were rated as having psychopathology, whereas 48 had no pathology.

Retrospectively, we ask the question, Given that there are ten children who have psychopathology at 6 years of age, how many of them or what percentage of them were insecurely attached at 1 year of age? Given the data, it can be concluded that 80% of the children with psychopathology were insecurely attached at 1 year of age and 20% were securely attached. Prospectively, we ask the question, What percentage of insecurely attached children at 1 year developed psychopathology? The base rate, when asked in this fashion, is 20 not 10, and now it can be concluded that only 40% of the insecurely attached children develop psychopathology. Forty percent is significantly higher

	Pathology at Six	No Pathology at Six	
Insecurely Attached at One	8	12	20
Securely Attached at One	2	36	38
	10	48	

Figure 1b. Pathology at 6 years as determined by attachment differences at 1 year.

than 6%, so that attachment is related to subsequent psychopathology, but the percentage is not 80%. In fact, a prospective analysis reveals that 60% of insecurely attached children at 1 year do not develop psychopathology.

Prospective analysis avoids the problems of base rate and is the analysis of choice. Why then do we not use such procedures more often? The problem has to do with the location of maladaptive behavior or psychopathology. In the Lewis *et al.* study (1984), the rate of psychopathology in these children was 17% (10 out of 58). If the rate was 1%, we would have found one child with pathology, hardly enough to study in terms of statistical procedures. Prospective studies require that we have very large sample sizes in order to identify large numbers of subjects with later psychopathology. The cost of research for such large sample sizes precludes this approach. Alternatively, a unique sample could be identified, one in which the incidence of subsequent psychopathology is higher. A number of developmental studies have tried this procedure by studying the children of parents with a psychopathological dysfunction. Interestingly, this has not resulted in sufficient numbers of subjects since Garmezy and others have found that these "at risk" children, for the most part, do not develop psychopathology (Garmezy & Tellegen, 1984). These findings suggest that prospective analysis, though the preferred technique, will be costly to implement. Prediction as a causal statement requires that prospective analysis be conducted. Retrospective analysis is possible, but only given that samples are matched appropriately.

Nature of Maladaptive Behavior

The topic addressed here is definitional. We have seen that the study of developmental psychopathology requires that prospective analysis be undertaken. Given the relatively low base rate of most clinical disorders, very large samples need to be collected. Select subjects, who are at high risk for a disorder, can be used, but the likelihood of obtaining a high rate of disorder, though increased, does not give us very large numbers of subjects. Moreover, the selection of unique samples of high-risk children has its own problem. For example, the selection of a large schizophrenia sample for a study of development requires the examination of schizophrenic mothers (Garmezy, 1983; Sameroff & Seifer, 1981). We know that the numbers of children showing early disorders, but not schizophrenia, are relatively lower than would be expected (Garmezy, Masten, & Tellegen, 1984). Parenthetically, this fact results in our interest in stress resistance and the issue of invulnerability (Garmezy, 1981, 1989). Even when enough children are located, there are serious problems in logic when sampling procedures of this kind are employed.

When we look at unique samples and find that children are different from a normal, not-at-risk group, we are apt to conclude that the at-risk status itself is the cause of the children's differences. Although this may be the case, we cannot logically conclude this to be true. For example, when we compare Down syndrome children to normal children in terms of a particular function (e.g., attentional processes), differences in function appear (Lewis & Brooks-Gunn, 1982). From such findings, one might conclude that the differences are due to the Down syndrome. When we compare attentional differences between other dysfunctional groups and Down syndrome children, we find that there is no difference between these different dysfunctional children, and all of them are different from the normal child. The difference, therefore, is not due to Down syndrome *per se,* but, as it turns

out, to lower mental age; any child, regardless of the group label, with a low mental age will perform in the same way (Lewis & Brooks-Gunn, 1984).

The unique selection of high-risk samples, although increasing the likelihood of finding more developmental disorders, does not allow us to conclude whether the unique risk status is causal to the subsequent maladaptive behavior. Such difficulties are likely to occur whenever we observe children at high risk because their parents are clinically ill. Zahn-Waxler, Cummings, McKnew, and Radke-Yarrow (1984) observed children with manic-depressive parents. They found that the children with bipolar parents show heightened distress and preoccupation with the conflicts and suffering of others. The question remains as to whether these findings are specific to the manic-depressive disorders of the parents, which cannot be tested without another group of high-risk subjects. It seems reasonable that whatever specific illness their parents have, the children of these parents may be more likely to be preoccupied with these problems. Thus, the relationship between a parent's manic-depression and the children's behavior, established through this technique, must remain tentative.

The selection of specific high-risk groups, in order to increase the likelihood of a large subsequent set of children with illness, does not work as well as we would wish. Given this problem, and our need to study prospectively developmental psychopathology, alternatives are necessary. We need to increase the number of usable subjects for study, and to do this, we may need to reconsider our definition of disorder.

There have always been two broad classes of disorders in psychopathology, those labeled a *neurosis* and those labeled a *psychosis*. Although the definition of these classes can be made on the basis of many factors, we need to employ a classification system that can help our sampling problems. One class of general disorders are those that you have or do not have; that is, they have a two-state status. One cannot be a little psychotic; either you are or you are not psychotic. Such a system appears to move us away from a yes/no system. The psychotic kinds of disorders are the easiest to classify, because they are the most obvious. Being schizophrenic is an example of this class of illness. The second set of disorders differs from the first because this class does not have a two-state status; that is, one can have degrees of a disorder. It would seem that depression is an exemplar of this second class.

Depression may be part of a continuum that varies along the dimension of unhappy. This continuum starts with happy and then moves toward unhappy, dysphoric, and, finally, depression. Depression may be defined in a yes/no fashion by using the DSM-III-R classification (American Psychiatric Association, 1987), but until more is known about the disorder, it may be reasonable to think of it as part of a continuum of unhappiness. Our classification systems, like those of the DSM-III-R, make this approach difficult because we often use a yes/no criterion.

In the first class, the two-state status disorders are by their nature infrequent. We are going to find very low base rates for these illnesses because most children will not be ill (a no rather than a yes status). However, the second class, those illnesses that belong to the continuum status, potentially have large numbers of subjects that can be studied. Such a class of disorders will allow prospective studies. Retrospective studies are needed because, in part, we cannot find enough yes responses in the yes/no status disorders.

Let us consider how a view of the yes/no versus continuum classification might work in a prospective study. In a recent study of childhood depression, we were interested in the developmental features, specifically the role of attachment, stress, and social network size (number of friends), on subsequent depression. One hundred and fifty subjects were followed prospectively from birth to 9 years of age. They were a normal, non-at-risk sample. First, consider the yes/no classification. Of the 150 subjects, 17 appeared to be depressed using the DSM-III-R classification. Such a small number of subjects followed prospectively was insufficient to study statistically because our 133 versus 17 distribution made analysis difficult. In order to cope with this difficulty, we used a continuum classification system. We divided the 133 children into three additional groups: those who were not unhappy, those who were unhappy, and those who were dysphoric. We actually had a fourth group who had some depressive symptomology but who were not dysphoric. These groups, including the depressive group, could be studied from the point of view of looking at levels of stress or numbers of peers, in order to observe whether levels of happiness/unhappiness/depression were related to the dependent variables. Our results support such a type of analytic approach (Horwitz, 1988).

More importantly, this approach of looking at levels or degrees of particular disorders should be useful for other problems; for example, acting out

that we focus on disorders most likely to fit into the second class. Moreover, it may be useful to consider other illnesses from this perspective. Clearly, some disorders cannot easily be defined in this manner; nevertheless, the agreement about borderline disorders suggests that this approach may have general usefulness (Kernberg, 1967). Our focus on prediction in developmental psychopathology requires that we move from the yes/no definition to a definition utilizing a continuum of disorder.

Diagnostics, Screening, and Evaluation

Developmental psychopathology presents us with a unique problem related to diagnostics. For adults, the processes of diagnostics involve them directly, because it is the adults themselves who seek out the therapist. For children, however, these processes involve the children themselves but only indirectly, because it is the adults, usually their parents but at times their teachers, who seek out the therapist for them. This difference in diagnostics and in the process of screening and evaluation involves us in the problem of the criteria of disorder and therefore of prediction.

As an adult, we seek out a psychotherapist because we have decided that we have a problem. When adults appear at psychotherapists' offices and say they have a problem, they are not asked if their mothers think they have a problem. If, however, the patient is 9-years-old, the child comes to the office because the parents believe their child has a problem.This difference in who brings the prospective patient to the office is an issue of development. When the patient is an adult, it is the adult who decides whether or not there is a problem. But when the patient is a child, it is usually the parents or the teachers who decide the child has a problem, because it is they who bring the child to the therapist. The contact with therapy is dependent upon the parents' believing that their child has a problem.

The second difference is the parents' attitudes toward their children's problems. This difference gives rise to two problems. The first is related to how another person, even a parent, knows the internal and therefore unavailable life of the child. We have found that, in studying depression, parents are unaware of those symptoms that were not behaviorally obvious; for example, parents were unaware of their children's death thoughts (Horwitz, 1988). Parental report of children's problems must suffer from some of these difficulties. Even though parents are concerned about their children's mental health and want them to get help if they have a problem, they are also ashamed or wish to deny that their children have a problem. After all, it is a parent's problem that the child has a problem. Thus, the use of the parent as the reporter of the child's psychopathology presents differences from adult psychopathology. This developmental issue has to affect classification and diagnostic accuracy as well as the practice of therapy and the criteria against which we can judge whether the outcome of development has led to psychopathology. For example, should we use the child's report or the maternal report of psychopathology as the outcome measure of developmental processes?

Figure 2a presents this model of classification. For adults, two possibilities arise: they do or do not think they have a problem (yes or no). The therapist can say yes and no for both cases of the adult report. When the adult says "Yes, I have a problem," the therapist can concur or not. We assume that the therapist concurs over 95% of the time, in part, because if an adult goes to a therapist he or she probably needs to be there. Moreover, the therapist, looking for psychopathology, is likely to find some in this condition. Then the bias, if any, is to find pathology when none exists. The distribution of 95% to 5% yes/no for the therapist, given the patient's yes, is likely. This problem is somewhat unique because, in medicine, the percentage of concurrences between clinician and patient is considerably less (Gagnor & Deloger, 1982). The patient yes and the therapist yes group, although containing some bias, is assumed to be a more pure group, that is, this group is not likely to contain many false positives.

Consider now the case in which the adult patient thinks there is no disorder. What percentage of these adults would be classified as having a problem if they were seen by a therapist? To answer this, we have to have an estimate of the number of people in the total population who have problems. Estimates vary, but there is some indication that 20% of the population suffer from some serious disorders, especially if we include alcoholism. Of course, we do not have a good estimate for the adult no group because they do not appear for therapy. Nevertheless, those who do not think they have any psychopathology, and if the therapist also does not think they have any (the no/no group), should be another group that does not contain any false postures. These four groups constitute differences in terms of developmental outcomes; that is, different events may lead to each of these four outcomes.

Adult Respondent

Self-Report		Self-Report	
Yes Pathology		No Pathology	
Therapist Report		Therapist Report	
Yes	No	Yes	No

Parent Respondent

Pathology in Child				No Pathology in Child			
Yes				No			
Child Report				Child Report			
Yes		No		Yes		No	
Therapist Report				Therapist Report			
Yes	No	Yes	No	Yes	No	Yes	No

Figure 2. (a) Adult model of classification. (b) Parental model of classification.

Given the model for adults, let us now turn to the case of the child (Figure 2b). In real life, the first division is usually made by the adult who decides whether or not the child has a problem. Parents, therefore, determine whether or not their children have a problem. At the next level, the child's opinion generates four groups. The value for each of these four cells is determined by the amount of agreement between parents and child. The data on question of agreement are limited but some exist for the agreement relative to depression. In general, the level of agreement is rather weak (Brody & Forehand, 1986; Carlson & Garber, 1986; Chambers, Puig-Antich, Hirsch, Paez, Ambrosini, Tabrizzi, & Davies, 1985; Friedlander, Weiss, & Traylor, 1986; Herjanic & Reich, 1982; Kaslow, Rehm, Pollack, & Siegel, 1983; Kazdin, 1981; Kazdin, French, Unis, Esveldt-Dawson, & Sherick, 1983, Reynolds, Anderson, & Bartell, 1985; Schaughency & Lahey, 1985; Smucker, Smucker, Craighead, Craighead, & Green, 1986; Stavrakaki, Vargo, Roberts, & Boodoosingh, 1987; Weissman, Orvaschel, & Padian, 1980). This being the case, it is not clear which person (the child or the parent) should constitute the criteria measures of outcome. Perhaps these outcomes each have different developmental patterns. In that case, there are 2 child, 2 parent, and 4 parent/child outcomes. From a developmental perspective, each of these outcomes may be related to a different set of antecedent conditions and, in turn, may be related to a different set of outcomes at some later point in time. It is not unreasonable to believe that these different beliefs about the child's psychopathology are likely to have different outcomes. For example, children who believe they have a problem are likely to have a different outcome from children who do not believe they have a problem, even when both their parents believe the same thing; it is likewise for parents who do or do not believe their children have a problem regardless of what the child may think. Such analyses lead us to believe that outcomes can vary as a function of the nature of the respondent and that different outcomes are not necessarily correct or incorrect. Rather, the task is to relate or predict these differences from the set of earlier events.

Up to this point, the adult and the child respondent models are quite different. This difference is complicated still further at the next level. Consider now the therapist who also can say yes or no to

each of the four possible cells (see Figure 2b). The eight cells that are now created each have different problems. When the mother and the child agree (yes/yes and no/no cells), the therapist is likely to agree with them; however, the therapist's decision for the other six cells is not so clear. For example, is the therapist more likely to agree with the parent or with the child and does this vary with the presence or absence of reported psychopathology? Let us consider the various cells. When the parent says yes and the child no, the therapist is likely to say yes rather than no because, even if the child does not have psychopathology, there is a problem because the parent believes there is a problem.

When the parent says no but the child says yes, the therapist is not likely to see the child, so the data are difficult to construct. Even so, especially among young children, the therapist is more likely to agree with the parent. However, given the general bias in diagnosing psychopathology even if there is none, the therapist's yes cell is not going to be zero.

This model allows us to examine the relation between parent and child report, as well as between parent and therapist, child and therapist, and parent, child, and therapist cells. What is important to consider is that the complexity of the problem is a direct function of the age of the patient. The level of complexity is itself a developmental problem. It is also important to realize that each of these cells, four in the case of the adult and eight in the case of the child, may have different sets of antecedent conditions and different outcomes. Rather than focusing on which cells are more "correct," the focus should be on the differential predictive factors leading to these outcomes and how these different cells predict subsequent developmental psychopathology. Given the low levels of agreement on psychopathology that exist, this is especially true. Minimal data on this topic are available, so it is necessary that we again look at our longitudinal study.

In this study of 125 children, followed from birth to 9 years, we obtained a variety of information concerning depression at 9 years. Mothers filled out the Child Behavior Profile (Achenbach & Edelbrock, 1979) and answered a set of questions about their children, whereas the children filled out the Children's Depression Inventory (CDI) (Kovacs, 1983) and answered a set of questions about themselves, including a Q-Sort (Block & Block, 1980) and a self-questionnaire. Teachers filled out the Classroom Behavior Inventory, which is a teacher behavior checklist (Schaefer, Edgerton, & Aaronson, 1979). From these various sources, it was possible to derive a dysphoric score for each child, using the mother, the teacher, and the children themselves as respondents. These data allowed us to consider several of the questions concerning agreement between respondents.

Moreover, in order to see if different sets of antecedent events differentially predicted these different outcomes, data were available concerning the children's attachment at 1 year (a laboratory-based measure called the "Strange Situation," see Ainsworth, Blehar, Waters, & Wall, 1978), the amount of stress in the children's lives (Life Stress Questionnaire), and the size of the children's social network.

Figure 3 presents the data that are relevant to this question. A total of 28 mothers (22%) reported that their children are dysphoric. Of these children, five or 18% of the children agreed. Of the 78% of the mothers who reported their children are not dysphoric, seven or 6% of the children disagreed. Although 22% of the mothers reported their children

Mother's Response

Yes Pathology (28)				No Pathology (97)			
Child Report				Child Report			
Yes (5)		No (23)		Yes (7)		No (90)	
Teacher Report		Teacher Report		Teacher Report		Teacher Report	
Yes (2)	No (3)	Yes (8)	No (15)	Yes (0)	No (7)	Yes (26)	No (64)

Figure 3. Rating of dysphoria of mothers, children, and their teachers.

to be dysphoric, only 12 or 10% of the children reported themselves so. One can see, on the one hand, that the agreement between parent and child for this classification is rather low; on the other hand, there is relatively high agreement on who is not dysphoric. As is often the case with clinical problems, there are more absolute cases of children reporting dysphoria in the group of mothers not reporting dysphoria than in the group of mothers who do report this disorder (7 vs. 5 subjects). Such findings have general implications for screening problems regardless of the disorder that is investigated. Although screening by the mother increases the number of cases of child report of dysphoria (22% vs. 6%), there are more cases of children reporting yes in the mother no group. Paradoxically, screening does increase the percentage of the children who have the dysfunction; in absolute numbers, there are always more in the dysfunctional group that get through the screening procedure. This problem in screening can be reduced by relaxing the criteria of dysfunction. However by doing this, we increase the numbers in the yes cell and thereby reduce the effectiveness of the screening.

The final cells in Figure 3 are based on the teacher's ratings. Although the model we presented involved the therapist's evaluation, such data are not available in this study. At least, the use of the teacher's report allowed us to observe an independent judgment from those obtained within the family. We found teachers to be accurate judges of children's personality, particularly in the school setting (Lewis & Feiring, in press) as have others (Schaefer *et al.*, 1979). The teachers were unaware of the mother's or the child's report. When the mother reported dysphoria, ten teachers (36%) also reported dysphoria. On the other hand, 26 teachers reported dysphoria when the mothers reported none. Thus, teacher and mother agreement is only moderate, the most agreement occurring when both report none (66%). Teachers' and children's agreement is less for the child's yes report (17%), but more for the child's no report (70%). When mother and child agreed, the teacher agreed 50% of the time and 40% when both said yes. When they disagreed, the teacher usually agreed with the mothers (86%) somewhat more than with the children (75%), this being most true for the case in which the mother reported no dysphoria while the child reported some.

These data provide the opportunity to consider the problem of criteria measures of outcome vis à vis maladaptive behavior. Results from this study make clear that the developmental issues are complex and that multiple outcomes are possible. To begin with, in the study of developmental psychopathology, the diagnosis of the outcome measure is different depending on the child's age. Even though adults or even adolescents themselves report their maladaptive behavior—either directly as in such questionnaires as the CDI (Kovacs, 1983)—with young children the process is radically different because it usually involves an adult (parent) in the process of the report. This can be a problem as parents may not be privy to all the internal processes, thoughts, and feelings of the child. For example, in our studies of depression, we found that although more mothers than children reported dysphoria, many more children reported symptoms that are associated with depression than did the mothers. In part, the difference in reporting is based on symptoms not externally manifest. In terms of classifying depression, children reported more than parents because the DSM-III-R requires both dysphoria and symptomology to make the classification.

Once the possibility of different outcome measures as a function of the reporter (mother, father, child, and teacher) is considered, the problem of accuracy and selection of outcome becomes an issue. Reporter as well as situational differences give rise to the problem of which observation is correct and which should be used as criteria. For example, we can choose therapists as criteria because they are trained. Although this is true, therapist agreement is not high, and many people who have maladaptive behavior are not seen by anyone trained to make a judgment of their maladaptive behavior. Parents of young children can be chosen because they live with the child and presumably know their children. However, they are often not privy to the internal processes of the child and, moreover, may identify their children's illness. Children know about themselves but they, too, may either wish to deny their internal states, be influenced by a specific context, or be unable to articulate their problems. Teachers interact with many children and so have the best actuarial data, but their information may be limited to specific situations. One way to determine which judgment may be more accurate is to compare them to some outcome. Is the child's judgment or the teacher's, or the mother's, or some combination of them more likely to be related to subsequent maladjustive behavior? This is an empirical question.

Although we have no data on subsequent outcomes, we do know, from our longitudinal study, that different antecedent conditions affect the dif-

ferent reports. The mother's report of dysphoria is best predicted from insecure attachment and high stress, whereas the teacher's report of dysphoria is best predicted from high stress and a small social network. But the children's report is not related to any earlier events (Horwitz, 1988). This analysis suggests that there may not be one "correct" judgment and that multiple measures of maladaptive behavior are likely and are needed. Moreover, such differences support the belief that there are multiple outcomes to the developmental process and that these outcomes have different antecedent conditions.

Catastrophe and the Notion of Predictability

Predictability in the study of developmental psychopathology constitutes an important aspect of our definition (Santostefano, 1978; Sroufe & Rutter, 1984). Such a focus on prediction as a central feature is understandable because the origins of maladaptive behavior require an understanding of continuity and change. Even so, it is surprising that such a focus is required. Freud (1920/1955) doubted the ability of prediction. In truth, he appeared to be cognizant of the fact that retrospective prediction was much easier than prospective prediction (see also Freeman, 1984). His belief about the complexity involved in the development of maladaptive as well as normal behavior made him skeptical about the ability to predict outcome.

The question arises then as to whether prediction is necessary for the study of developmental psychopathology. Clearly, Piaget (1952) was able to articulate an elaborate theory of cognitive development without the need for prediction. That theory on maladaptive development is possible in the absence of predictability or in the absence of high predictability should not be questioned. For example, Garmezy and Rutter (1983) have generated important principles concerning vulnerability, risk, and resistance from data on the low base rates of disorder from high-risk subjects. Such findings as the lack of a predictable outcome should not cause despair. Finally, Darwinian theory is not dependent on prediction (Eldredge & Gould, 1972).

Even more important for our discussion is the recognition that elaborate debate exists within the domain of normal development to question the issue of continuity and therefore of prediction. It would be a mistake to assume that prediction is always possible or even a desired goal. The relationship between continuity and prediction allows us to view this problem from a developmental perspective. Much has been written on this topic (Brim & Kagan, 1980; Lewis, in press; Reese & Overton, 1970).

The idea of continuity also involves the idea of gradualism. As espoused by Darwin (1871), gradualism assumes that a series of small changes can account for the development of complex outcomes. Gradualism in evolution has been attacked by many. Eldredge and Gould (1972), for example, proposed a theory of gradual change punctuated by sudden change. Alvarez (1982) and Raup (1986) demonstrated sudden change in evolutionary history. When applied to individual development, notions of continuity and gradualism take several forms, the most common form assumes that a person's development is an intra-individual process. Such theories assume that what the person is like now will determine what the person will be like in the future. It is a "trait" notion of development because the features residing within the individual leads to or becomes, in some one-to-one fashion, some other trait. Lewis (1987) has shown that this model of development predominates, especially in theories of social development that rely on the attachment concept. Traits such as invulnerability are further examples of this model (see Lewis, Chapter 2 in this volume).

Some aspects of development can be characterized by an environmental trait model. Here, developmental continuity resides within the child's environment as well as within the organism. For example, a maladaptive environment (poor mothering) affects the child at each point in time. To the degree that the environment remains constant, so too the degree of continuity of the child's behavior; to the degree that the environment changes, so too will the child. The developmental thrust comes from the environment (see Luria, 1976, for an excellent study of cognitive continuity as a function of environmental change).

The alternative to a trait model, shared by most developmental theorists who consider themselves interactionists, assumes that an individual's development is the result of a continuing interactive process in which people adapt to their changing environments and which, in turn, affects the environments themselves (Featherman, 1983). Such a model makes prediction difficult if not impossible.

Prediction over time is limited in science in general (Heisenberg, 1962), and this may be true for development. The study of individual lives may

have a greater degree of uncertainty than the study of physics, for example, but the principle is the same. For example, Faulconer and Williams (1985), after an analysis of positivism and historicism in human action, concluded that historicism is an illusion; that is, "true" history does not exist, only our reconstruction of it is real (a point also recently made by Spence, 1982). Given this condition, "such a goal (the search for certainty or prediction) is, in principle, impossible for any science, especially a human science" (Faulconer & Williams, 1985, p. 1186). If historical prediction is not possible, it may be possible to understand individual differences from concurrent events or from current beliefs about historical events. Such a view does not endanger developmental study if we do not make prediction a criterion for development or developmental psychopathology (Piaget, 1952; Wohlwill, 1973).

As for continuity in development, the answer remains in some doubt, even if we move from more specific traits to more general ones like the coping strategies suggested by Sroufe and Rutter (1984). Consider the effect of wars and military service on men's lives. Wars are exogenous (and presumably random) to men's lives and yet profoundly affect men's lives, altering them in ways not readily predicted even if we were to have an accurate historical record of their lives before the war (Elder, 1986). We could consider less dramatic events, such as death, illness, floods, and fires, all of which are random to individuals' lives and which may profoundly alter them. For example, in therapy, an adolescent I knew had a terrible bicycle accident which resulted in permanent facial disfiguration. The change in her personality as a consequence of this accident could not have been predicted from her previous behavior or from knowledge of her coping style. Such data confirm a model of discontinuity due to environmental change not of the person's choosing and suggest that prediction may be impossible or difficult at best.

Even if some lives are not influenced by such strong exogenous events, prediction may be difficult. Implicit in Piaget's analysis of development and the nature of stages and change is the concept of discontinuity. We would agree with Piaget that there can be little prediction across the stages that make up early and normal life events. Within the first 2 years of life, we know that at 3, 8, and 15/18 months significant central nervous system events occur that are likely to lead to discontinuities (Lewis, Jaskir, & Enright, 1986; McCall, Eichorn, & Hogarty, 1977). Such happenings, both within and without the organism, mean that the prediction of developmental psychopathology must be difficult if not impossible. This presents a problem if our definition of developmental psychopathology requires prediction.

Summary

We have defined developmental psychopathology as the study of the prediction of the development of maladaptive behaviors and processes. Four separate issues challenge the idea that prediction of abnormal development is possible. *Prospective studies* are required for the best causal relationship between early and later behavior, but prospective studies are difficult to conduct for any number of reasons. In similar fashion, the *classification systems* we currently use make for difficulty in prediction because we are dependent most often on the criteria measures of maladaptive behavior that are either positive or negative vis à vis our outcome. Likewise, age changes in who reports or diagnoses the criteria measures of maladaptive behavior present a problem. Whoever assesses and diagnoses may vary by developmental level. Finally, the notions of *prediction* and *continuity* in normal development are shown to be difficult and not necessary for the study of normal development; more so for psychopathology. All four of these issues are challenges to a theory of developmental psychopathology, which is dependent on prediction. Although the ability to predict maladaptive behaviors and processes from earlier behavior remains a goal, there is no reason to believe that the study of abnormal development is dependent on our success in doing so.

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CHAPTER 4

Culture and Psychopathology

Sara Harkness and Charles M. Super

Anthropological research and, more recently, cross-cultural studies in general have played a delicate combination of roles as they converse with psychological theory. On the one hand, they have provided a method of answering questions about human behavior and development that are central to contemporary thought; on the other, they have often returned only partial answers and, in addition, a new set of questions. The balance of psychological theory contributing to the agenda for anthropological research, in contrast to cross-cultural findings challenging accepted theories of the person, has shifted from time to time, responding in part to the needs and opportunities of the larger disciplines. Throughout the interdisciplinary dialogue are woven the complementary themes of human universals and cultural particulars, and this is no less true for research on psychopathology than for studies of normal development.

The challenge from one perspective is to collect and integrate comparative data about human behavior into a theory that accounts for the universal and the particular; from another, it is to understand the role of culture in the normal and in the deviant. In both cases, a focus on *development* recasts the dichotomy into a question of process. In the case of psychopathology, as other chapters in this Handbook illustrate, a developmental approach substantially alters the terms of understanding from the earlier, adult-oriented model of diagnosis and therapy. With regard to the oft-posed dichotomy in culture and development, we concluded in an earlier review that "it no longer seems appropriate to ask which behaviors are universal and which are culturally variable; all behaviors probably are both, at different levels" (Harkness & Super, 1987, p. 238). An understanding of culture and psychopathology is in many ways only a more specific case of understanding culture and development.

In this chapter, we will review the intellectual history of anthropological contributions to the study of psychopathology across cultures and relate them to the more recent work in developmental psychology and psychiatry that addresses issues of individual functioning in social context. As theories of culture, development, individuality, and psychopathology have themselves evolved, the conversations across perspectives yield a new potential synthesis for research, understanding, and intervention.

Early Formulations of Culture and Psychopathology: Benedict and Mead

A point of departure for much thinking about culture and psychopathology in twentieth-century anthropology was established by Ruth Benedict. In an often-quoted article first published in the *Journal of General Psychology* in 1934, Benedict

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(1934/1973) argued that mental illness exists only in relation to cultural definitions of normal and abnormal. Each culture, she proposed, chooses a portion of the spectrum of possible human behavior as socially appropriate, labeling as "abnormal" those other behaviors that would conflict with the dominant ethos. Benedict suggested further, building on her theory of "patterns of culture" (Benedict, 1934), that the degree of cultural integration in any particular society should coincide with a greater tendency to sanction behavior that from the perspectives of other cultures would appear aberrant.

Benedict made her case on the basis of ethnographic accounts, such as the following description of reaction to the death of loved ones among the Kwakiutl, an American Indian group of the Pacific Coast whose mores had been recorded by ethnographers starting in the late nineteenth century.

> A chief's sister and her daughter had gone up to Victoria, and either because they drank bad whiskey or because their boat capsized they never came back. The chief called together his warriors. "Now I ask you, tribes, who shall wail? Shall I or shall another?" The spokesman answered, of course, "Not you, Chief. Let some other of the tribes." Immediately they set up the warpole to announce their intention of wiping out the injury, and gathered a war party. They set out, and found seven men and two children asleep and killed them. "Then they felt good when they arrived at Sebaa in the evening." (Benedict, 1934/1973, p. 90)

According to Benedict "the point which is of interest to us is that in our society those who on that occasion would feel good when they arrived at Sebaa that evening would be the definitely abnormal" (1934/1973, p. 90). Benedict's larger point here was that individual variation in personality and behavior exists across all cultures, and that it is thus somewhat arbitrary just which kinds of orientations are regarded locally as "good," "normal," or deviant.

Ruth Benedict's younger colleague and friend Margaret Mead shared with her, and with their mentor Franz Boas, an interest in individual psychological functioning in cultural context, and her first field research, which became an American classic, was directed to exploring the role of culture not only in labeling psychopathology but in actually creating it (Mead, 1928). Among adolescent girls of Samoa, as Mead described them, there was no evidence of the social and psychological upheavals that had come to be expected in American society. Mead contrasted the permissive cultural environment of adolescence in Samoa to the strictures of the late Victorian era in the United States. Far from being the normal expression of biological forces, she argued, the troubles of American adolescence could be seen as cultural artifacts (Mead, 1928).

Mead shared with Benedict a view of culture as a larger version of personality: in Benedict's words, "cultures from this point of view are individual psychology thrown large upon the screen, given gigantic proportions and a long time span" (1932, p. 24). The lessons to be derived from this observation, in their views, were twofold: on the one hand, that "human nature is not rigid and unyielding" (Mead, 1939, p. x)—thus, cultural forces could be used to improve as well as to diminish emotional well-being. On the other hand, those who were deviant by the standards of their own societies should be regarded with greater sympathy by people aware that, in other times or other places, these same individuals might not be considered abnormal.

Freudian Theory in the "Culture and Personality" School

The consideration of culture as a magnified version of individual dispositions set the stage for the application of theories drawn from psychopathology to the study of human social groups. This, coupled with the dominance of Freud in lay and in professional circles during the years between the two World Wars, led to a period of close collaboration between anthropologists and their colleagues in clinical psychology and psychiatry. A central event in this period was Abram Kardiner's seminar, organized in 1936 at the New York Psychoanalytic Institute, which brought together practicing analysts with major anthropological figures of the day (see Harris, 1968, pp. 435–448). Drawing from Freudian theory, Kardiner suggested that different societies were characterized by particular "basic personality structures" derived from culturally shared early childhood experience. To pursue this approach required the collection of field data that could be subjected to clinical analysis. This was provided by Cora DuBois, an anthropologist participant in Kardiner's seminar who returned in 1939 from 2 years of field research among the people of Alor in the Dutch East Indies. DuBois's data included Rorschach test protocols, children's drawings, and eight extensive life histories including a great deal of detailed interview material on the recalled experiences of early childhood. These materials were given to Kardiner and his colleagues in

psychiatry for independent analysis; strikingly, all came up with rather similar portrayals of Alorese personality. A description by psychiatrist Emil Oberholzer, based on the Rorschach drawings, is representative:

> From this we assume that the Alorese are suspicious and distrustful; they are so not only toward everything that is new and unknown to them, such as foreigners, for instance, but also among themselves. No one will trust another. Moreover, they are fearful and timid in their heart of hearts, feeling uneasy and insecure. . . . The Alorese must be lacking in individual personal contact, living beside one another but not with one another. . . . Either there are no friendships and relationships or there are none that are deeply rooted. (DuBois, 1944, pp. 598–600)

From the idea of a shared personality structure, as this quotation indicates, anthropologists and their colleagues in psychiatry had moved to the next logical inference: shared psychopathologies. Interestingly, the validity of this notion was not questioned by contemporary and even some later anthropologists, who instead focused their questions on trying to explain the *cause* of the proposed adult personalities—whether they were due to "oral deprivation" of infants when their mothers went off to the fields to work, or, alternatively, whether frequent illness during childhood was to blame (e.g., Barnouw, 1973, p. 159). The fact that societies where there were "no friendships and relationships" might not long survive did not seem to occur to these observers.

The primacy of early experience in the etiology of adult personality was to remain an organizing principle for research in psychological anthropology for the next three decades; and along with it, a continuing identification among the constructs of culture, personality, and psychopathology. Building from Kardiner's hypothesized "primary" and "secondary" institutions, John Whiting proposed a model for psychocultural research that placed child-rearing practices as the crucial connecting link between the culturally constructed environments of early childhood and the development of adult personality, expressed at the cultural level, in turn, by "projective systems," such as religion and belief systems (for a recent statement of this position, see J. W. M. Whiting, 1974, 1977). Whiting and his colleagues put these hypothesized relationships to the test through a series of cross-cultural analyses on the co-occurrence of different kinds of cultural practices, such as mother–child sleeping arrangements and adolescent circumcision ceremonies (J. W. M. Whiting, Kluckhohn, & Anthony, 1958), and early childhood socialization practices and adult customary behavior (J. W. M. Whiting, 1961). Although the studies were correlational, the researchers proposed causal relationships following the proposed cultural sequences, usually with Freudian theories providing specific proposed mechanisms. In J. W. M. Whiting and Child's 1953 landmark study, *Child Training and Personality*, for example, the authors found that strict socialization practices were associated with cultural attributions of guilt in the event of illness; but extremely permissive rearing was not related to a particular pattern of illness beliefs. Whiting and Child concluded that although the cross-cultural data supported Freud's idea of negative fixation, the idea of positive fixation was not upheld.

Some projective systems, such as adolescent circumcision ceremonies, were hypothesized as antidotes to the otherwise pathogenic effects of early childhood experience (in this case, extended mother–child sleeping practices), thus freeing adults of psychopathology. In other cases, however, it seems that the projective systems were themselves regarded as manifestations of culturally shared psychopathology. Thus, for example, Herbert Barry, a prominent cross-cultural researcher, entitled a review paper on much of this literature "Cultural Variations in the Development of Mental Illness" (Barry, 1969). It is important to note, however, that the identification of certain cultural beliefs or practices as pathological (due to unhealthy experiences in childhood) did not seem to imply that one should expect to find whole societies of nonfunctional adults or even large numbers of adults needing psychological treatment. This apparent contradiction is perhaps best understood as a metaphorical use of the concepts of clinical theory for the purpose of understanding cultural dynamics. Reliance on poorly differentiated concepts of culture, personality, and psychopathology, however, led to so many problems of data collection and analysis that their use contributed significantly to the eventual decline of this approach (see LeVine, 1973).

Cross-Cultural Perspectives on Mental Illness: Epidemiology and the "Culture-Bound Syndromes"

In contrast to the long-standing relationship between anthropology and clinical approaches to

psychopathology, the epidemiological study of psychopathology cross-culturally is still in its infancy. Indeed, until fairly recently, the question in this literature was whether mental illness existed at all in preindustrial societies, or whether it was, as Freud had proposed, a by-product of "civilization and its discontents" (Freud, 1930). Anthropological research, such as that of Jane Murphy among the Eskimo of Alaska and the Yoruba of Nigeria (J. Murphy, 1976), and of Edgerton (1966) among four East African tribes helped establish the fact that major disorders, such as schizophrenia, exist in a wide variety of sociocultural settings. More recently, the drive to establish standardized diagnostic techniques (American Psychiatric Association, 1980, 1987; World Health Organization, 1977) has provided the basis for large-scale, cross-cultural psychiatric epidemiology surveys, most notably those carried out by the World Health Organization (e.g., Sartorius, Jablensky, Gulbinat, & Ernberg, 1980; World Health Organization, 1973, 1983).

Epidemiological studies of psychopathology, which start with a template of symptoms recognized as representing a distinct disease entity, have shown that it is possible to find similar-looking patients in widely differing cultures. There are methodological problems in this approach, however, as Lotter (1980) observed in his study of childhood autism in Africa. Lotter encountered problems related to disease classification, finding cases, assessing symptoms in different cultural settings, establishing reliable case histories, and estimating rates of prevalence. Because autism is a rare syndrome in developed countries, Lotter thought it prudent to survey widely. In the end, he found a total of only nine cases of childhood autism among the hospitals of all the major cities of sub-Saharan Africa. Although he noted that estimating rates of population prevalence on the basis of this survey would be impossible, he found that the proportion of autism cases was low even relative to the known (i.e., institutionalized) population of severely subnormal children—about 2.5% in Africa, compared to 5% to 8% in Britain. The possible explanations for this difference include differences in survival or care (including institutionalization) of such children, as well as genetically or culturally based differences in actual incidence.

The problems encountered in cross-cultural epidemiological studies of psychopathology intersect with issues identified through research on mental illness from an "emic" perspective, that is, as defined by members of the cultures where they occur. Anthropologists and other observers have long recognized the existence of culturally distinctive forms of psychopathology. The list of these illnesses, which have come to be known as "culture-bound syndromes," includes such oft-cited examples as *windigo* psychosis among Canadian Indian groups (Fogelson, 1965; Parker, 1960), *latah* among Malay groups (H. B. M. Murphy, 1976; Simons, 1980, 1983), and *susto* (O'Nell, 1975) among Hispanic populations. Another example of one such syndrome is *taijin-kyofu-sho* ("fear of other people," or TKS syndrome), a common psychiatric disorder in Japan. One case history of TKS syndrome has been discussed by Prince and Tcheng-Laroche (1987), based on an account by Honda (1983):

> A 48 year old, unmarried woman presented with the chief complaint of "thinking aloud unknowingly and hurting the feelings of other people around her." She lived with her elderly mother and a brother and sister. After graduation from high school, she had worked as an assistant teacher, a clerical employee, a cartographer, and a clerk for a construction company. In personality she was described as reserved, prudent, conscientious, and tender in sentiment. She was not overly sociable but had some friends and worked well in her jobs.
>
> In the preceding five years, her brother began to think aloud while bathing at home, which the patient disliked; she feared that some day she might do the same thing. When she was about 45 years old, she did begin to feel that she was thinking aloud; she believed she must have spoken ill of other people and hurt their feelings. As evidence, she reported for example that she had received several phone calls in which no one had spoken and these she believed were made by someone who knew her habit of thinking aloud; they had attempted to evoke her habit while she was holding the phone receiver to her ear. She believed her habit of thinking aloud was known to the whole community and she hesitated to go shopping, fearing that people would avoid her, or turn their heads away from her. . . . She wanted to die rather than live under such miserable conditions. The patient's judgment was intact except for this unrealistic fear of thinking aloud. In spite of her fear of other people, she kept on working both at home and at part-time jobs. (Prince & Tcheng-Laroche, 1987, p. 10)

The culture-bound syndromes raise essential issues of cultural causation in psychopathology, and they also challenge current Western-based psychiatric disease classifications. In order to accommodate the case described above in the standard category of "Social Phobia" in the DSM-III (American Psychiatric Association, 1980), for example, Prince and Tcheng-Laroche suggest that several of the criteria would have to be altered. TKS patients, unlike Americans suffering from social phobias, do not admit that their fears of offending

others are excessive. The DSM-III criteria that the social phobia is seldom disabling, that it is relatively rare, and that there is a special relationship between a social phobia and substance abuse would have to be changed to accommodate the Japanese TKS syndrome. Although Prince and Tcheng-Laroche (1987, p. 11) suggest that such changes in the DSM-III would be "minor," they do not address the issue of the loss of specificity for recognizing American syndromes that would inevitably result from this kind of modification. If TKS and "social phobia" are identical at the core, they are distinctly different manifestations.

The pervasive presence of culture in psychopathology has led to a proliferation of disorders described as "culture-bound syndromes." It has recently been suggested, for example, that premenstrual syndrome and postpartum depression are culture-specific disorders of Western society (Harkness, 1987; Johnson, 1987). More generally, cross-culture studies of depression (Kleinman & Good, 1985) suggest that even this presumably universal disorder is so strongly colored by cultural context that to call it by the same name everywhere may not be appropriate. Specifically, as summarized by Marsella, Sartorius, Jablensky, and Fenton (1985, p. 306):

> Results indicate . . . the experience and expression of depression varies across ethnocultural boundaries. Reviewers concur that feelings of guilt, self-deprecation, suicidal ideas, and feelings of despair are often rare or absent among non-European populations, whereas somatic and quasi-somatic symptoms, including disturbances of sleep, appetite, energy, body sensation, and motor functioning, are more common.

Because of the many links between culture and the development and manifestations of psychopathology, it can be argued that *all* illness is culture bound (Cassidy, 1982; see also Marsella & White, 1982). The question then becomes one of utility: how useful is it to develop broad categories of psychiatric disorder that can be applied across cultures, as opposed to narrower categories that evoke culturally recognizable illness experiences? Is the gain in coverage worth the loss in precision? Further, as Carr (1978; Carr & Vitaliano, 1985), has demonstrated for *amok,* disease classifications in other cultures may cut across Western categories, including a variety of psychiatric and socially or physiologically based behavior patterns. Kleinman (1987) has concluded that

> the anthropological model of an idiom of distress offers a more accurate mapping of the experience of culture-bound disorder, and its sources and consequences, than does the medical model. (p. 450)

Consequently, he argues, the application of psychiatric disease categories across cultures can lead to a "category fallacy," or

> the reification of a nosological category developed for a particular cultural group that is then applied to members of another culture for whom it lacks coherence and its validity has not been established. (p. 450)

The problem is strikingly illustrated when another culture's illness is taken as the starting point (Shweder, 1985): It would be possible to study the epidemiology of "soul loss" in an American population, but how would we interpret the results and how might such findings aid clinicians treating depressed patients?

Individual Differences and the "Goodness of Fit"

Cultural approaches to psychopathology have highlighted the necessity to distinguish between sickness and deviance, just as they have also pointed to the importance of differentiating between biological diseases and culturally constituted illness experience. In this context, the problem of individual differences has been recognized in anthropological research, though not always dealt with successfully, since Benedict's early formulation on the cultural relativity of psychopathology. The most substantial progress in this area, however, has taken place in psychiatry and developmental psychology, where considerable energy has been invested in the study of individual variation along dimensions encompassed by the term "temperament."

Best known of the pioneering works on temperament is the New York Longitudinal Study (NYLS) of Thomas, Chess, and their colleagues (Chess & Thomas, 1984; Thomas & Chess, 1977; Thomas, Chess, Birch, Hertzig, & Korn, 1963). The initial thrust of this work was to emphasize the importance of inborn, constitutionally based behavioral dispositions, such as activity level, approach to new situations, and regularity of biological functions as evidenced in feeding, sleeping, and elimination. The target of this thrust, that is, the theoretical position against which this work argued, was the prevailing emphasis on individual differences as the product of experience. The context for the argument was the dominance of the psycho-

analytic perspective in the field of mental health, because the major figures in these theoretical developments were psychiatrists and clinical psychologists. If individual differences in behavioral style were identifiable at birth, went the thinking, and these differences could be traced longitudinally during the early years of life, then differences observed at any one point in childhood could not necessarily be attributed to child rearing. The frequency of intense negative reactions during infancy, for example, might reflect temperament, not the quality of maternal care. One of the first major contributions of the NYLS was the identification of the "difficult infant syndrome," a constellation of irregular biorhythms, avoidance of change, slow adaptability to new situations, negative mood, and intensity of expression that was particularly demanding and upsetting for parents. The crowning irony, in the eyes of the clinician-researchers, was that such behaviors in an infant and toddler suggested to many observers that the mother was doing a poor job. Not only did she have a temperamentally difficult child, but she was blamed for it. This was, in Chess's (1964) words, the ultimate "mal de mère."

As the NYLS progressed, the emphasis shifted from establishing the existence and importance of temperamental differences to understanding the secondary but more critical issue of "goodness of fit" (Chess & Thomas, 1984). This shift was accelerated by parallel research with working-class, Puerto Rican families in New York City that provided a striking contrast with the original middle-class, largely Jewish sample. Because bedtimes were not scheduled and enforced with the same regularity in the Puerto Rican families as was the case in the initial sample, differences in the children's disposition toward a regular schedule were of less significance. Hence, this dimension of temperament did not contribute to parental difficulties and concern (Thomas, Chess, Sillen, & Mendez, 1974). But when the children reached school age and the demands for regularity increased, more families encountered temper tantrums at bedtime and related problems. In contrast, bedtime problems were faced and largely resolved, by some means or another, much earlier in the middle-class sample. In a related analysis that focused more explicitly on the relationship of temperament to behavioral symptomology, Korn and Gannon (1983) found that clinical problems at age 5 years were significantly related to the original cluster of "difficult" temperament scores in the middle-class group for whom this definition of difficulty was initially derived, but that symptomology and "difficult temperament" were not related for the Puerto Rican children. For these families, clinical referrals were more likely to involve excessive motor activity—a disposition possibly related to nutritional and medical factors, but certainly more difficult to handle in small, crowded apartments than in the more spacious middle class environment. In the original sample, there were virtually no clinical referrals for this cluster that was so problematic for the Puerto Rican families. More detailed study of individual cases within the original sample further supports the idea that how families handled their children's behavioral style was critical in determining whether or not potentially poor fit of temperament to environment did ultimately result in the development of behavioral pathology.

The group-comparison method of examining goodness of fit has yielded a few other interesting examples in the literature, but none with psychiatric outcome measures. DeVries (1984), in the most dramatic study, found that Masai infants in Southern Kenya who were most like the classic definition of "difficult" (fussy and irregular, not approaching or adapting, and intense in their responses) were more likely to survive a period of drought and famine. This was interpreted as reflecting the adaptive properties of being a "squeaky wheel" in the context of scarce resources, and several lines of evidence are supportive of this interpretation. Super and Harkness (1981, 1982; see also below) have presented data on the development of sleep in a rural Kenyan farming community that illustrates the ethnographic observation that what constitutes a "difficult" behavioral profile depends heavily on the cultural structuring of the infant's niche.

Cultural Structuring of Responses to Individual Differences

Despite the theoretical importance attached to the match between temperament and environment, relatively little attention has been given to a theory of the environment. In contrast to the several well-constructed instruments for the assessment of individual differences in the opening years of life (Brazelton, 1973; Carey & McDevitt, 1978; Lerner, Palermo, Spiro, & Nesselroade, 1982; Rothbart, 1981), there is no comparably developed or widely known method of assessing relevant aspects of the environment for the individual to "fit." This can be attributed, in part, to very real methodological and logistical problems, as evidenced in efforts to as-

sess the environment for its support of early cognitive growth (e.g., Caldwell & Bradley, 1978). Of greater importance, however, is a synergistic relationship in the behavioral sciences among the methodology, epistemology, theory, and topical orientation of the various disciplines. Psychology and psychiatry, the primary disciplines that are concerned with individual functioning, are subject to a bias toward personological interpretations (Harkness, 1980; Super & Harkness, 1981), as is Western culture in general (Shweder & Bourne, 1982). Even though developmental psychology, in particular, has undergone a substantial reorientation in the past decade in its modeling of the environment (see Bronfenbrenner & Crouter, 1983; Harkness & Super, 1987), the individualistic disciplines lack conceptual traditions for the environmental half of their interactionist theories.

A significant step in this direction, however, draws from the relatively new emphasis on environmental responsiveness to individual behavior. Marked in its beginning by Bell's (1968) reinterpretation of the directional effects in socialization research, and followed by formulations by Hartup (1978), Lerner and Busch-Rossnagel (1981), Scarr and McCartney (1983), and others, this shift in theory has focused attention on the fact that not only do socializing agents influence the growing organism, but also the active, self-directing child stimulates and elicits differential responses from the human environment. The sequential, mutual adjustments and influences can be seen to be a "circular function" (Schneirla, 1957) or a self-regulating, open system (von Bertalanffy, 1968). Temperament theory attempts to describe the initial disposition and the constraints on change in the individual.

By focusing on cultural values and customs, work in anthropology has made salient the fact that culture plays a major role in setting the initial disposition of a child's environment. As ethnographic techniques have grown with the quantitative methodologies forged in psychology and elsewhere in anthropology, the newer literature on child rearing in non-Western societies continues to be persuasive on this point (e.g., B. B. White & J. W. M. Whiting, 1975). In addition, greater attention is now being given to the processes involved in accomplishing the cultural tasks of socialization and preparation for adult functioning (B. B. Whiting & Edwards, 1988).

The next theoretical step in understanding the role of culture in the development of behavior problems and psychopathology, a step not yet fully realized, is to see the environment's pattern of response to individual differences as equally structured by the cultural context. What happens when the fit is a difficult one? Clinically, we have good case studies within Euro-American society of more- and less-adaptive responses, and substantial clinical effort is devoted to promoting more-adaptive ways of responding (e.g., Chess & Thomas, 1986; Turecki & Tonner, 1985). There is a small body of related quantitative research (e.g., Patterson, 1982). New models of family functioning drawn from family therapy point to the importance of socially regulated systems (e.g., Minuchin, 1985), and there is growing recognition that the origins and the solutions to many individual problems are shaped by culturally defined family values and roles (e.g., McGoldrick, Pearce, & Giordano, 1982). A more complete theory, however, has yet to be developed.

One empirical demonstration of the cultural patterns of response has been presented for Kokwet, a community of Kipsigis farm families in Kenya who were studied in detail by Super and Harkness (Harkness & Super, 1983; Super & Harkness, 1981, 1982). Infants over the age of 4 months were often found in the immediate care of an older sibling, a *cheblakwet*, typically an 8-year-old sister. This pattern of sibling caretaking is a common one in world perspective (Weisner & Gallimore, 1977), and one direct consequence is a freeing of the mother to pursue household economic chores that would otherwise be more difficult. In Kokwet, the mother was often nearby, working in the garden or preparing food and supervising the child care as well. The task for the cheblakwet was to entertain and protect the baby. Among activities the cheblakwet could use to keep the baby safe and happy were carrying him or her on her hip or back, strapped on with a cloth. This custom of baby-carrying (again, a common method throughout Africa and the tropical world) not only kept the baby safe from ground-level dangers, such as farm animals and the open fire, it also provided soothing body contact and rocking. In addition, the caretaking sister was often skilled in entertaining the baby through social interaction, talking and cooing, tickling and giving objects for play as caretakers do around the world. When these techniques were insufficient to keep the baby calm, the cheblakwet would return her charge to the mother, who would then nurse and comfort. The close of this common sequence would often be for the infant to fall asleep.

Infants who were not comforted by back-carrying or who demanded more attention from the mother presented, on the one hand, a difficult situa-

tion for the family in Kokwet, for this disposition was one that upset the smooth functioning of family routines. On the other hand, the irregularities of sleeping that are so difficult for American families (and which constitute a major element in Thomas and Chess's "difficult infant syndrome") were of little consequence in Kokwet (Super & Harkness, 1981, 1982). This is the cultural shaping of poor fit.

Of further interest, however, is the different functional consequences of similar infant dispositions (Super, 1988). Although in Kokwet and in a Boston sample infant irregularity (e.g., in sleeping) was highly correlated with maternal attention, the direction of association was opposite in the two contexts. In Boston, infants who were relatively unpredictable in their sleep patterns spent more time interacting with their mothers than did the predictable infants. It appears that the biologically irregular infants more often disrupted their mothers' periods of quiet work or leisure, demanding more attention, and (if coupled with other difficult behaviors) made finding reliable help in child care all the more problematic. In contrast, it was the predictable infants in Kokwet who were more often found with their mothers. The Kipsigis mothers could easily adjust their daily chores to be available for nursing and casual interaction while the baby was likely to be awake, if they could have some sense of when that was likely to be. If the baby were less predictable, some of this interaction would be missed, and the cheblakwet would spend more time keeping her wakeful charge entertained.

Not only did the relationship between this dimension of temperament and the amount of maternal care differ in Kokwet and Boston, but so also did the relative content of mother and caretaker interaction. In Boston, it was the mother whose interactions were more expressively active, with high frequency of vocalization and face-to-face interaction, whereas alternate caretakers were more involved in feeding and direct care routines. In Kokwet, the mothers were more focused on quieting their infants through intimate contact, whereas the sibling caretakers spent relatively more time in active social entertainment.

Several important points are illustrated in the comparison of infant care among rural Kipsigis and upper-middle-class Bostonians. First, it is evident that the environment in which a new human develops is strongly disposed to find certain patterns of behavior more easily acceptable. By itself, this is a reformulation of the classic theorem of Benedict and others that cultures value certain traits or behaviors. A developmental perspective, however, makes visible that the constraints are structured at every stage of life, and that there is a sequence of fits to be made. Beyond individual variation and beyond goodness of fit, there is variety in how families adjust to the challenges presented by infants and children who do not easily adapt to the typical routines and values. Who is available to help with problematic behavior, what techniques are used, how other aspects of the human environment are consequently altered, all these possibilities are prepared in broad outline long before they become activated for any particular child. What culture does is embed individual variation in a matrix of potential responses, personal and institutional.

Cultural Structuring of Responses to Behavioral Distress in the United States: The "New Morbidity"

As reviewed here, anthropological and cross-cultural research on psychological development and psychopathology suggests three main functions of culture in relation to developmental psychopathology. First, cultural settings contribute to the development and manifestation of mental illness and distress. This view of individual variation in relation to cultural context was modern anthropology's first and best-known contribution to the study of psychopathology, and it was further elaborated by the cross-cultural study of child-rearing practices and cultural "projective systems" or aspects of adult behavior. More recently, research in temperament and goodness of fit has provided a new framework for analyzing the mutual adaptations—or lack thereof—in a developmental framework.

The relativistic view of psychopathology that lies behind this work is supported by a second function of culture in relation to psychological illness: Culture labels psychological distress or dysfunction, organizing it into categories and thus further influencing its manifestations. This principle, derived from anthropological and epidemiological research on psychopathology across cultures, does not deny the reality of the biological dimension of phychological illness. In contrast to disease-based epidemiological reseach, however, recognition of the cultural organization of psychopathology leads to the conclusion that there is no universal "ultimate reality" of mental disorders that can be captured by purely medical models. As the examples of TKS syndrome, depression, and "soul loss" illus-

trate, disease categories in different cultures may bear resemblances to each other, but the imposition of strict universal categories would involve considerable loss of specificity for identifying illnesses where they occur and where they are problematic, that is, within particular cultural contexts.

Finally, culture structures responses to mental distress and problematic behavior through the provision of formal and informal theories for understanding by individuals, and through the creation of institutions and professional roles outside of the family, who have responsibility and power to intervene. At the more microscopic level, as indicated in the discussion above, culturally organized responsiveness to individual differences plays a role in the evolution of illness conditions. More broadly, however, social responses to a fully developed problem are overtly structured by the social context. The kinds of interventions that are permitted are socially defined, and the networks for seeking help are socially constructed.

These three functions operate concurrently, as can be seen in the ethnographic analysis of virtually any mental illness. Harkness's (1987) study of postpartum depression, for example, illustrates the intertwining of socially defined settings for pregnancy and birth that promote the illness, of local theories of distress that label emotion states, and of social values and folk theories of disease that determine the societal response.

The relevance of these three functions to developmental psychopathology in American culture is evidenced by the growing concern with "the new morbidity," a term proposed by Haggerty, Roghmann, and Pless (1975) to describe the growing clinical workload presented by psychosocial, behavioral, and related problems. For pediatricians, whose profession has been dramatically altered in a single generation by antibiotics and public health measures (Pawluch, 1983; Russo & Varni, 1982), the shift toward syndromes that involve processes outside the traditionally medical is profound, for it involves

> learning difficulties and school problems, behavioral disturbances, allergies, speech difficulties, visual problems, the problems of adolescents in coping and adjusting . . . family social problems and the management and handling of everyday life stresses. (Haggerty *et al.*, 1975, p. 316)

In some settings, as much as 10% of a pediatrician's time with patients in primary health care is devoted to psychosocial issues (Korsch, Negrete, Mercer, & Freemon, 1971), even though the majority of such problems may be overlooked (Starfield & Borkowf, 1969). For specialists in mental and behavioral problems, that is, for psychiatrists, clinical psychologists, and social workers, the great majority of professional effort is spent with similar issues, including hyperactivity, conduct disorder, functional enuresis, depression, and school failure. The cultural construction, labeling, and responsiveness to each of these syndromes are inherent in their briefest description. Although culture also plays a critical role in the more severe psychopathologies, it is the new morbidity, a specifically cultural construction, that dominates the front lines of professional clinical practice. Epidemiological evidence suggests approximately one-fifth of all children in Western settings suffer psychosocial morbidity (Earls, 1981; Starfield *et al.*, 1984), and only a portion of them receive professional treatment.

Rubenstein and Perloff (1986) pursued the issues of socially constructed syndromes with particular attention to the epidemiology and ethnography of Attention Deficit Disorder with and without Hyperactivity (American Psychiatric Association, 1980). Drawing the distinction between true disease entity and a product of complex individual–environment interactions, they argued that the disease-oriented approach represented by inclusion in the DSM-III is erroneous. Instead, they pointed to the possibility that "hyperactivity is not a disease of the child, but rather the reification of social processes which result in the labeling as sick of children who act badly" (Rubenstein & Perloff, 1986, p. 319). Given the structure of the helping professions in America, this becomes the "medicalization" of psychosocial problems, for the mechanism of response in the primary health care system, and in most psychosocial referral networks, is to treat deviant behavior as sick. Cultural as well as theoretical issues impede the emergence of treatments based on other models that explicitly include a cultural orientation (Pedersen, 1985; Pedersen, Sartorius, & Marsella, 1984).

Culture and Developmental Psychopathology

Culture is the major determinant of the developmental niche of young humans (Super & Harkness, 1986a). The physical and social settings, the customs of child care and rearing, and the psychology of caretaking, these together define what kinds of behavior will be experienced as stressful to

the child's family, what behaviors will be problematic for the child's development. They structure the adaptations that a family and community can make to a poor fit of individual and environment. The sequence of adjustments are the ingredients for learning, for the development of self, and for the acquisition of conflict and coping. At the same time, the growing child is following his or her own parameters of change. The process of adaptation is a mutual one, and at each step of the way cultural meaning and local logistics shape the progression. Some kinds of problems are more likely to occur in a particular context, and some kinds of responses are more likely to be given. Specific institutions for intervention may be activated. The resulting coevolution of individual and environmental systems, in other words, is pointed in one direction or another by cultural forces. Waddington's epigenetic landscape (Waddington, 1957, 1971), in this regard, is an elastic one, responsive to the shape and texture of the rolling ball, but the particular channeling and the pattern of elasticity vary from one human group to another. The outcome, summed over many individuals, yields culture-bound syndromes and the epidemiological landscape, that is, the cultural patterning of psychopathology.

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CHAPTER 5

Taxonomy in Developmental Psychopathology

Consider the Source

Donald K. Routh

The past decade has seen a large investment of scientific and professional energy in research and debate about how to classify psychopathology. Among the most visible results of this process are the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1980), commonly known as the DSM-III, and the third revised edition of this book (American Psychiatric Association, 1987), called the DSM-III-R. There has already been some discussion of a DSM-IV (e.g., Millon & Klerman, 1986). These existing and projected manuals include, of course, consideration of the taxonomy of psychopathology in children and adolescents as well as in adults.

It is important that scientists who are interested in development become more involved with the taxonomy of psychopathology than they have in the past. It cannot be an accident that a number of mental disorders have characteristic ages of onset and often a typical course across the lifespan. For example, according to the DSM-III-R, Autistic Disorder usually has its onset during infancy or (less commonly) childhood. Developmental Reading Disorder is usually apparent by age 7. Conduct Disorder typically emerges by 8 years, and usually not later than early adolescence. Schizophrenia usually has its onset during adolescence or early adulthood. The mean age of onset of manic episodes is in the early 20s, and that for Major Depressive Episodes in the late 20s. Dementia is found primarily among the elderly. Understanding the reasons for these facts alone would undoubtedly contribute greatly to our understanding of development as well as of psychopathology.

The current intense scientific interest in the taxonomy of psychopathology is in marked contrast to the state of affairs only two decades ago. In the 1960s (when I was a graduate student), psychiatric diagnosis was widely criticized by scientists as unreliable. The enterprise seemed to have few friends or supporters. On the one hand, psychoanalysts considered formal diagnosis to be too static and mechanical and invested their energy instead in the description of the idiosyncratic psychodynamic processes of their patients. On the other hand, behaviorists tended to reject the formal diagnosis of psychopathology as part of an outmoded medical model and to describe their subjects only in terms of objectively observable behaviors. The change in our attitudes toward the classification of psychopathology came about largely through the efforts of neo-Kraepelinian researchers who were centered in

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the Department of Psychiatry at Washington University in St. Louis, Missouri. These researchers began to try to develop operational criteria for each diagnostic category that would meet rigorous scientific standards of reliability and validity. They did succeed (in the political sense, at least) in that they captured the endorsement and financial support of the American Psychiatric Association for the project of developing the DSM-III.

In its seven years of existence, the DSM-III has already made a very large impact on research and clinical practice and also has elicited criticism from many quarters. For example, some researchers have criticized the DSM-III for being too empirical in its orientation (Faust & Miner, 1986) and others for not being empirical enough (Quay, 1986). Recently, my colleagues and I (Quay, Routh, & Shapiro, 1987) published an article on the psychopathology of childhood in the *Annual Review of Psychology* that included a critique of the DSM-III and other approaches to the taxonomy of child psychopathology (e.g., approaches based on factor analysis of behavior checklists). We recommended that admissibility to the status of "syndrome" (whether defined as a category or a dimension) should be restricted to disorders that

> (a) can be discriminated from other syndromes and thus reliably diagnosed and measured; and also meet one or more of the following criteria: (b) are associated with different causes, (c) have different outcomes, or (d) respond to different interventions. (Quay *et al.*, 1987, p. 493)

It was not required that the symptoms making up a disorder be tightly intercorrelated; indeed, it seems possible for some symptoms that are legitimately part of a particular disorder to be relatively independent of other ones.

We (Quay *et al.*, 1987) reviewed the following specific disorders as defined in the DSM-III: Attention Deficit Disorder, Undersocialized Aggressive Conduct Disorder, Socialized Aggressive Conduct Disorder, Infantile Autism, and Depression, because these seemed to be the ones of greatest current interest. Our overall judgment was that

> application of these criteria would seem to rule out many of the categories of DSM-III and not a few of the empirically derived dimensions as well. (Quay *et al.*, 1987, p. 493)

There seems to be little doubt, however, about the validity of Conduct Disorder and Autism as syndromes. In fact, Rutter and Schopler (1987) recently concluded that the evidence for the validity of Autism was stronger than that for any other type of child psychopathology. Definite progress is being made toward elucidating some of the basic social deficits of autistic children, for example, in recognizing other people's expressions of emotion (Hobson, 1986; Weeks & Hobson, 1987) and of age- and sex-related characteristics of other people (Hobson, 1987). At least two new possible prognostic indicators have been identified for Autism or Pervasive Developmental Disorder; namely, the presence of hyperlexia (Burd, Fisher, Knowlton, & Kerbeshian, 1987) or of Tourette disorder (Burd, Fisher, Kerbeshian, & Arnold, 1987). There are also some very hopeful recent developments in the behavioral treatment of autistic children (Lovaas, 1987). Thus, progress in validating syndromes of psychopathology may be related to other kinds of scientific progress concerning them.

Like its predecessor, the DSM-III-R may also have erred by excluding some valid behavioral syndromes. Woolston (1987), for example, comments on how eating disorders that result in excessive weight gain are virtually ignored by the present taxonomy. This omission exists despite the fact that, for example, child obesity is related to behavioral factors, such as diet and exercise, and is reliably predictive of obesity in adulthood (Stark, Atkins, Wolff, & Douglas, 1981).

Recently, I (Routh, 1989) elaborated on what is meant by "validity" in child psychopathology. Briefly, the validity of a category or dimension of psychopathology refers to the correctness of inferences one might make about the child on the basis of the child's membership in the category or position on the dimension. The particular inferences one might make depend, in turn, on one's theory, conceptual framework, or hypothesis about the disorder. Clinicians' hypotheses about psychopathology seem most often to have to do with prognosis, response to treatment, or etiology. Thus, the current concept of Tourette's disorder in the DSM-III-R (American Psychiatric Association, 1987, pp. 79–80) is "validated" by observations that (a) this condition tends to have a lifelong course, (b) it responds to medications such as haloperidol, and (c) it can be precipitated in some cases by stimulant drugs. The DSM-III, however, has tried to maintain an atheoretical stance in which it does not use etiological inferences in its classification unless there is a wide consensus about them in the field. Thus, for example, the previous DSM-II label of neurosis (viewed as caused by internal conflict) was deleted in the DSM-III.

Whatever the merits of the above remarks, it

now seems that specific criticisms of the DSM-III may be moot because of its replacement by the DSM-III-R. I do not propose to attempt here any kind of complete evaluation of the DSM-III-R as it relates to child psychopathology but might make some general comments about it before proceeding to the main theme of the chapter. The approach taken by the DSM-III-R is still primarily to assemble committees of experts who, through discussion, will arrive at the definitions of various disorders. A total of 26 advisory committees were appointed, consisting of over 200 members, and they include many recognized experts, a majority of whom are psychiatrists. In addition to the discussions of the advisory committees, three national field trials were held to help in the development of diagnostic criteria for such disorders as Attention-Deficit Hyperactivity Disorder, Oppositional Defiant Disorder, Conduct Disorder, and Autistic Disorder. The data from these field trials enabled items on the symptom lists for some disorders to be listed in descending order of discriminating power. The criteria for syndromes are often polythetic, so that a child does not have to have any one particular attribute to qualify for the diagnosis but only a stated number of the ones on a symptom list. The DSM-III-R continues to conceptualize mental disorders as categories rather than as dimensions. This decision is important, because diagnoses based on the two approaches often do not agree. For example, Steinhausen and Goebel (1987) reported point-biserial correlations ranging from .07 to .21 between parent questionnaire scales and the corresponding child psychiatric diagnoses. In a study by Shekim, Cantwell, Kashani, Beck, Martin, and Rosenberg (1986), it was found that of 14 children diagnosed as having Attention Deficit Disorder by the use of the Diagnostic Interview Schedule for Children–Parent version and the categorical criteria of the DSM-III, only two were similarly diagnosed by the dimensional approach of Achenbach's Child Behavior Checklist.

It is clear that the authors of the DSM-III-R have been reading the research literature and that they have tried to take into account many of the criticisms of the DSM-III in the revision process. For example, I (Routh, 1983), among others, had complained that there was little evidence that Attention Deficit Disorder *without* Hyperactivity was an independent syndrome. The DSM-III-R eliminated this category. Similarly, Quay *et al.* (1987) judged that the current evidence supported the existence of only two distinct types of conduct disorder, not four as in the DSM-III. Indeed, the DSM-III-R eliminated its controversial four-fold classification of conduct disorder and replaced it with two choices, namely, Conduct Disorder, Solitary Aggressive Type and Conduct Disorder, Group Type (plus an undifferentiated category for those who show characteristics of both).

The Importance of Sources of Information

The main goal of the present chapter is to discuss an improvement that might be made in the DSM-IV or in other future classifications of child psychopathology that I believe would be of considerable benefit; namely, greater specification of the particular *sources of information* to be used for each diagnostic category or dimension.

Because the concept of psychopathology continues to be based on a medical analogy, let me use a medical example to explain what is meant by sources of information. In a medical examination, the physician evaluates the signs and symptoms presented by the patient, with *signs* referring to objective observations (such as a skin lesion) and *symptoms* referring to subjective complaints (such as a report of pain). In this relatively simple situation, signs are one source of information and symptoms are another. If the patient is a child, the physician might also have to depend upon some observer, such as the child's parent, to report particular information; for example, a high fever that occurred the night before, or the child's previous complaint of nausea. The parent's reports are thus an important third source of information. Medical technology permits the use of such instruments as a stethoscope or an electrocardiograph to make more refined observations of bodily phenomena, and such laboratory tests as chemical assays of blood or urine. If need be, surgical procedures can be used to obtain biopsy specimens. Instrumented observations and laboratory procedures are a fourth key source of information in medical diagnosis. The use of all these sources of information multiplies the complexity of the diagnostic process further but also has great potential to make it more precise and accurate.

The diagnosis of child psychopathology may also involve the use of multiple sources of information, many of which are the reports of various observers each in a position to have some kind of unique knowledge about the child. Obviously, one can observe the child's behavior directly in the clinic or in a natural setting, such as the home or

school. (For example, an observed behavior, such as stereotyped rocking, in a child beyond the age of infancy might be regarded as analogous to a medical "sign." The situational context of such observation may be more important for behavior than for physical signs of illness.) The clinician can also interview the child, for example, about the child's worries or fears ("symptoms"). Interviews with the child's mother or father are probably the source of information that is used most often in the diagnosis of child psychopathology. Instrumentation (such as electronic motion detectors to record motor activity level) can provide an additional relevant source of information. There are an increasing number of biological laboratory procedures that are useful in the diagnosis of child psychopathology (an example is the use of cytology to diagnose chromosomal anomalies, an increasing number of which are associated with mental retardation or specific types of behavior disorders). In 1986, McCauley, Ito, and Kay showed Turner's syndrome, involving the absence of one X chromosome in females, to be associated with poor peer relationships and social isolation. According to Bregman, Dykens, Watson, Ort, and Leckman (1987), fragile-x syndrome is associated with quite a number of cases of Attention Deficit Disorder and of Autism. Surgical procedures have not been of much diagnostic help in child psychopathology. However, there are numerous psychometric or behavioral science laboratory procedures (e.g., cognitive ability tests, standardized achievement tests, and the Continuous Performance Test) that give relevant information. And as is discussed in more detail below, in evaluating children's behavior, information from a wide circle of observers, such as teachers and peers, may be of help.

One of the noteworthy changes in the DSM-III-R as compared to the DSM-III is that it does, sometimes at least, specify the sources of information to be used for particular diagnosis. For example, in the DSM-III-R, Mental Retardation is defined by (a) subaverage general intellectual functioning, (b) concurrent deficits in adaptive behavior, and (c) onset before age 18 (American Psychiatric Association, 1987, pp. 28–33). It is explicitly stated in the DSM-III-R that intellectual functioning is to be assessed by one or more individually administered general intelligence tests, such as the Wechsler Intelligence Scale for Children-Revised, Stanford Binet, or Kaufman Assessment Battery for Children. It is also stated that such scales as the Vineland Adaptive Behavior Scales and the American Association of Mental Deficiency Adaptive Behavior Scale (which are based on the reports of the parents or other observers familiar with the individual's behavior in everyday situations) have been found useful for quantifying adaptive functioning. It is the thesis of this chapter that further specification of sources of information is a goal to work toward in the definition of any type of psychopathology. Such detailed specification of Mental Retardation by the DSM-III-R is not necessarily an indication that there is total consensus in the field about the disorder. Indeed, a lively controversy exists as to whether the diagnosis of retardation should be made on the basis of the IQ alone (Zigler, Balla, & Hodapp, 1984) or continue to be based on IQ and adaptive behavior (Barnett, 1986). Even though there is at this time relatively good acceptance of particular intelligence tests at the operational level, we are still far from agreement in theory on a concept of intelligence. Research on normal cognitive development continues to be very active, and historically there has been good empirical support for the linkage of normal and retarded mental development in terms of sequences and levels attained (Inhelder, 1968).

But more commonly, the DSM-III-R, like its predecessors, simply indicates the criteria for a particular diagnosis without specifying the sources of information to be used as a basis for judging whether a child meets the criteria. For example, the statement of the diagnostic criteria for Conduct Disorder in DSM-III-R begins in the following way:

> A disturbance of conduct lasting at least six months, during which at least three of the following have been present: (1) has stolen without confrontation of a victim on more than one occasion (including forgery), (2) has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning), (3) often lies (other than to avoid physical or sexual abuse), (4) has deliberately engaged in firesetting, (5) is often truant from school (for older person, absent from work). (American Psychiatric Association, 1987, p. 55)

This symptom list continues and includes 13 items in all. Obviously, information concerning such behaviors as the above could potentially be obtained in various ways. Among the possible sources of information are: direct observation, asking the child himself (or herself), talking to parents, talking to neighborhood or school peers, asking the child's teacher, or searching police records. Each of these methods has some obvious advantages and disadvantages. If it were available, direct observation would seem the most veridical source of information but would be limited by the covert nature of much stealing or firesetting, and might not be economically feasible in view of the low base rates of many such events. The child, having personally

carried out these misbehaviors (or having not), undoubtedly has the knowledge to answer questions but understandably may have a motive to conceal or minimize. The parents may be more forthcoming in discussing their child's misbehaviors but may not know about all of them. In fact, parents bringing their child for treatment might tend to exaggerate the severity of the child's behavior, showing a negative halo effect to justify their request for help (Treiber & Mabe, 1987). Although reports by the two parents are usually correlated with each other, they are not equivalent. There is evidence that fathers do not observe the same, or as many, child behaviors as mothers (Hurlbert, Gdowski, & Lachar, 1986). Peers and teachers may be more candid than even the parents and may have unique knowledge about the child's activities outside the home (e.g., truancy), yet they may not be aware of some misbehavior known to the child or to the parents. In other words, these various sources of information are logically not equivalent to each other but can be seen as complementary. Yet the DSM-III-R does not tell us which sources of information to use. This fact no doubt limits the reliability and the validity of the diagnosis.

In the Isle of Wight study, Rutter, Graham, and Yule (1970) pioneered in the development of systematic procedures for collecting information from children, parents, and teachers in the epidemiological study of child psychopathology. Rutter was also a leader in proposing a multiaxial diagnostic system in child psychiatry. Achenbach (1985) argued for a multiaxial system that incorporates a somewhat larger number of sources of information into the clinical assessment of children and adolescents. In this system, Axis I would be based on parent perceptions, Axis II on teacher's perceptions, Axis III on cognitive assessment, Axis IV on physical conditions, and Axis V on clinician's assessment via observations or the self-reports of the child. This system seems to me praiseworthy in that it encourages systematic data collection and recording. However, it does not take the next step and specify how this information would be integrated in making particular diagnostic statements.

Review of Research Concerned with Different Sources of Information in Assessment

The Multitrait–Multimethod Matrix

The classic work on the use of different sources of information in psychological assessment was Campbell and Fiske's (1959) paper on the multitrait–multimethod matrix. Campbell and Fiske presented an idealized conception in which the goal was to measure each of several independent traits by each of several independent methods. What they labeled as a method corresponded to what is called here a source of information.

If Traits A, B, and C are measured by three different methods, for example, self-report, parent report, and teacher report, it is possible to construct a triangular matrix of all the resulting intercorrelations. On the main diagonal of such a matrix are the *reliabilities* of the measures—that is, how well each measure (involving a particular trait and method) correlates with itself in the sense of internal consistency or test–retest reliability. Obviously, good measures should be reliable.

There are several smaller arrays of correlations in such a matrix called *convergent validity* diagonals. These show how different measures of the same trait (by different methods) correlate with each other. Convergent validity is present if these correlations are high in an absolute sense. For example, children considered by their teachers to be aggressive might also be so described by their parents and by themselves. (Actually, the true situation is not so simple, as already pointed out above, we cannot necessarily expect all sources of information to agree with each other.)

In an earlier era, the discussion of the validity of procedures for evaluating psychopathology centered around convergent validity—or concurrent validity as it was usually called (American Psychological Association, 1966). The idea was to find some criterion measure or "gold standard" against which to validate the procedure being evaluated. Thus, early IQ tests were evaluated against the criterion of teacher judgment, and the original Minnesota Multiphasic Personality Inventory was validated against the criterion of psychiatric diagnosis. The criterion measures that were selected thus acquired a kind of sanctified status that hindered investigators from looking too closely at them. Current thinking (e.g., Joint AERA, APA, NCME Committee, 1985) has begun to recognize that it may be unrealistic to expect a single such criterion to emerge. Thus, all validity has been recognized to be what used to be called "construct validity," and the correlation of two particular measures (i.e., sources of information) is only one source of evidence as to the correctness of inferences specified by theory.

To return to our consideration of the multitrait–multimethod matrix, one concept highlighted by this approach was that of "method variance"

(also called "source variance"). This shows up in the multitrait–multimethod matrix in the form of relatively high correlations among different traits measured by the same method. For example, teacher ratings of children might show a "halo effect" in which the children the teacher likes receive high ratings on ability, achievement, and popularity, regardless of their actual standing on these distinct dimensions. On self-report measures, method (source) variance might be seen in the form of social desirability responding in which the same individuals tend more than others to deny the presence of all kinds of unfavorable attributes. It should not be supposed that method variance is confined to ratings by humans; it can also be seen in standardized laboratory measures of behavior in the form of "apparatus factors" and the like—for example, a particular polygraph might give simultaneously distorted recordings of heart rate, respiration, and electrodermal response from some individuals.

One of the classic demonstrations of method variance in the assessment of child psychopathology was seen in Langhorne, Loney, Paternite, and Bechtoldt's (1976) study of hyperactive children. In a factor analysis of their own data and that of a previous study by Routh and Roberts (1972), these investigators did not find factors for different types of psychopathology (e.g., hyperactivity, conduct disorder, and the like). Instead, their analysis revealed "source" or method factors, for example, a teacher factor, a parent factor, or a clinician factor.

According to Campbell and Fiske (1959), if traits have *discriminant* validity, the intercorrelations of the same traits measured by different methods will be high enough to override "method" variance and also higher than other correlations in the matrix.

Criticisms of the Multitrait–Multimethod Matrix

Although the multitrait–multimethod matrix has been used as a framework for a considerable amount of research, the validity criteria of Campbell and Fiske (1959) have proved to be too stringent for most applications to actual data. For one thing, it is commonly true that traits of interest are, in fact, correlated with each other rather than independent. A commonplace physical example might be that of height and weight. Tall people tend to be heavier than short people, though they need not be so. In a matrix in which the traits were height and weight, the fact of their nonindependence might erroneously lead one to conclude that there was bias or method variance in their measurement (e.g., because self-reported height and weight are correlated), even if the self-reports or physical measures were totally veridical.

Another common event in actual studies is that the different "methods" of measurement are not equally credible, whereas the matrix treats them as if they were. For example, Burton (1970) compared various measures obtained through direct observation of children in nursery schools with retrospective reports by the parents of the children and with retrospective reports by the children themselves many years later. The study showed that there was very little agreement of these three sources of information about various "personality" variables. Thus, what an interviewer gets when asking an adult about childhood events, although it may be interesting in itself, must be regarded as largely fiction (i.e., created by filling in material missing from memory through confabulation) in comparison to the veridical information. If historical information of this kind were necessary in the diagnosis of some kind of psychopathology, the diagnostician would be well advised to specify the necessary source of information, such as the childhood records, and not recall by the parent or by the individuals themselves years later.

Use of Multisource Information in the Diagnosis of Child Psychopathology

It can be risky to use only a single source of information in the diagnosis of psychopathology because of the possibility of bias. For example, Griest, Forehand, Wells, and McMahon (1980) found that mothers' depression as measured by the Beck Depression Inventory was related to maternal ratings of child psychopathology. However, these mothers' ratings of their child's disturbance were found to be unrelated to behavioral observations of the children. In child diagnosis, the use of information solely from the mother could thus cause the clinician to treat a child's behavioral difficulty instead of more correctly trying to alleviate the mother's depression. More recent research on this topic by Brody and Forehand (1986) has suggested that there may be an interaction between maternal depression and the level of child noncompliant behavior—high levels of maternal depression and of child noncompliance are especially predictive of mothers' perceptions of child maladjustment.

One way to use multiple sources in diagnosis is

simply to combine them, possibly using some kind of weighted average. The reasoning behind this practice is simply that any one source of information is contaminated by certain biases and that these biases may tend to cancel each other out when multiple sources are used. One of the most sophisticated attempts to use information from different sources in defining child psychopathology is that of Patterson (1986). To measure his "antisocial" (overt and clandestine) construct, Patterson combined information from the teacher (Child Behavior Checklist, Achenbach & Edelbrock, 1979), from peers and from parents (two mother questionnaires, two father questionnaires, and telephone interviews with each parent), and from the child himself (telephone interview). Similarly, each of the 17 or more constructs in Patterson's model, such as Child Coercion or Inept Discipline, was built up from multiple sources of information, and the model was evaluated by use of structural equation modeling. This approach seems much more satisfactory to me than, for example, the way in which the DSM-III-R criteria for Conduct Disorder are stated. Similarly, Fergusson and Horwood (1987a,b) present a mathematical method for estimating the trait- and method-specific components of maternal and teacher ratings of childhood conduct disorder. Adjustment of the data for method-specific factors produced trait estimates that were highly stable over time, as shown by a correlation of .92 over a 1-year period.

A second way to use multiple sources of information in the diagnosis of child psychopathology is to combine them *selectively*, excluding information thought to be less valid for the purpose. A study by Rapoport and Benoit (1975) illustrates why such selectivity might be needed. These investigators obtained teacher questionnaire information, ratings by clinical examiners, and parent questionnaires concerning children's hyperactivity. They also made home visits, usually after school, obtaining counts of the children's spontaneous activity shifts in play and the number of negative interactions, as well as global observer ratings of hyperactivity at home. In addition, they had mothers complete a standard 4-day diary of their child's behaviors and activities. The parent questionnaires were found to be *unrelated* to all the other sources of information about the child; however, the home visit observations and the mothers' diaries were related to clinic and school ratings, demonstrating that the boys' behavior was similar across settings. Similarly, Rapoport, Donnelly, Zametkin, and Carrougher (1986) compared teacher-rated hyperactive children who were or were not also so described by parent ratings. These two groups did not differ in terms of directly observed or mechanically measured activity levels. Therefore, the distinction between so-called situational and pervasive hyperactivity seems to lie in parent attitudes (i.e., source variance) rather than in the characteristics of the child. The implication is that the diagnosis of Attention-Deficit Hyperactivity Disorder should make use of teacher and clinician ratings and home observations (and parent diaries if available), but should not use parent ratings.

Use of Specific Sources of Information

There does not seem to be any general rule for what sources of information to use in the diagnosis of all child psychopathology. Clinicians depend most upon interviews with the parents, and yet there are instances in which other sources of information could be much more important. For example, in assessing affective states, such as depression and anxiety, in children, it would seem to be important *a priori* to get information directly from the child. Who, after all, has more veridical information than the person who is experiencing such affective states? In recent years, research has gone far toward providing convincing information that children can be reliable reporters of their own experiences and behavior and toward developing standardized child-interview procedures (Herjanic, Herjanic, Brown, & Wheatt, 1975; Herjanic & Reich, 1982; Hodges, McKnew, Cytryn, Stern, & Kline, 1982). In an interesting study of 229 children referred to a psychiatric clinic, Edelbrock, Costello, Dulcan, Conover, and Kala (1986) found that parent–child agreement on symptoms increased with age and was higher on some symptoms than on others. Parents reported more conduct problems in their children, whereas the children reported more affective and neurotic symptoms. Perhaps it is not so much a question of who is right and who is wrong about such symptoms but rather whose point of view is taken in describing the same events. For example, if I feel "depressed," I might seem to you merely "irritable." A study by Wolfe, Finch, Saylor, Blount, Pallmeyer, and Carek (1987) had children and their teachers answer questions about the children's experiences of negative affects, such as depression, anxiety and anger. Although the teachers' ratings of internalizing were, in general, predictive of the children's reports of depression and anxiety, discriminant validity was not found for teacher rat-

ings of the particular emotions. Thus, it may be that teachers are even less sensitive than parents to children's subjective experiences.

In other instances, laboratory procedures seem to provide the most definitive information. For example, Bornstein and Sigman's (1986) review makes a convincing case that, after all, there may be continuity in mental development from infancy, provided appropriate procedures are used to assess infants' cognitive status. Thus, in screening for mental retardation, such tests as that being developed by Fagan (1984) seem destined to replace existing infant-testing technology, such as the Bayley (1969) Scales of Infant Development. Certainly, parent impressions of the infant's intellectual status have no predictive validity.

One highly valid source of prognostic information that seems to be neglected in the assessment of child psychopathology is peer ratings. The classic study by Cowen, Pederson, Babigian, Izzo, and Trost (1973) of third-grade children in Rochester, New York, demonstrated that negative role nominations on the peer measure known as The Class Play were the best predictor of membership on a community psychiatric register 11 years later, better than clinician ratings, teacher ratings, achievement test scores, or anything else. Similarly, Huesmann, Eron, Lefkowitz, and Walder (1984) found that peer ratings of aggression obtained when children were in the third grade significantly predicted adult criminal behavior, number of moving traffic violations, convictions for driving while intoxicated, aggressiveness toward spouses, and severity of punishment of the subjects' own children 22 years later. Clinicians have been reluctant to incorporate peer ratings into their standard assessment procedures, partly because of the difficult logistics involved in collecting such information. There appears to be a need for a change in this attitude.

A further source of information that should not be neglected in diagnosis is the clinician's observations and ratings. An example of an excellent procedure of this type is the Childhood Autism Rating Scale (CARS) for Diagnostic Screening and Classification of Autism (Schopler, Reichler, & Renner, 1986). From the beginning, the field of autism has been plagued by the lack of reliable operational criteria for diagnosis. The lack of such measures meant that there was considerable variation from one center to another in the group of children labeled autistic. The manual for CARS presents data from the use of this clinical rating scale with 1,606 children estimated to include 46% who were handicapped but not autistic and 54% who were autistic. CARS consists of 15 separate ratings, each one ranging from 1 (within normal limits for age) to 4 (severely abnormal for that age). Each point on the scale is anchored by a specific verbal description. For example, on Scale I, Relating to People, a rating of 4 means

> severely abnormal relationships. The child is consistently aloof or unaware of what the adult is doing. He almost never responds to the adult or initiates contact with the adult. Only the most persistent attempts to get the child's attention have any effect. (Schopler *et al.*, 1986, p. 28)

The individual scales are reported to have a mean interrater reliability of .71. The overall scale had an internal consistency (Cronbach's alpha) of .94 and a test–retest reliability over a 1-year period of .88. The criterion-related validity of the CARS in relation to independent clinical ratings of the children was found to be .84. CARS ratings are usually based on observations of the child during psychological testing, but information is presented suggesting that equivalent ratings can be obtained from parent interviews, classroom observations, chart review, and similar sources. CARS is not considered to provide all the information necessary for diagnosis. Other information, for example, from the medical history and physical, must be integrated with the ratings for this purpose. Information is given in the manual on the rationale for inclusion of each of the 15 rating scales and on the relation between the items and each of 5 major diagnostic systems, including the DSM-III. No doubt the scale can be easily adapted for use with the DSM-III-R criteria as well. It deserves a try.

A final source of information that future versions of the taxonomy of child and adolescent psychopathology may wish to include is that on the environment or ecology of the behavior disorder. The inclusion of such information would, however, depend on a consensus in the field that it had etiological significance for particular disorders. Such research as that by Rutter, Maughan, Mortimore, and Ouston (1979) on the effects of secondary schools on children has continued to open our eyes to the importance of psychosocial environmental factors in psychopathology. Examples of current ecological measures that might be considered for such purposes include the Home Observation for the Measurement of the Environment (HOME, Caldwell & Bradley, 1980), Sines' Home Environment Questionnaire (HEQ, Laing & Sines, 1982), and Olson, Bell and Portner's (1978)

FACES scales. Drotar and Crawford (1987) have made a cogent case for the clinical importance of information obtained from home visits in selected cases, for example, infants with Non-Organic Failure to Thrive. Such information should ultimately be of relevance to formal diagnosis as well.

Conclusions

In recent years, there have been a number of significant advances in the area of taxonomy in child psychopathology. A proper appreciation of taxonomic issues thus seems essential to the field of developmental psychopathology. The DSM-III seems to be an improvement over its predecessors, and the DSM-III-R also seems to have gone a step further toward a satisfactory scheme. However, most of the definitions of disorders in the DSM-III-R do not adequately specify the particular sources of information to be utilized in diagnosis. The DSM-IV should therefore "consider the source" much more seriously than has been done in the past.

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PART II

Undercontrolled Disorders

CHAPTER 6

Attention Deficit Disorders
History, Definition, and Diagnosis

Russell A. Barkley

Attention Deficit Disorder (ADD) is the most recent label for children who display developmentally inappropriate degrees of poor sustained attention, impulsivity, overactivity, and problems with adherence to rules and instructions.[1] Children with ADD comprise a heterogeneous population with there being considerable variation across children in the extent to which they demonstrate all these symptoms. Moreover, a large percentage of ADD children display a higher prevalence of other medical, psychiatric, and developmental disorders, further contributing to the diversity of characteristics of this group. The disorder represents one of the most common reasons children are referred to child guidance centers in this country, and is one of the most prevalent of the childhood psychiatric disorders. This chapter will review briefly the history of the disorder, its nature, and its present definitions, as well as recently proposed research and clinical diagnostic criteria for identification of children with the disorder. Socialization problems and etiologies are discussed in other chapters in this volume (see Campbell, Chapter 7, and Deutsch and Kinsbourne, Chapter 9).

[1]This chapter had been written before 1987, when Attention Deficit Hyperactivity Disorder (ADHD) replaced Attention Deficit Disorder (ADD) as a label.

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History

Descriptions of children with behavioral symptoms similar to ADD have been reported as far back as the 1860s, but Still's report (1902) is probably the most complete. In a series of lectures to the Royal College of Physicians in England, Still described a group of children in his clinical practice that would likely now be diagnosed as ADD with oppositional or conduct disorders. These children were often aggressive, defiant, resistant to discipline, excessively emotional or passionate, and showed little "volitional inhibition" or self-control. Deficits in attention and regulation of activity level were common in this group, whom Still referred to as having "defects in moral control." A greater proportion of males to females were noted to have the disorder. Still was perhaps the first researcher to describe the greater incidence of minor physical anomalies among these behavior disordered children. A family history of alcoholism, conduct disorder, and affective illness was observed more frequently in such children than in other clinic samples. Still believed that a chaotic family environment might lead some children to develop such problems, but he was struck by the fact that these children could come from homes

with seemingly adequate upbringing environments. He proposed a biological predisposition to the disorder that was hereditary in some children or the result of pre- or postnatal central nervous system damage in others.

A few papers (Childers, 1935; Levin, 1938) dealing with restlessness in children appeared over the next 35 years but were of limited impact upon the field. However, a significant series of papers appeared from 1937 to 1941 (Bradley, 1937; Bradley & Bowen, 1940, 1941; Molitch & Eccles, 1937), describing the efficacy of stimulant medication with behavior disordered children, many of whom were probably ADD or ADD with conduct problems. This set the stage for the beginnings of modern pediatric psychopharmacology in general as well as the now widespread treatment of ADD children with stimulant drugs. Yet truly widespread interest in childhood disorders did not emerge until after World War II. The most prominent among the publications of the time was the work of Strauss, Lehtinen, Kephart, and others (Strauss & Lehtinen, 1947). These authors reasoned that if hyperactivity, poor attention span, impulsiveness, and distractibility were the sequelae of brain damage in children, then children presenting with such behaviors must certainly be brain-injured, despite any documented evidence of such a history. Recommendations for educating these "brain-injured" children were set forth, chief among which was the need to reduce distracting stimuli in classrooms to a minimum so as not to interfere with the children's limited focused attention. Strikingly austere classroom environments were recommended in which teachers could not wear brightly colored or patterned clothing or jewelry and pictures could not adorn the walls of the class. In less extreme forms, such recommendations remain commonplace today despite minimal support for the efficacy of this idea (Routh, 1978) and evidence that ADD children are not necessarily more distractible than normal children (Douglas, 1983).

In the next decade, several papers appeared referring to these children as hyperkinetic and examining possible central nervous system deficits in the filtering of stimuli reaching the cortex (Laufer, Denhoff, & Solomons, 1957). By 1960, the Hyperactive Child syndrome (Chess, 1960) was a well-accepted childhood psychiatric disorder with greatest emphasis being given to overactivity as the defining feature of the disorder. Research in the 1960s was chiefly devoted to the measurement of activity level via a variety of creative means (e.g., grid-marked playrooms, modified self-winding wristwatches, pneumatic pads, and ultrasonic sound-wave generators; (see Tryon, 1984, for a review). The criteria for diagnosis was felt to be a level of daily motor activity that placed the child two standard deviations above the mean for normal children of similar age and sex (Werry & Sprague, 1968). Significant during this era was the belief that hyperactivity was part of a syndrome known as Minimal Brain Damage (MBD) and later, after much criticism from child neurologists, Minimal Brain Dysfunction (Wender, 1971). Regardless of label, the belief that hyperactivity resulted from brain damage would be widely held until the late 1970s when a series of papers (Rie & Rie, 1980, for a review; Rutter, 1977) would disprove the syndromal nature of the disorder and its relationship to brain damage. Although the term MBD has apparently passed into disuse as a diagnostic syndrome, some researchers (Taylor, H. G., 1983) continue to support its utility as an etiological hypothesis that at least directs attention away from environmental or emotional causes to those involving the central nervous system.

In the early to mid-1970s, a series of studies was reported that strongly questioned the central role of hyperactivity in this disorder (Douglas, 1972, 1980; Douglas & Peters, 1979) and instead argued that deficits in sustained attention and impulse control were as central to understanding such children as was activity level. Over time, this conceptualization of ADD would come to view the disorder as arising from impairments in (1) investment, organization, and maintenance of attention, (2) the inhibition of impulsive responding, (3) the modulation of arousal levels to meet situational demands, and (4) an unusually strong tendency to seek immediate reinforcement (Douglas, 1983). The model has proven extremely heuristic, generating numerous hypotheses and research studies to test them. This body of research was probably the most important factor contributing to the decision to relabel the diagnosis of Hyperkinetic Reaction of Childhood (American Psychiatric Association, 1968) to Attention Deficit Disorder, with or without Hyperactivity (American Psychiatric Association, 1980). Although this diagnostic category has now been revised and renamed Attention Deficit-Hyperactivity Disorder (American Psychiatric Association, 1987), attention deficits and impulsivity remain central to its conceptualization.

The author has recently proposed (Barkley, 1989) that deficits in attention and impulse control may not necessarily be the fundamental behavioral deficiencies in ADD children, although they cer-

tainly characterize this group. Instead, the deficits in sustained attention, impulse control, and poor regulation of activity level to situational demands stem from more basic or fundamental deficits in (1) rule-governed behavior, (2) the regulation of behavior under partial schedules of consequences, and/or (3) rapid behavioral extinction (heightened satiation or habituation to consequences). Such deficits are developmentally inappropriate for the child's mental age and sex and give rise to apparent short attention span, poor response inhibition, and problems regulating activity to setting demands. The need for such a reconceptualization of ADD symptoms was founded on the commonly accepted evidence for substantial cross-situational variation in the level of symptoms and the response of ADD children to various reinforcement schedules. Much research is needed before it can be determined whether this is a more heuristic yet parsimonious theory of ADD relative to the cognitive attention/impulsivity model of Douglas.

Description

Primary Symptoms

By definition, children with ADD are deficient in their ability to sustain attention or concentration to activities relative to same-age peers. Although this may be noted in free play activities, as measured in average time played per toy or activity (Barkley & Ullman, 1975; Routh & Schroeder, 1976), it is most obvious in situations in which the children are required by others to sustain their responding to especially dull, boring, repetitive, or effortful tasks that have little intrinsic appeal (Luk, 1985; Ullman, Barkley, & Brown, 1978). The difficulties with sustained attention are frequently assessed using vigilance or continuous performance tasks (Barkley, 1988; Douglas, 1983). In these tasks, children are required to watch a screen while stimuli are presented in rapid succession, and they must respond to only certain target stimuli while not to others. Errors of omission, or missed target stimuli, are taken as evidence of poor sustained attention. The deficit does not appear to be one of heightened distractibility, defined as the probability of a child being drawn off of an assigned task by ambient task irrelevant stimuli (Douglas, 1983; Ross & Ross, 1982). Instead, the problem appears to be one of persistence in responding, wherein these children become more readily bored or disinterested in activities than same-age children. Although differences from normal children have been noted in other components of attention, such as span of apprehension (Denton & McIntyre, 1978) or poor selective attention (Plomin & Foch, 1981), these have not been reliably demonstrated.

Impulsivity, or the failure to stop and think before responding to a task, is also frequently included as a hallmark of this disorder. This is most often demonstrated on such tasks as the Kagan Matching Familiar Figures Test (Brown & Quay, 1977; Kagan, 1966) or in direct-reinforcement-of-latency (DRL) paradigms (Gordon, 1979; McClure & Gordon, 1984). Many studies have also used commission errors (responses to nontarget stimuli) on vigilance tests as indices of impulsivity in children. Like attention, impulsivity is a multidimensional construct (Milich & Kramer, 1985), and it is not always clear in studies which aspects of impulse control are impaired in ADD children. Although not as thoroughly studied, the degree of impulsivity also seems to show significant situational fluctuations (Draeger, Prior, & Sanson, 1986). Furthermore, studies that have factor-analyzed behavioral ratings or objective measures of impulsivity along with other constructs have not shown that a separate factor emerges representing the impulsivity measures (Achenbach & Edelbrock, 1983; Milich & Kramer, 1985). Instead, these measures appear to relate most highly to measures of either activity level or attention. This calls into question whether impulsivity is in fact a separate, primary symptom or merely another facet of the difficulties with sustained responding to tasks. This would certainly be consistent with views of impulsivity as active inhibition of behavior. Attention is therefore viewed as poor persistence of action, whether of initiation or inhibition (Barkley, 1989).

Hyperactivity was once believed to be the *sine qua non* of this disorder (Chess, 1960), until later research questioned its primacy (Douglas, 1972) and it became relegated to the status of a co-existing symptom among a triad of primary deficits (American Psychiatric Association, 1980)—a symptom not even necessary to render the diagnosis. Numerous studies have demonstrated that ADD children are often more active or restless than normal children (Barkley & Cunningham, 1979; Porrino, Rapoport, Behar, Sceery, Ismond, & Bunney, 1983), but here, too, situational fluctuations in activity level are common as is often seen in normal children (Jacob, O'Leary, & Rosenblad, 1978; Luk, 1985; Routh, Schroeder, & O'Tuama, 1974). However, it has not been convincingly shown that ADD children differ from control groups of non-

ADD psychiatrically disturbed children in rates of activity (Firestone & Martin, 1979; Sandberg, Rutter, & Taylor, 1978; Shaffer, McNamara, & Pincus, 1974).

Recent conceptualizations of ADD (Barkley, 1981; 1989) have included a fourth primary deficit: developmentally inappropriate deficiencies in rule-governed behavior. Rules are stimuli constructed by an individual or the verbal community (e.g., parents and teachers) that specify relations (contingencies) among behavior, antecedents, and consequences (Skinner, 1969). Behavior that appears to be under stimulus control by such stimuli is said to be rule-governed. In its simplest form, it is the demonstration that linguistic statements, particularly rules, act as discriminative stimuli over children's motor responses. As with the other primary symptoms, rule-governed behavior represents a multidimensional construct (Zettle & Hayes, 1982). "Pliance" is often used to refer to the immediate initiation of behavior in compliance with a stated rule whereas "tracking" refers to the sustained persistence of compliance over time. Problem-solving refers to that form of rule-governed behavior in which children use self-directed questions to generate rules to follow in situations for which the child has not previously been prepared (Skinner, 1969).

From this perspective, tracking would seem to be the more problematic of these components in ADD children, especially those who are not necessarily oppositional or defiant (Barkley, 1985), although defective problem solving has certainly been demonstrated in these children (Douglas, 1983). Given that most experiments using measures of attention and impulsivity with ADD children involved the assignment of instructions or rules to the children in performing these tasks, such research could be viewed as demonstrating poor rule-governed behavior by ADD children rather than deficits in sustained attention or impulse control. It therefore seems that ADD children are primarily "contingency-shaped" rather than rule-governed in their behavior. As such, they seem more under the influence of immediately available stimuli in the environment than of previously given rules. Because of its recent association with ADD in children, it is not yet clear from research which components of rule-governed behavior are deficient nor whether this is a primary problem or secondary to some other deficiency in the regulation of behavior by its consequences (Barkley, 1989). What is more, it must be acknowledged that acceptance of rule-governed behavior as deficient in ADD children is not yet widespread.

As noted above, all the proposed symptoms of ADD vary to a significant extent as a function of situational factors (Zentall, 1984). Several of these factors have been delineated. First, the degree of "structure," or, more appropriately, the extent to which the setting or responsible caregivers make demands on ADD children to restrict their behavior and adhere to rules, affects the degree of ADD symptoms displayed. In free field or low-demand settings, ADD children may be less distinguishable from normal children than in highly restricted or demanding settings (Barkley, 1985; Jacob *et al.*, 1978; Luk, 1985; Routh & Schroeder, 1976). Second, ADD children appear to be somewhat less problematic for their fathers than for their mothers (Tallmadge & Barkley, 1983; Tarver-Behring, Barkley, & Karlsson, 1985). Third, in settings or on tasks in which instructions are repeated frequently to the child, attention deficits are less noticeable (Douglas, 1980, 1983). Fourth, ADD children seem to have fewer behavioral problems in novel or unfamiliar surroundings but increase their degree of disruptiveness as time in and experience with the setting increase (Barkley, 1977). Finally, settings or tasks that involve a high rate of reinforcement to the child for sustained performance often result in sizeable reductions or even amelioration of the attentional deficits on those tasks in contrast to tasks with minimal, delayed, or no feedback for performance (Barkley, 1989; Barkley, Copeland, & Sivage, 1980; Douglas & Parry, 1983).

Prevalence

Research suggests that between 3% and 5% of the school-age population have ADD (Ross & Ross, 1982). Epidemiological surveys using behavior rating scales have found that between 5% and 7% of boys and 2% to 4% of girls fall approximately 2 standard deviations above the mean on scales assessing inattention and hyperactivity (Trites, Dugas, Lynch, & Ferguson, 1979). Obviously, the criteria for defining the disorder and the extent of agreement across caregivers (e.g., parents and teachers) greatly determine the prevalence figures that are likely to be obtained in any study (Lambert, Sandoval, & Sassone, 1978). More boys than girls are reported to have the disorder, with ratios of 6:1 to 10:1 reported in clinic samples (American Psychiatric Association, 1980) to ratios as low as 3:1 in

epidemiological surveys (Trites *et al.*, 1979). The prevalence of ADD varies slightly across countries (O'Leary, Vivian, & Nisi, 1986; Trites *et al.*, 1979) and socioeconomic strata but occurs to some degree in all cultures and countries (E. Taylor & Sandberg, 1984).

Associated Characteristics

A plethora of research exists to show that ADD children are at greater risk of having numerous other difficulties. Problems with aggression and conduct disorders can be found in up to 70% of clinic samples (Loney & Milich, 1982; Paternite & Loney, 1980). Poor peer relations and deficiencies in social skills have been reported in as many as 50% to 60% (see Campbell, Chapter 7 in this volume; Pelham & Bender, 1982) of ADD children. Deficits in general intelligence, academic achievement, and motor coordination are often described (Cantwell & Satterfield, 1978; Ross & Ross, 1982; Safer & Allen, 1976) as are higher numbers of minor physical anomalies (Quinn & Rapoport, 1974), accidental poisonings, health problems, and injuries (Hartsough & Lambert, 1985; Stewart, Thach, & Freidin, 1970). Although results are often conflicting, studies employing psychophysiological measures tend to show underreactivity to stimulation as the most consistent abnormality noted in ADD as compared to normal children (Ferguson & Pappas, 1979; Hastings & Barkley, 1978).

Given the heterogeneity created in the ADD population by the variation and combination of these associated features, it is hardly surprising that investigators have studied ways of subtyping these children into more homogeneous subgroups. Such subtyping might reveal patterns of differential etiologies, developmental courses and outcomes, or responses to treatments between the subtypes. To date, the most promising subtyping criterion seems to be the presence and degree of aggression and conduct problems (Loney & Milich, 1982). ADD children with aggression are more likely to have peer relationship problems, poorer academic outcomes, greater difficulties with delinquency, and a greater incidence of psychopathology in their parents than ADD children with lesser degrees of hostile-defiant behaviors (Paternite & Loney, 1980; Weiss & Hechtman, 1986). Others have subtyped ADD children on the presence and degree of hyperactivity, as suggested in the DSM-III (American Psychiatric Association, 1980). Results are inconsistent and often conflicting (Carlson, 1986) but suggest that ADD without hyperactivity is associated with greater learning disabilities and social withdrawal, whereas ADD with hyperactivity often correlates with higher levels of aggression, poorer peer interactions, and poorer academic performance (Edelbrock, Costello, & Kessler, 1984; King & Young, 1982). Some (Schachar, Rutter, & Smith, 1981) have shown that ADD children who display their problems at home and at school (pervasive ADD) are more severely impaired than those displaying their problems in only one of these settings, but others have not replicated these results (Cohen & Minde, 1983). Highly anxious and depressed ADD children may be more prone to poor or adverse reactions to stimulant medications than those without such symptoms (Taylor, E., 1983; Voelker, Lachar, & Gdowski, 1983), but such a subtype requires more rigorous investigation before its value as a predictor of treatment response can be accepted.

Developmental Course

It is now well recognized that symptoms of ADD arise early in childhood and that most children will have been identified as deviant from normal prior to 7 years of age (Barkley, Fischer, Newby, & Breen, 1987; Ross & Ross, 1982). Potential predictors in early infancy have been studied, with early infant negative temperament and minor physical anomalies being the most well-researched (Carey & McDevitt, 1978; Quinn & Rapoport, 1974). However, others have not found these to be predictive of ADD specifically but of psychopathology in general (Chamberlin, 1977; Krouse & Kaufman, 1982). By age 3 years, at least half of all ADD children are noted to be difficult to manage and toilet train (Hartsough & Lambert, 1985; Safer & Allen, 1976). Symptoms of ADD in children at this age are highly likely to be stable characteristics of the children, predicting significant behavioral maladjustment at home and at school by age 6 years (Campbell, Schlieffer, & Weiss, 1978). Between 3 and 6 years of age, the ADD symptoms are likely to be at their peak of severity, with defiant and noncompliant behavior a common complaint of their parents (Mash & Johnston, 1982). Parental distress is also likely to be at its zenith, with maternal depression, poor parental self-esteem, and maternal health problems more prevalent than at later ages (Mash & Johnston, 1983).

By age 6 years, the majority of ADD children

have developed their symptoms, with many now beginning to display signs of oppositional, defiant, and aggressive behavior (Ross & Ross, 1982). As noted above, this latter group is more likely to encounter peer relationship problems, poorer academic performance, lower self-esteem, and poorer adolescent prognosis than those ADD children who remain low in these qualities. Academic underachievement most often characterizes their middle childhood years (Cantwell & Satterfield, 1978), along with problems adhering to rules at school and in the community. By now, those likely to have learning disabilities will have manifested these academic delays, further crippling their likelihood of success in school. Those with more emotional lability, defiant and hostile behavior, and lying and stealing during these childhood years are more likely to gravitate toward adolescent conduct disorders and antisocial personality in young adulthood (Paternite & Loney, 1980; Thorley, 1984; Weiss & Hechtman, 1986).

During adolescence, the degree of severity of their primary symptoms (e.g., of inattention and impulsivity) is considerably less than seen in their childhood years but may remain significantly different from normal in the majority of ADD children (Gittelman, Mannuzza, Shenker, & Bonagura, 1985). Those whose only difficulties are in ADD symptoms are often faring better than their conduct disordered ADD peers with their problems primarily limited to underachievement in school (Paternite & Loney, 1980). ADD children displaying aggression and conduct problems will have greater contacts with police, poorer peer acceptance, truancy, drug use, and family conflicts during this stage of development than the purely ADD group (Weiss & Hechtman, 1986). Many continue to report problems with low self-esteem, depression, and lack of friends (Hechtman, Weiss, & Perlman, 1980; Thorley, 1984). It has been estimated that up to 30% will fail to complete high school and, among those who do complete school, the vast majority will fail to pursue any advanced education (Weiss, Hechtman, Milroy, & Perlman, 1985).

What little research their is on young adult outcome (Weiss & Hechtman, 1986) indicates that many are making a better social adjustment than during their adolescence. They are likely to be indistinguishable from other employees in the opinion of their employers, are less likely to have trouble with legal authorities, and report less depression and self-esteem problems. Yet over 60% continue to describe themselves as restless, inattentive, and impulsive and report more feelings of depression and low self-esteem than non-ADD young adults. Between 25% and 45% may receive a diagnosis of Antisocial Personality based on continued aggression, conduct problems, and criminal behavior (Gittelman *et al.*, 1985; Loney, Whaley-Klahn, Kosier, & Conboy, 1981; Weiss & Hechtman, 1986). Over one fourth may be alcoholic, but this seems best predicted by previous conduct disorders rather than a history of ADD symptoms (Loney *et al.*, 1981). Clearly, ADD and especially conduct disorder are likely to predispose children to a greater likelihood of adult psychopathology and maladjustment.

Current Diagnostic Criteria

Until 1980, there was little, if any, criteria available to assist clinicians in determining which children in their practices should actually be diagnosed as having ADD, or any of its ancestral labels. As a result, many clinicians developed their own idiosyncratic guidelines for rendering the diagnosis. This led not only to great confusion in the field but also great frustration among parents whose children received diverse diagnostic labels from different clinicians. Those guidelines available in the DSM-II (American Psychiatric Association, 1968) were more vague descriptions than explicit criteria and so proved of no value in clarifying the issue for practitioners. Clinicians continued to agree in principle as to what symptoms constituted the disorder but showed little agreement as to what criteria they actually used in rendering the diagnosis (Barkley, 1982; Schrager, Lindy, Harrison, McDermott, & Wilson, 1966).

With the revision of the DSM in 1980 (DSM-III), more explicit guidelines were set forth for clinical diagnosis and they rapidly became the standard for the field. The disorder was now called Attention Deficit Disorder with or without Hyperactivity in place of Hyperkinetic Reaction of Childhood. Summarized briefly, these new criteria stipulated that a child have developmentally inappropriate inattention (has 3 of 5 descriptors) and impulsivity (3 of 6 descriptors), with an age of onset of problems before 7 years and a duration of symptoms of 6 months. Diagnoses of schizophrenia, affective disorder, or severe mental retardation precluded the diagnosis of ADD. Children meeting these criteria were labeled Attention Deficit Disorder *without Hyperactivity*. An additional 5 descriptors were provided under the symptom of Hyperactivity.

Those children having at least 2 of these 5 descriptors were than labeled as Attention Deficit Disorder *with Hyperactivity*. This is the first time formal subtypes of ADD have been proposed, despite the fact that little research support for this subdivision existed at that time (see description above).

Although they were a laudable step in the direction of providing more explicit criteria for ADD, these guidelines were problematic on several counts. First, the descriptors provided with each symptom do not necessarily pertain to that symptom. For instance, "Needs a lot of supervision" is assigned as a descriptor of impulsivity when it could actually refer to the other symptoms of ADD as well. Second, the cutoff scores for the number of descriptors needed for each symptom were not empirically established. Hence, it is difficult to say how many normal children would meet these cutoff points. This leads to a third problem, and that is that no developmentally appropriate reference points are provided in making these decisions, such as meeting a statistical criterion on some measure of ADD symptoms. Although it is stated that the child's symptoms should be developmentally inappropriate, it is not indicated how the degree of deviance from normal is to be established or what that specific degree for establishing abnormality is to be. And fourth, the age of onset and duration of symptoms were also not established in any empirical way.

Fortunately, some of these problems have been addressed in the recent revision of the DSM-III (DSM-III-R, American Psychiatric Association, 1987). These new criteria appear in Table 1. The disorder is now referred to as Attention Deficit-Hyperactivity Disorder (ADHD). Rather than three separate lists of descriptors, one for each symptom, there is now a list of 14 descriptors. These have previously been shown to cluster together in some factor-analytic studies (Achenbach & Edelbrock, 1983; Trites, Blouin, & Laprade, 1982). The cutoff score will now be 8 of 14. This was established during a clinical field trial in which a large sample of children having various diagnoses were studied using these descriptors, and 8 of the 14 appeared to produce the best classification rate. No subtyping of ADD into with or without Hyperactivity is provided due to conflicting research findings on the utility of such a subtyping. Pervasive Developmental Disorder is now the only exclusionary condition, whereas

Table 1. Diagnostic Criteria for Attention Deficit Disorder

Note. Consider a criterion met only if the behavior is considerably more frequent than that of most people of the same mental age.

A. A disturbance of at least 6 months during which at least 8 of the following are present:
   1. Often fidgets with hands or feet or squirms in seat (in adolescents, may be limited to subjective feelings of restlessness)
   2. Has difficulty remaining seated when required to do so
   3. Is easily distracted by extraneous stimuli
   4. Has difficulty awaiting turn in game or group situations
   5. Often blurts out answers to questions before they have been completed
   6. Has difficulty following through on instructions from others (not due to oppositional behavior or failure of comprehension), for example, fails to finish chores
   7. Has difficulty sustaining attention in tasks or play activities
   8. Often shifts from one uncompleted activity to another
   9. Has difficulty playing quietly
   10. Often talks excessively
   11. Often interrupts or intrudes on others, for example, butts into other children's games
   12. Often does not seem to listen to what is being said to him or her
   13. Often loses things necessary for tasks or activities at school or at home (e.g., toys, pencils, books, assignments)
   14. Often engages in physically dangerous activities without considering possible consequences (not for purpose of thrill-seeking), for example, runs into street without looking

Note. The above items are listed in descending order of discriminating power based on data from a national field trial of the DSM-III-R criteria for Disruptive Behavior Disorders.

B. Onset before the age of seven

C. Does not meet the criteria for a Pervasive Developmental Disorder

Note. From the *Diagnostic and Statistical Manual for Mental Disorders* (3rd ed., rev.), 1987. Washington, DC: American Psychiatric Association.

age of onset and duration of symptoms remain the same as in the DSM-III. As a result, some problems still remain with these criteria, such as the lack of empirical validation for the onset and duration criteria. Nevertheless, clinicians should find these guidelines more useful than those previously available.

I have elsewhere described a set of working guidelines to serve as research criteria for ADD in children (Barkley, 1982). Briefly, these included the following: (1) parent and/or teacher complaints of poor sustained attention, impulsivity, restlessness, and difficulties with rule-governed behavior (e.g., following instructions); (2) behavior ratings of these symptoms on a well-standardized parent or teacher rating scale which place the child two standard deviations above the mean for same age and sex normal children; (3) an age of onset by 6 years; (4) a duration of symptoms of 12 months; (5) problems with these symptoms in at least 50% of the settings on the Home or School Situations Questionnaires (Barkley & Edelbrock, 1987) to establish cross-situational pervasiveness of symptoms; (6) an IQ score greater than 70 or, if below 70, comparison to children of the same mental age in criteria (2) above; and (7) exclusion of deafness, blindness, gross brain damage, autism, psychosis, or severe language delay as primary causes of the symptoms. Such criteria have the advantage of including some indication of how abnormality of symptoms is to be demonstrated and incorporate the further requirement that symptoms be situationally pervasive to a significant degree. Adjustment for generalized delays in mental development is also made to control for the likely deficiency of attention and impulsivity resulting from such delays.

Summary

The history of ADD in children has been shown to be quite long and complex, filled with numerous changes not only in diagnostic terminology, but also in which symptoms define the disorder and in its etiologies. The disorder is now conceptualized as a developmental delay in sustained attention, impulse control, regulation of activity level to situational demands, and, in some views, rule-governed behavior. These symptoms tend to have an onset in early childhood, or earlier, to be chronic over development in most cases, and to be significantly pervasive across most, but not all, situations. Speculation now exists that these symptoms may stem from more fundamental deficits in the manner in which rules influence behavior or in the way in which consequences regulate sustained responding in children. ADD in children is quite prevalent and is often associated with a number of other developmental, academic, social, and familial problems. More boys tend to manifest these problems than girls. Although the primary symptoms are chronic, lasting into young adulthood in many cases, they tend to predict only problems in academic functioning in children without associated aggression or conduct problems. Where these other characteristics are present, young adult prognosis may be poorer with delinquency, antisocial personality, poor academic attainment, and poor employment functioning being more common than in purely ADD children. Over time, more explicit and empirical criteria for diagnosing the disorder have been set forth thus allowing for more homogeneous groups of children with ADD to be studied and the issue of subtyping to be explored.

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CHAPTER 7

The Socialization and Social Development of Hyperactive Children

Susan B. Campbell

Introduction

Although much work has focused on the definition, course, and treatment of hyperactivity and related behavior disorders in children (see Campbell & Werry, 1986; Ross & Ross, 1982, for recent reviews), surprisingly little attention has been paid to the social development and socialization of hyperactive children (but see Whalen & Henker, 1985). This partly reflects the once-prevalent view that hyperactivity (or minimal brain dysfunction as it was called) resulted from cerebral injury and that socialization was not particularly relevant to an understanding of the problem (see Werry, 1986). A second reason for the relative neglect of this issue is the lack of a developmental focus in most research on childhood disorders.

With the adoption of DSM-III criteria (American Psychiatric Association, 1980), the definition of hyperactivity has been systematized, resulting in more comparability across studies. These criteria limit the disorder to specific core features with predefined inclusion and exclusion criteria. However, the DSM-III does not address developmental manifestations of symptomatic behavior adequately, making the diagnosis of hyperactivity in young children problematic (Campbell, 1985), nor does it include specific indicators of social problems (Whalen & Henker, 1985). Despite these drawbacks, I will be using the DSM-III definition of Attention Deficit Disorder (ADD) as my working definition of hyperactivity, a term I prefer because it has a less pathological and medical ring and is less unwieldy. The term *hyperactivity* will be used to denote children referred for help because of inattention, impulsivity, and overactivity, whose problems were apparent before age 7, and were sufficiently chronic to rule out an acute reaction to a specific stressful event. This diagnosis applies only to children of at least low average intelligence, without obvious evidence of brain damage.

Although this chapter will focus on children with a clearly defined cluster of behaviors, much of the work that I will be citing has been conducted on nonclinical samples with higher than average levels of activity, inattention, and/or impulsivity according to parent or teacher reports. Thus, I will be extrapolating from studies of nonclinical groups; similarly, almost all the developmental research with implications for the socialization of hyperactive children has been conducted on nonreferred samples. However, it will be made clear throughout

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when I am referring to clinical and when to nonclinical samples. Finally, because hyperactive boys outnumber hyperactive girls by a wide margin (Ross & Ross, 1982), much of the work to be cited will focus on boys; only a few studies have recruited sufficient numbers of hyperactive girls to analyze sex differences.

In addition, research on clinical samples is rarely conducted on "pure" cases of hyperactivity. Much work has focused recently on the differentiation between disorders of attentional and impulse control and disorders of conduct (e.g., Milich, Loney, & Landau, 1982; Sandberg, Wieselberg, & Shaffer, 1980), and there has been considerable debate about whether these are distinct disorders (Milich, Loney, and Landau 1982; Rutter, 1983) or merely different facets of the same disorder with deficits in self-regulation at their core (Gorenstein & Newman, 1980). This debate is far from resolved. In any event, most studies of hyperactive children include some unknown proportion with symptoms of oppositional or conduct disorder as well; that is, some children with a diagnosis of hyperactivity will be highly aggressive and noncompliant as well as inattentive and impulsive.

Although studies do suggest that school-aged youngsters with externalizing symptomatology may be primarily hyperactive, primarily aggressive, or both hyperactive and aggressive (e.g., Milich, Loney, and Landau, 1982), these diagnostic distinctions are more difficult to make in preschool-aged children. Indeed, the few studies that have examined these behaviors in preschoolers indicate that there is a marked overlap between symptoms of hyperactivity and symptoms of aggressive and uncooperative behavior in unreferred samples (Buss, Block, & Block, 1980; Crowther, Bond, & Rolf, 1981) and in referred samples (Campbell, Szumowski, Ewing, Gluck, & Breaux, 1982; Schleifer, Weiss, Cohen, Elman, Cvejic, & Kruger, 1975). Thus, for example, Campbell *et al.* (1982) noted that it was difficult to identify toddlers and preschoolers whose parents complained of overactive, distractible, and impulsive behaviors in the absence of complaints about discipline problems and aggression toward siblings and peers. Longitudinal studies of young children also indicate that these behaviors have similar antecedents, correlates, and outcomes in early childhood (e.g., Bates, Maslin, & Frankel, 1985; Campbell, Breaux, Ewing, & Szumowski, 1986; Fischer, Rolf, Hasazi, & Cummings, 1984). These data are somewhat at variance with follow-up studies of older hyperactive children that suggest that hyperactive and aggressive behavior may have different antecedents and outcomes (e.g., Milich & Loney, 1979; Paternite & Loney, 1980). This may mean that hyperactivity and aggression have different developmental courses, that salient symptoms vary as a function of developmental level, or that hyperactivity and conduct disorder develop from a basic cluster of externalizing symptoms, with different environmental factors influencing outcome.

With these caveats in mind, I will bring together literature from developmental psychology and child psychopathology that has a bearing on the social development of active, impulsive, and inattentive children, some of whom might reach clinical attention at some point in their lives and some of whom would be likely to experience academic and social problems, even if they were never actually referred for mental health services. First, I examine the relationship between crucial developmental tasks and the symptoms of hyperactivity. Then, I will address family and peer group influences on the socialization of hyperactive children.

Developmental Tasks and Hyperactivity

Wenar (1982) has suggested that psychopathology in children can best be conceptualized as "normal development gone awry." It is instructive, therefore, to examine the core features of hyperactivity from a developmental perspective, because symptomatic behaviors may also be construed as exaggerations of normal developmental phenomena.

Among the major goals of early infancy are the development of state control and self-soothing ability, the establishment of routines, the modulation and coordination of motor activity, and the ability to focus attention on and to begin to derive meaning from environmental events (Kagan, 1984; Kopp, 1982; Sroufe, 1979). It is generally agreed that these early acquisitions are under strong biological/maturational control (Kagan, 1984; McCall, 1981), although the role of the caretaking environment is also seen as central to the successful negotiation of these developmental challenges (Campos, Barrett, Lamb, Goldsmith, & Stenberg, 1983; Kopp, 1982; Sroufe, 1979). In addition, individual differences in infant activity level, alertness, regularity, and self-soothing behavior have been well documented (Korner, 1971; Thomas, Chess, & Birch, 1968). Evidence suggests that infants who are particularly difficult to care for by virtue of their

unpredictable routines, high activity level, irritability, and unconsolability are at risk to develop later problems including hyperactivity (Bates *et al.*, 1985). It is impossible, at this stage of our knowledge, to predict when a difficult infant is showing early signs of a persistent and potentially serious problem and when she is merely having difficulty adapting to environmental demands and developmental expectations. The irritable and irregular 3-month-old may emerge into a smiling and content 6-month-old, as developmental hurdles are passed over and the nervous system matures; or the fussiness and unpredictability may persist, interfering with the establishment of positive parent–child relations and the exploration of the world of objects.

By the end of the first year or the beginning of the second, most children begin to walk and to use rudimentary language skills to communicate. These advances in motor and language abilities usher in a period of rapid cognitive, affective, and social development. The hallmarks of this period include exploration and mastery of the environment and the development of autonomy and independence. At this developmental juncture children often practice their developing locomotor skills as well. Thus, it is not unusual for toddlers to appear excessively active. Their emerging motor skills paired with their tendency to touch and explore everything in sight may give their behavior a driven, frenetic, and inattentive quality, which, though perfectly normal, alarms some parents. The result is often an increase in parental prohibitions that elicit noncompliance on the part of the toddler who is attempting to establish his or her own sense of limits and autonomy. It is obvious that the developmental tasks of toddlerhood and the early symptoms of hyperactivity have many features in common. Thus, it is often difficult to discriminate between the active toddler who is eagerly exploring the environment and asserting his or her identity and the overly active, inattentive, and defiant toddler whose frenzied exploration actually interferes with mastery and active problem solving, and whose search for independence degenerates into tantrums, frustration, and anger.

By the preschool period, new expectations for self-regulation and cooperation in the peer group are added to the continuing consolidation of exploration, mastery, and the establishment of independence (Kopp, 1982; Sroufe, 1979). Children are expected to be able to focus attention for sustained periods in order to complete tasks, whether playing alone or with others; they are expected to begin to share, negotiate, and take turns; and they are expected to monitor their own behavior to some degree in the absence of adult prohibitions and reminders. However, it is not unusual for young preschoolers to have difficulty amusing themselves, sharing toys and taking turns, or regulating their own behavior. Despite the gains made in self-regulation, parents and teachers alike complain frequently of management difficulties, inattention, and overactivity in representative samples of 3- and 4-year-olds (e.g., Crowther *et al.*, 1981; Richman, Stevenson, & Graham, 1982). These complaints may reflect inappropriate adult expectations, or these behaviors may be exaggerations of normal developmental transitions, which are transient though annoying, or early signs of more severe and persistent difficulties with behavioral control. Although the severity, frequency, and constellation of problematic behaviors as well as their chronicity will determine whether one is dealing with a clinically significant problem or merely a transient phenomenon, studies delineating predictors of outcome in young children have been rare.

Most children diagnosed as hyperactive come to clinical attention when they reach school age and cannot cope with the demands of the classroom (Ross & Ross, 1982). At this developmental stage, children are expected to master a range of cognitive tasks in the structured and controlled school environment. They are expected to sit still for long periods of time and to focus attention on tasks prescribed by others. In the peer group, they are expected to follow group norms, to negotiate to solve disputes, to begin to take the perspective of the other, and to form stable friendships that move beyond the here and now. In the family, school-aged youngsters are expected to participate in family routines and to engage independently in a number of self-care activities and daily chores. All these behaviors require organization and self-regulation, cooperation with others, attention to task demands and to social cues, and the ability to put group goals ahead of individual desires and impulses. In short, by early elementary school age, children are expected to be well enough socialized to be contributing members of their peer group and family.

It is at this developmental stage that the differentiation between hyperactive children and their peers becomes most clear-cut, for in all of these tasks, hyperactive children appear to have difficulty. Thus, their behavior at home is often characterized by carelessness and disorganization, inability to follow routines (completing homework, putting away toys, feeding the family dog) on their own, noncompliance with parental requests and ex-

pectations, and problems cooperating with siblings. In school, hyperactive children are likely to perform poorly academically, not to complete assignments, and to be careless, messy, and disorganized in their work. During structured classroom activities, they are likely to daydream or to call out inappropriately. Hyperactive children are also likely to get into squabbles on the playground. During group activities, they are the ones most likely to instigate a fight or to disrupt the ongoing game, and to be rejected by peers because of this inappropriate and provocative behavior. Thus, the core and associated features of hyperactivity lead to pervasive social difficulties at home, at school, and in the peer group. Indeed, it is the cross-situational and chronic nature of these problems in social and cognitive functioning that lead to referral and are the hallmark of the diagnosis.

The general failure in socialization that characterizes the school-aged hyperactive child becomes more serious in adolescence when peer problems become particularly salient. Antisocial and delinquent behavior may also develop or become apparent. It is to socialization influences that I now turn.

Socialization Influences

Parental Responsiveness and Childrearing Practices

There is a large body of evidence emphasizing the importance of qualitative features of early maternal care (e.g., Campos *et al.*, 1983; Sroufe, 1985). Appropriately timed parental interventions and parental warmth and sensitivity have been shown to influence concurrent infant behavior and to predict the quality of the infant–mother attachment at 12 months. Theorists have stressed the important role the mother plays in early infancy in establishing smooth caretaking routines, facilitating state control, and protecting the young infant from overstimulation (e.g., Campos *et al.*, 1983; Kopp, 1982; Sroufe, 1979). Even in the neonatal period, however, maternal competence will be influenced somewhat by individual differences in infants; some infants, as noted earlier, will be easier to care for than others as reflected in greater adaptation to feeding and sleeping schedules, less fussiness, and greater consolability when distressed (e.g., Korner, 1971). Sroufe (1985) has argued that sensitive and responsive mothering in early infancy can overcome individual differences in infants, and there is some support for this perspective (e.g., Belsky, Rovine, & Taylor, 1984). Studies also suggest that infant characteristics can influence maternal caretaking (e.g., Campbell, 1979; Osofsky & Danzger, 1974), and that the relationship between infant characteristics and caretaking style is best seen as a reciprocal one (Maccoby & Martin, 1983; Thomas *et al.*, 1968), with both members of the dyad contributing to the nature of the interaction and its changes over time (Sameroff & Chandler, 1975).

If, as has been suggested by prospective (Bates *et al.*, 1985) and retrospective (Campbell *et al.*, 1982) data, hyperactivity in toddlers and preschoolers is associated with a more difficult infancy period, there is reason to suppose that negative and conflicted mother–infant transactions may begin quite early. Bates, Freeland, and Lounsbury (1979) define infant difficultness in terms of intensity of protest, fussiness and irritability, and unconsolability. Although some particularly flexible and responsive mothers will undoubtedly be able to cope adequately with such babies (Sroufe, 1985), other women, especially those coping with more stress, will be less likely to respond appropriately and patiently to irritable and demanding infants (Crockenberg, 1981; Vaughn, Egeland, Sroufe, & Waters, 1979). For example, difficult infants may be more likely to be left to cry themselves to sleep, although they may be just the infants who require more adult intervention in view of their own less developed self-soothing abilities. They may also require relatively more structured routines and protection from overstimulation than less difficult infants.

Attachment theorists, such as Sroufe (1985), argue that the lack of appropriate maternal intervention in situations such as this will contribute to continued infant irritability and ultimately to the infant's assessment of the world as unresponsive and rejecting. As a result of this lack of maternal responsiveness and sensitivity, the infant does not expect to be comforted when distressed and eventually learns to avoid or resist contact with the mother. The absence of a secure attachment to the mother, as reflected in the inability to derive comfort from her, in turn, is seen as leading to the development of behavior problems (Sroufe, 1983); that is, the relationship between infant difficultness and later behavior problems is mediated through maternal caretaking and the quality of the attachment relationship that develops subsequently. Other theorists argue that the interaction or transaction between infant irritability and maternal behav-

ior is crucial to an understanding of the development of problems and, with some especially difficult infants, even the most sensitive caretakers may prove ineffective (e.g., Campos *et al.*, 1983; Maccoby & Martin, 1983; Thomas *et al.*, 1968). Thus, it appears theoretically logical to hypothesize links among infant irritability, lack of effective maternal response, and later behavior problems including hyperactivity, despite debates on relevant mediating factors.

The relationship between childrearing practices and child behavior begins to crystallize into even clearer patterns by toddlerhood and the early preschool years. First, the quality of the earlier mother–child attachment is associated with greater child compliance, enthusiastic and persistent problem solving, and sociability with peers (e.g., Londerville & Main, 1981; Matas, Arend, & Sroufe, 1978; Pastor, 1981). In addition, mothers of securely attached toddlers, consistent with their history of sensitive and responsive mothering during the first year of their infants' lives, are more supportive and positively involved during problem solving and peer interaction. They are also less likely to set arbitrary or punitive limits on their toddlers who, in turn, are more responsive to gentle prohibitions. Other studies of maternal control and child compliance indicate that most children are compliant most of the time. Further, studies conducted in the lab, the home, and the natural environment indicate that maternal control strategies that are positive, including anticipating trouble, diverting or redirecting attention, providing choices and explanations, and suggesting rather than demanding are associated with more child compliance, whereas physical restraints and negative prohibitions are more likely to elicit noncompliance as well as power struggles (Holden, 1983; Lytton, 1980; Minton, Kagan, & Levine, 1971; Schaffer & Crook, 1980). The ability to ignore minor infractions of household rules in favor of more serious violations also appears to be important.

Zahn-Waxler, Radke-Yarrow, and King (1979) studied maternal responses to toddler's transgressions, such as physical aggression or taking a toy, that caused distress in others. In such situations, mothers engaged in teaching and explaining in order to emphasize the relationship between the child's behavior and the victim's distress. Further, these explanations were often expressed with intense affect that conveyed the seriousness with which the mother viewed the transgression; these mothers also communicated expectations that their children would understand why the behavior was wrong, thereby pressuring children to take responsibility for their own behavior and highlighting the links between the behavior and its consequences. In general, prohibitions that included an explanation were correlated with more prosocial behavior and reparations for harm caused to others, whereas unexplained verbal prohibitions were associated with low levels of prosocial behavior. Affectively toned verbal prohibitions and explanations were also associated with a history of mother's empathic caregiving, which not only was a salient quality of the mother–toddler relationship, but also served a modeling function, demonstrating care and concern for the feelings and rights of others.

Taken together, these studies of nonclinical samples of mother–toddler dyads indicate that limit-setting is more effective when it occurs in the context of a positive mother–child relationship and provides educative guidance and clear expectations rather than punitive and apparently arbitrary rules. In Baumrind's (1967) terminology, discipline is more effective when it is authoritative rather than authoritarian or power assertive (see Maccoby & Martin, 1983). These findings also suggest that negative and power-oriented disciplinary practices are particularly likely to trigger defiant and resistant behavior in toddlers who are attempting to establish autonomy and self-regulation. Limit-setting that is seen to be routinely angry and impatient in tone and that consistently communicates displeasure is likely to elicit anger and aggression, even in young children (e.g., Cummings, Zahn-Waxler, & Radke-Yarrow, 1984), who appear sensitive to affective communication at an early age and tend to mirror the affective displays of caregivers, as well as model other aspects of behavior. Thus, consistent with Maccoby and Martin (1983) and Sroufe (1979), the nature of the mother–child relationship over time, particularly its affective tone, appears crucial to effective socialization.

Several studies have examined disciplinary encounters in children who are identified as difficult or highly active (though not clinically so). Lee and Bates (1985) reported that toddlers who were rated as difficult to handle in infancy were more likely to ignore household rules and test limits, and that their mothers were more likely to engage in power-assertive disciplinary techniques which, in turn, led to further noncompliance. Buss (1981) found that active preschoolers and their parents became engaged in more frequent power struggles during a laboratory teaching situation than their less active counterparts; mothers of more active youngsters were also less responsive to their preschoolers'

needs during task solution and less able to convey clearly the nature of the tasks.

Campbell, Breaux, Ewing, Szumowski, and Pierce (1986) examined the mother–child interactions of parent-referred active and defiant preschoolers and nonproblem controls during an undemanding play interaction. Dyads were observed first when children were aged 3 and again 1 year later. At age 3, mothers of problem youngsters tended to redirect play activities more often than control mothers; at age 4, mothers of problem youngsters made more negative control statements than mothers of comparison children, although mothers in both groups decreased their involvement overall. Problem youngsters were more active than controls during the play session at age 4 and they also tended to engage in more aggressive play. This is consistent with Bell's (1968) notion of upper limit controls and may suggest that when more benign forms of control, such as refocusing attention, do not appear to be effective in decreasing high-intensity behavior, mothers engage in stronger control attempts, which also become more negative.

Mash and Johnston (1982) found that preschool-aged, clinically diagnosed hyperactive children were more negative and noncompliant during free play and structured tasks than nonhyperactive age-mates or older hyperactive children, and their mothers were more directive and less rewarding of independent play or compliance. Other studies of clinical samples of young hyperactive children confirm that a negative flavor characterizes their interactions with their mothers (Barkley, 1985; Cohen & Minde, 1983; Prior, Leonard, & Wood, 1983). Patterson (1980) has conducted observations in the homes of families with aggressive preschoolers, many of whom are probably also hyperactive, and his findings highlight the reciprocity inherent in these aversive interactions as well as the child's typical role as initiator.

A number of studies of clinical samples of school-aged hyperactive children and their mothers have been conducted in laboratory settings in which task difficulty or degree of structure is varied. Findings consistently indicate that, relative to nonclinical controls, hyperactive children are less compliant and more negative whether observed during free play or structured tasks (e.g., Barkley, 1985; Campbell, 1973; Cunningham & Barkley, 1979; Mash & Johnston, 1982). However, their mothers are likewise more negative and controlling, both in response to their children's noncompliance *and* compliance. Furthermore, the mothers of hyperactive children and the children themselves appear to find interacting, even during play, less rewarding than do comparison dyads. Hyperactive children initiate less interaction with their mothers during observations of free play than control children and when they do, their mothers are less responsive than comparison mothers (Barkley, 1985). These findings have been replicated in several studies that highlight group differences and situational effects: although mothers and their hyperactive children engage in more conflict even during free play, the amount of conflict and its intensity appear to escalate during structured tasks that require the children to focus attention and monitor their own behavior in response to situational demands and also place more pressure on mothers to gain their children's compliance. Patterson's (1980) observations of troubled families suggest that both members of the dyad escalate the intensity of the confrontation, with children escalating more rapidly, for example, from whining to tantruming; however, mothers appear to respond negatively to the aversive and the prosocial behaviors of problem children, thus contributing to the angry tone of the relationship as well as to the confusion about what behaviors are acceptable.

In one study, the interactions of hyperactive children with fathers and mothers were examined. Tallmadge and Barkley (1983) found that hyperactive children were less likely to be noncompliant with their fathers than with their mothers during structured tasks, but these differences were slight and far less dramatic than parental reports and clinical impressions would suggest. Mothers and fathers alike tend to report that their hyperactive youngster is more likely to disobey maternal requests. Other studies confirm that children more readily comply with fathers (Patterson, 1980) and that they are more willing to comply with mothers when fathers are present (Lytton, 1980). Patterson (1980) suggests that in normal families mothers are the principal caretakers, although fathers contribute to child management both directly and indirectly by supporting the mother in her disciplinary efforts. However, in families with children who are difficult to control, mothers function as "crisis managers," whereas fathers withdraw to avoid dealing with unpleasant situations. These findings are consonant with the complaints of many mothers of hyperactive children who report feeling isolated and demoralized by their lonely battles for compliance and control (Barkley, 1981; Mash & Johnston, 1983). Thus, the psychological unavailability of

some fathers of hyperactive children, even in two-parent families, may contribute to the intensity and duration of mother–child conflict and to more general problems in socialization.

Researchers have also examined age differences in mother–child interaction using both cross-sectional and longitudinal designs. The cross-sectional studies have been consistent in finding that mothers and their younger hyperactive children engage in more conflict around compliance than older hyperactive child–mother pairs (Barkley, Karlsson, Pollard, & Murphy, 1985; Mash & Johnston, 1982). In a study of clinically hyperactive children, Barkley (1985) reported that mothers of younger hyperactive children gave more commands than mothers of older hyperactives, but these commands were also more likely to go unheeded, setting up a cycle of frequent comments that were increasingly likely to be ignored. However, even when hyperactive youngsters complied with commands, mothers often gave additional commands and responded negatively rather than positively to the compliance, consistent with the view that expectations of noncompliance rather than the specifics of ongoing behavior influenced maternal responses.

The longitudinal data of Campbell, Breaux, Ewing, Szumowski, and Pierce (1986), alluded to earlier, indicated that mothers of both problem youngsters and controls intervened less when their children were aged 4 than they had a year earlier, presumably in response to their children's advances in cognitive functioning, their more organized play, and their improved attention. Although the behavior of mothers changed, relative to their own initial levels, mothers of problem children made more frequent negative control statements than mothers of comparison children. Barkley *et al.* (1985) noted that hyperactive children appear to show a developmental spurt in their ability to remain on task and to comply with requests around age 6; that is, they show improvement in behavioral control relative to younger hyperactives. Similar improvements are found in comparison dyads (Barkley, Karlsson, & Pollard, 1985), indicating that although group differences continue to be in evidence across the age range studied, hyperactive children and their peers show developmental changes in behavior that suggest improved self-regulatory abilities (Kopp, 1982) at a time when the transition to school requires greater independence and self-control.

Several attempts have been made to disentangle the direction of effects by examining changes in mother–child interaction in hyperactive children treated with stimulant medication (Barkley *et al.*, 1985; Barkley, Karlsson, Strzelecki, & Murphy, 1984). In general, hyperactive children on medication become more compliant and their mothers become less directive and less negative. The medication effects may be interpreted as "normalizing" the interaction because hyperactive children and their mothers behave more like normal comparison dyads when children are on active medication as compared to placebo. From a systems or reciprocal interaction perspective, it is not surprising that the interaction changes in response to changes in the behavior of one member of the dyad. Although these data indicate that mothers respond to changes in their children, it is also possible that the children would likewise change, possibly becoming less noncompliant, if their mothers were less demanding. Thus, these findings demonstrate that the direction of effects can be from child to mother (Bell, 1968), but they do not demonstrate that this is necessarily the case, nor do they indicate how the negative cycle of interaction initially developed.

Follow-up studies using representative samples as well as groups of referred children reveal that mother–child conflict predicts the persistence of problems several years later. Richman *et al.* (1982) found that maternal reports of parent–child conflict obtained at age 3 were associated with ratings of behavior problems 5 years later. Campbell, Breaux, Ewing, and Szumowski (1986) observed mother–child interaction in a sample of parent-referred hard-to-manage 3-year-olds and controls. A composite measure of observed negative maternal control predicted maternal ratings of hyperactivity and aggression at age 6. Studies following hyperactive school-aged children into adolescence have produced similar results (Paternite & Loney, 1980; Weiss, 1983). A relationship between parent–child conflict and persistent problems also has been obtained in follow-up studies of aggressive (Olweus, 1980) and delinquent adolescents (Loeber, 1982).

This pattern of interaction is likely to emerge as a result of child noncompliance and maternal exasperation and impatience, rather than as a response to situation-specific contingencies (Lytton, 1980; Maccoby & Martin, 1983), and the negative affective tone of the relationship probably contributes to its escalating angry and aversive quality. The finding that mothers of problem youngsters often respond punitively to compliant and prosocial behavior (Barkley *et al.*, 1985; Patterson, 1980) is consistent with the view that maternal expectations

and affect play a larger role in determining the nature of the interaction than specific child behaviors. As Maccoby and Martin (1983) have suggested, qualitative aspects of the affective relationship as well as a history of negative encounters probably contribute to the persistence of parent–child conflict that, in turn, interferes with the internalization of self-regulatory behavior and limits the child's willingness to put family goals over individual ones.

This body of data examining mother–child interaction from early infancy through preadolescence, focusing on normal children, hard-to-manage children, and clinically diagnosed children, who were studied in laboratory and in natural settings, emphasizes the dual roles of mothers as caretakers and socialization agents. By the end of the first year, as children begin to develop symbolic thinking, a sense of self, and the ability to act on their environment in effective ways, the responsibilities of the parents shift from one of primarily providing nurturance and protection to one in which nurturance must be balanced by limit-setting and the use of effective control strategies. At this developmental stage, parents must begin to confront the difficult but rewarding task of teaching children the rules of appropriate behavior, of helping them to master the rules of social exchange, and facilitating the internalization of values and morals.

Maccoby and Martin (1983) have suggested that some children are easier to socialize than others. In the case of hyperactive children, clinical wisdom and common sense suggest the need for firm, reasonable, consistent, and predictable limit-setting, clear expectations for what constitutes acceptable behavior, and parental agreement on child-rearing strategies. These are the *sine qua non* of Baumrind's authoritative parenting, a style that may be reasonably easy to adopt with nonproblem children. But maintaining sufficient composure and patience with hyperactive children would understandably be more difficult, particularly in toddlerhood and the preschool years when parent–child conflict is especially likely to escalate. Zahn-Waxler *et al.* (1979) have suggested that high-affective involvement also may be important to the internalization of certain moral values. Although this may be the case with "normal" children, hyperactive children who show poor self-regulation of emotion and behavior may attend to and more readily process reprimands and reasoning that are firm, but not affectively charged, because high-intensity emotion may distract them from the intent of the parental communication and increase their arousal beyond an optimal level, continuing a cycle of escalating negative affect.

Family Climate

Not surprisingly, parent–child conflict rarely occurs in isolation. Numerous studies indicate a relationship between a disturbed family environment and children's problems (see recent reviews by Emery, 1982; Hetherington & Martin, 1986). In general, childhood problems, especially externalizing problems in boys, are associated not only with parent–child discord but also with marital distress, separation and divorce, parental psychopathology, maternal depression and general malaise, and more general family stresses including unemployment and poor housing. The nature of these relationships is undoubtedly complex. Many studies utilize mothers as the major data source, thereby confounding reports of environmental stress and adversity with reports of children's symptoms (e.g., Richman *et al.*, 1982). In addition, most studies rely on checklists to assess children's problems, and, therefore, findings are not examined in terms of specific disorders. Problems of data interpretation are compounded further: because many of these indicators of family climate are highly intercorrelated with each other as well as with methods of childrearing, the impact of any one factor on children's behavior can rarely be considered in isolation. Finally, family stresses may have a direct impact on the child as well as an indirect one, mediated through the impact of the particular stress on the parents and on their availability to fulfill their parenting functions. Since these issues have been addressed in several recent publications (e.g., Belsky, 1984; Emery, 1982; Hetherington & Martin, 1986; Parke & Slaby, 1983; Richman *et al.*, 1982), I will focus primarily on the few studies that have examined the family environments of hyperactive children.

Sibling Conflict

Mash and Johnston (1983) examined the interaction of dyads of diagnosed hyperactive boys and their brothers and a group of normal sibling pairs during free play and structured tasks. Few significant differences were found within sibling pairs. However, hyperactive-sibling dyads engaged in more reciprocal conflict than comparison dyads. Moreover, high rates of negative behavior in the hyperactive-sibling pairs were associated with ma-

ternal reports of less skill and knowledge of parenting, suggesting that sibling conflict may have contributed to mothers' negative self-evaluations. This is the only study to assess the relationships of hyperactive children with family members other than parents, but the results are consistent with parental reports of high rates of sibling conflict. The role of the hyperactive child in initiating or maintaining the conflict is not clear from this study and probably varies somewhat with the relative age of the diagnosed child and his sibling. For example, it is reasonable to suppose that older hyperactive youngsters are more aggressive and bullying of their younger siblings, whereas younger hyperactives probably provoke and irritate older sibs. Patterson (1980) has noted that in families with aggressive children, both target children and their siblings initiate and escalate high-intensity conflicts, although target children generally function at higher levels of anger and aggression than their siblings. The few studies of sibling relations in unreferred samples suggest that age differences influence the nature of interaction patterns (Abramovitch, Corter, & Lando, 1979) and that sibling conflict is a normal aspect of family life (Dunn & Kendrick, 1982). However, one can assume that conflict between sibs is both more frequent and intense in families with a hyperactive child, contributing to the overall negative climate of the home and to the pervasive problems hyperactive children have with peers.

Marital Distress and Disruption

Several studies indicate higher rates of marital distress and separation in families with hyperactive children (Ackerman, Elardo, & Dykman, 1979; Befera & Barkley, 1985; Cohen & Minde, 1983), but the data are more appropriately interpreted as revealing a general association between marital discord and children's behavior problems. Befera and Barkley (1985) found that mothers of clinically diagnosed hyperactive children reported more marital distress than did mothers of normal controls. Cohen and Minde (1983) reported more marital distress and parental separation in families of hyperactive kindergartners than in families of nonreferred controls. Ackerman *et al.* (1979) noted that 25% of their hyperactive clinic sample lived with a stepparent, whereas none of their learning disabled subjects did. However, when control groups include children with other externalizing symptomatology, the results no longer appear specific to hyperactivity. For example, McGee, Williams, and Silva (1984), in an epidemiological study of hyperactivity and aggression in New Zealand, found that marital separation was associated with aggression in children, whereas maternal reports of marital discord differentiated the hyperactive and the aggressive subgroups from the controls. These findings are consistent with a large body of other evidence demonstrating that children living in discordant families show a range of symptoms that can include fidgetiness, difficulties in concentration, and poor impulse control (e.g., Emery, 1982). Furthermore, there is evidence that children are sensitive to and influenced by expressions of anger in the home (Cummings *et al.*, 1984), and that parental disagreement over childrearing can have a negative impact on children (Block, Block, & Morrison, 1981). Although few people would suggest that marital discord or separation by themselves can cause a syndrome of hyperactivity, it is likely that family disruption can exacerbate and maintain symptoms of hyperactivity in children already vulnerable to develop problems and, conversely, that the presence of a hyperactive child in a troubled family may increase the intensity of family discord or hasten the decision to divorce.

Parental Psychopathology

Studies also have examined rates of parental and family psychiatric disorder as well as more general reports of maternal mood and feelings of well-being. As with the marital discord data, when families of hyperactives are compared to normal controls, higher rates of diagnosable psychiatric disorder are reported (Befera & Barkley, 1985; Cohen & Minde, 1983) as well as more depressed mood, more general malaise, and lower parenting self-esteem (Mash & Johnston, 1983). When the families of hyperactive children are compared to those of children with other disorders, however, few differences specific to hyperactivity are in evidence (e.g., McGee *et al.*, 1984; Sandberg *et al.*, 1980), and the subgroup differences that remain appear to be associated with aggression rather than hyperactivity (e.g., Stewart, deBlois, & Cummings, 1980). A number of studies have linked maternal reports of depression and marital dissatisfaction to externalizing problems in children, as noted earlier (e.g., Patterson, 1980; Porter & O'Leary, 1980; Richman *et al.*, 1982), and these findings support a general association, rather than one specific to hyperactivity. Such problems in a family will have an obvious impact on childrearing practices and the instability of the home environment and may also be related to increased biological vulnerability.

General Psychosocial Adversity

General measures of psychosocial adversity that include marital distress, parental physical and psychological disorder, unemployment, poor housing and/or other measures of environmental deprivation or instability have also been studied in relation to the onset and persistence of problems in young children and to hyperactivity in particular. Richman *et al.* (1982) found that environmental adversity was associated with higher rates of behavior problems in young children. Campbell, Breaux, Ewing, and Szumowski (1986) reported that lower socioeconomic status and higher levels of family stress were associated with maternal ratings of hyperactivity and aggression in a sample of parent-referred hard-to-manage 3-year-olds. Concurrent levels of family stress were also associated with ratings of hyperactivity, but not aggression, at age 6 follow-up. Similarly, Delameter, Lahey, and Drake (1981) found that a general family stress index differentiated hyperactive children from those with learning disabilities who were not hyperactive. Sandberg *et al.*, (1980) likewise reported a relationship between general psychosocial disadvantage and hyperactivity, but not conduct disorder. These results are difficult to interpret because measures and samples differ from study to study. More research will be needed before it can be concluded that these findings indicate more than the well-documented association between psychosocial stress and general externalizing symptomatology. It seems obvious that environmental stress and disorganization will have direct and indirect effects on the socialization of children.

Peer Relationships

Experiences with peers complement experiences in the family and both are necessary aspects of socialization (Hartup, 1983). Hartup suggests that learning to share, to negotiate, and to modulate aggression is facilitated by children's interactions with equals. Moral development, perspective-taking ability, and empathy are also likely to be dependent to some extent on experiences with peers (Piaget, 1965), although the foundations are probably laid within the family (e.g., Zahn-Waxler *et al.*, 1979). There is accumulating evidence that the nature of early peer encounters will be influenced by the quality of early experiences with parents, and that preschoolers with more secure relationships with parents will cope more effectively in the peer group (Sroufe, 1983). Learning to play cooperatively with other children is one of the major tasks of the preschooler, and it is not surprising that young hyperactive children who tend to be intense, impulsive, motorically active, and impatient have a difficult time learning the skills necessary to interact cooperatively with peers and to establish and maintain friendships (Campbell & Cluss, 1982).

Although hyperactive children are highly sociable, a number of recent studies document the pervasive and persistent problems hyperactive children have with peers (see Campbell & Cluss, 1982; Campbell & Paulauskas, 1979; Milich & Landau, 1982; Whalen & Henker, 1985, for more detailed reviews). Parents, teachers, peers, and even the hyperactive children themselves are aware of difficulties getting along with other children. Furthermore, these peer problems appear to have their onset quite early. Thus, aggression toward peers was one of the major complaints made by mothers of the hard-to-manage 3-year olds studied by Campbell *et al.* (1982), and independent teacher ratings and classroom observations confirmed these parent reports (Campbell & Cluss, 1982). Hyperactive youngsters were more aggressive than their classmates and were more likely to be the initiators of the aggressive encounter. Similar findings were reported by Schleifer and his colleagues in a study of clinically referred preschoolers (Schleifer *et al.*, 1975). Evidence also suggests that preschool children may be aware of the aggressive and domineering behavior of their hyperactive classmates, because teachers and preschoolers agree on which children, in nonclinical samples, are more active and aggressive, and these youngsters are more likely to be rejected than their less active and aggressive peers (Milich, Landau, Kilby, & Whitten, 1982; Rubin & Clark, 1983). Peers are less likely than parents to accommodate to the demands of the hyperactive child or to give in to his limited impulse control; rather they appear to seek other playmates, leaving the hyperactive child socially isolated. Thus, learning to share, to take turns, to wait, to attend to the needs of others, not to grab toys, or to hit—all important social behaviors that are learned in the preschool peer group—will be particularly difficult for the hyperactive child to master. Campbell & Cluss (1982) have argued that the core features of hyperactivity, inattention, overactivity, and impulsivity, make peer problems inevitable, particularly in preschoolers who are attempting to internalize the basic rules of social exchange. These

difficulties are probably compounded by the tendency of age-mates to avoid hyperactive children, setting up a cycle of inappropriate social behavior and peer rejection (Dodge, 1984).

Data indicate that early peer problems tend to persist and that school-age hyperactive children also experience significant difficulties establishing and maintaining cooperative relationships with other children. Follow-up studies of the Schleifer *et al.* sample and Campbell's parent-referred sample into elementary school indicated that youngsters identified as problems in the preschool years continued to be more disruptive than controls in the classroom (Campbell, Endman, & Bernfeld, 1977; Campbell, Ewing, Szumowski, & Breaux, 1986). Numerous studies of hyperactive children diagnosed in elementary school confirm that they engage in more inappropriate behavior in the classroom, including annoying peers and disrupting ongoing classroom activities; they are also more likely than normal controls to become involved in aggressive interactions in the classroom (Abikoff, Gittelman, & Klein, 1980; Klein & Young, 1979; Whalen & Henker, 1985). These socially inappropriate and overbearing behaviors characterize hyperactive children observed in unstructured play groups as well as in more structured classroom settings (Pelham & Bender, 1982).

The relationship between peer rejection and potentially symptomatic behaviors, such as aggression toward peers, disruptive and annoying behavior, noncompliance, and off-task behavior, is well documented (e.g., Dodge, 1984; Peery, 1979; Vaughn & Waters, 1981). Therefore, it is not surprising that studies using sociometric measures and peer-nomination techniques reveal that hyperactive children, whether selected by teacher ratings or clinical diagnosis, are more likely than their nonhyperactive classmates to be rejected by peers (e.g., Klein & Young, 1979; Pelham & Bender, 1982; Whalen & Henker, 1985). For example, Pelham and Bender (1982) reported that hyperactive children were more likely than controls to be described as getting mad, trying to get others into trouble, being mean, telling others what to do, and bothering others who are trying to work. In view of these behaviors, they are not likely to be sought after as playmates. Of particular interest in this study was the finding that after only one play group session, unacquainted peers rated hyperactive children as less likeable than other group members, suggesting that their deviant social behavior is far from subtle.

Several studies have examined the social information-processing skills of hyperactive children in an attempt to understand some of the reasons underlying their difficulties with peers. Although Paulauskas and Campbell (1979) did not find deficiencies in perspective-taking ability when hyperactive children were compared with age-mates, other studies suggest that information-processing deficits may contribute to their peer problems. Whalen and Henker (1985) have conducted a series of studies in which hyperactive children are required to interact with peers in group problem solving and referential communication tasks. In general, hyperactive youngsters have more difficulty shifting roles and adapting their behavior to the demands of the situation; they are also less responsive to communications from peers and more likely to ignore them or to behave inappropriately; finally, they are less helpful to their partners, providing less task-relevant information.

In a series of studies of peer-identified aggressive boys, many of whom may also be hyperactive, Dodge has identified several information-processing deficits that he suggests contribute to their consistent rejection by peers (reviewed in Dodge, 1984). These include a tendency to arrive at aggressive solutions to interpersonal problems, an attributional bias in which ambiguous interactions with peers are perceived as hostile in intent, thereby provoking an aggressive response, and a cue utilization deficit, reflected in quick decision making in the absence of complete information. Thus, Dodge suggests that aggressive, rejected boys see the world as hostile and react accordingly, thereby initiating a self-fulfilling prophesy, because their aggressive responses lead to their rejection by peers. Furthermore, such youngsters show a predisposition, possibly learned in the family, toward negative emotional responses in arousing and ambiguous situations that serve to trigger impulsive and angry responses, thereby short-circuiting more careful assessment of alternative, less provocative responses.

Milich and Dodge (1984) recognized that this model might apply to hyperactive children who are impulsive and rejected by peers. In their study of psychiatrically referred boys, hyperactive-aggressive boys showed these hypothesized deficits, although other groups of disturbed children also performed more poorly than normal control boys. Although this model has much intrinsic appeal, it fails to explain how these processes developed initially. Also, it has been tested using only hypo-

thetical stories. Assessment of actual encounters between hyperactive children and their peers will be necessary for a clear understanding of the deficits or distortions in their decision-making processes, response biases, and/or negotiation strategies.

Whalen and Henker (1985) suggested that hyperactive children may have adequate knowledge of social scripts, that is, of the behaviors that are expected in routine and well-rehearsed social situations, but they may have a production deficiency when they are called upon to generate such scripts, possibly because of their tendency to become easily aroused and overexcited. It is worth reiterating, in this context, that hyperactive children have difficulties in a range of daily situations that bring them into contact with others (Barkley, 1981; Whalen & Henker, 1985). Whalen and Henker also suggest that the intensity of hyperactive children's responses may reflect "social ineptitude" and insensitivity to the expectations of others, as well as limited awareness of their impact on others. Thus, even when they are trying hard to be helpful, these youngsters may get into trouble.

Parents often complain that their hyperactive child not only has few friends, but that he tends to play with younger children and to seek out other problem youngsters. This may be a function of his social rejection by more competent age-mates, a desire to play with those he can dominate, or a search for children at the same level of social functioning. Whalen and Henker (1985) report the intriguing finding that hyperactive boys in their summer camp program gave twice as many "like" nominations to boys who were also rated as causing trouble than did comparison boys. This is consistent with clinical reports and underscores the importance of assessing the patterns and stabilities of hyperactive children's friendships as well as their reasons for their friendship choices.

Taken together, the studies on peer relations in hyperactive children indicate that despite their sociable and exuberant interpersonal styles, these children tend to have consistent and persistent difficulties interacting cooperatively with other children in the classroom and in social settings. This is reflected in more aggressive, annoying, and irritating behavior and more rejection by peers. Studies are just beginning to explore the cognitive processes that may underlie these pervasive and long-standing difficulties, which appear to continue and possibly exacerbate in adolescence (Weiss, 1983). It is likely that socialization processes that had their start in the family (as a function of the child's biological vulnerability as well as family factors) ultimately influence the nature of hyperactive children's social behavior with peers. The intensity and inappropriateness that lead to rejection by peers also result in only limited positive experiences with others. Therefore, hyperactive children appear to seek out younger children or those with problems, giving them increasingly fewer opportunities to overcome their social interactional deficits. Over time, peer problems and family conflicts probably fuel each other, as children become more frustrated, angry, and lonely.

Summary and Conclusions

It is likely that the processes of socialization are similar for hyperactive and nonhyperactive children, but that the task of socializing a hyperactive child will be far more difficult for parents. Furthermore, given the wide individual differences in the behavior of hyperactive children and the considerable variations found in family functioning, some hyperactive children will be more difficult to socialize than others; similarly, some families will be better equipped to cope with the demands of parenting a hyperactive child. Thus, the success with which a particular hyperactive child is socialized will depend upon a complex mix of child characteristics, childrearing tactics and other family factors, the nature of the peer group, and other environmental and sociocultural factors. Follow-up studies suggest that outcome, which may be relabeled as successful socialization, is especially dependent upon family functioning and the quality of the mother–child relationship (Campbell, Breaux, Ewing, & Szumowski, 1986; Weiss, 1983). Thus, hyperactive children living in disorganized, punitive, or inconsistent environments tend to do poorly. However, an appropriately structured and positive environment does not guarantee a good outcome: some children will outgrow early difficulties given supportive, firm, consistent, and loving parents who are attuned to their needs at different developmental periods; other children will continue to have problems despite extremely patient and appropriate parenting (Campbell, Ewing, Breaux, & Szumowski, 1986); still others will develop problems (or be identified as problems) as the demands for conformity in the preschool or elementary school classroom overwhelm their limited capacity to monitor their own behavior, possibly a reflection of excessively lenient limits at home that may also interfere with the development of internalized controls. Even though it is possible to extrapolate from

studies of normal children to children with problems, more systematic studies of the specific aspects of parental behavior that predict successful socialization and, therefore, good outcome, need to be delineated. This chapter also highlights the importance of examining socialization and symptomatic behavior from a developmental perspective because developmental needs and salient symptoms appear to be related and both change over time.

The relationship between hyperactive children's family and peer group experiences has not been studied, nor have peer relations or friendship patterns been examined as potential predictors or mediators of outcome in adolescence or young adulthood. Although much progress has been made recently in describing the social behavior of hyperactive children in a variety of settings with mothers and peers, studies need to focus on the hyperactive child as a member of a family system, on the nature of hyperactive children's friendships, and on the interrelationship between these social systems. Furthermore, although data from several studies suggest that family stability and parent–child relations mediate outcome, it is unclear what role relationships with other children play in socialization. For example, it is unknown whether positive peer experiences can help a hyperactive child to overcome family problems and vice versa. Finally, prospective, longitudinal studies that examine the processes of socialization in hyperactive children need to be conducted.

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CHAPTER 8

Genetics and Biochemistry in Attention Deficit Disorder

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The heterogeneous nature of Attention Deficit Disorder (ADD) obscures its biological bases. One problem is that ADD subsumes a variety of patterns of inattention, impulsivity, and hyperactivity. Additionally, different sources of brain disorder functionally converge on these patterns. This chapter reviews how geneticists and biochemists have studied ADD under these handicaps and how they might turn heterogeneity to their advantage.

Some researchers consider genetic analysis of such heterogeneous disorders to be premature. They have recommended postponing analysis until a biochemical breakthrough provides clear nosologic entities. We take the alternative position that genetics itself may reveal natural subgroups of ADD and may provide the groundwork for a biochemical breakthrough. We will describe a strategy for obtaining more homogeneous subtypes: genetic latent structure analysis (Matthysse, 1985). But first we will review genetic studies of ADD derived from the staples of genetic epidemiology—family, twin, and adoption studies. (In previous research, no chromosomal abnormality has been detected; Warren, Karduck, Bussaratid, Stewart & Sly, 1971). As a framework for this review, we highlight some problems raised by diagnostic and etiological heterogeneity in ADD.

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Diagnostic Heterogeneity

There being no agreement as to ADD's biological bases, we rely on its nonspecific surface symptomatology. Because ADD symptoms are not qualitative but *quantitative* deviations from the norm, lack of specificity is particularly problematic. A further complication is the fact that ADD symptoms are qualified by developmental status (Kinsbourne, 1983):

1. The frequency and severity of ADD symptoms vary continuously in the general population. Thus, there are many transitional cases that could be interpreted as mild ADD or extremes within the normal range, which frustrates attempts to derive a discrete diagnosis.

2. The severity of ADD is expressed against a shifting developmental baseline. Parent and teacher symptom rating scales administered to normal school-aged populations document systematic declines in inattention, impulsivity, and especially restlessness as children grow older. Additionally, males exhibit more severe symptomatology at any given age (Goyette, Conners, & Ulrich, 1978; Swanson, Sandman, Deutsch, & Baren, 1983).

3. Surface symptomatology may change in ways that become clear only when the life span is taken into consideration. Symptoms of childhood ADD can persist into adulthood (see reviews by Amado & Lustman, 1982; Thorley, 1984). There may be even more serious outcomes, including

antisocial and borderline personality disorders, alcohol and drug abuse, and schizophrenia (Cantwell, 1978; Goodwin, Schulsinger, Hermansen, Guze, & Winokur, 1975; Morrison, 1979; Rieder & Nichols, 1979; Tarter, McBride, Buonpane, & Schneider, 1977).

Genetic analysis would be facilitated by understanding how ADD is manifested in childhood. Unfortunately, many outcome studies suffer from serious methodological flaws (Thorley, 1984). Even competent studies depict a wide range of prognoses, from grim (Satterfield, Hoppe, & Schnell, 1982) to benign (Gittelman, Mannuzza, Shenker, & Bonagura, 1985). Given such scant information, the geneticist cannot identify particular adult psychiatric diagnoses as markers for childhood ADD. Even an overrepresentation of adult disorders among the relatives of ADD probands (see below) cannot validate this assumption. At this time, comparably aged siblings are the most appropriate relatives for genetic analysis of childhood ADD.

Etiological Heterogeneity

ADD is the behavioral end point for a number of brain insults and injuries. Early observations of patients with von Economo's encephalitis revealed inattention and restlessness in the postencephalitic state (Chess, 1979). ADD symptomatology is attributed to damage of the frontal lobes (Strauss & Kephart, 1955; Strauss & Lehtinen, 1948) and brainstem (Kahn & Cohen, 1934). Also, ADD symptoms are among the cardinal signs of fetal ethanol embryopathy (S. E. Shaywitz, Cohen, & Shaywitz, 1980; S. E. Shaywitz, Caparulo, & Hodgson, 1981; Streissguth, Herman, & Smith, 1978).

Environmental determinants of ADD are difficult to assess. Genetic hypotheses lend themselves to more straightforward empirical tests (Gottesman & Shields, 1982; Plomin, DeFries, & McClearn, 1980). Only recently have more sophisticated quantitative genetic methods been applied to ADD, as they have been to the major psychoses (Elston & Rao, 1978; Gershon, Matthysse, Breakfield, & Ciaranello, 1981; Kidd, 1985; Morton, Rao, & Lalouel, 1983; Weissman, Merikangas, Wickramaratne, Kidd, Prusoff, Leckman, & Pauls, 1986). Genetic studies of ADD to date have set themselves two goals: to test the validity of a genetic hypothesis for ADD, and to describe which non-ADD disorders are statistically overrepresented among biological relatives.

Family Studies

Based on his clinical impressions, Wender (1971) hypothesized the presence of genetic influences for hyperactivity. His impressions were borne out by several family studies of ADD (Table 1). These studies compared the biological relatives of ADD with control index cases (probands). An excess of ADD among the relatives of ADD probands would support a genetic hypothesis. But because families share genes *and* environment, these are inextricably confounded. A family study can support a genetic hypothesis but cannot confirm it.

Table 1 summarizes the statistical overrepresentation of psychiatric diagnoses among the biological relatives. We computed certain contrasts that were not made in the original papers. Findings are summarized only for first-degree relatives (i.e., mothers, fathers, sisters, and brothers of ADD and control probands), for whom diagnoses are most reliable.

Combining groups of relatives may increase statistical power, allowing detection of differences not observed in contrasts of specific groups of relatives. For example, in the Biederman study (Biederman, Munir, Knee, Habelow, Armentano, Autor, Hoge, & Waternaux, 1986), ADD was not significantly overrepresented among mothers or sisters of ADD probands. But pooling these two groups (increasing the sample size) revealed a statistical overrepresentation of ADD among female relatives of ADD probands. Biederman *et al.* (1986) also found an excess of Major Depressive Disorder and Oppositional Disorder among pooled relatives of ADD probands. Depression was more prevalent among female relatives of ADD probands than among controls. Oppositional Disorder was more prevalent in parents, siblings, and male relatives, and female relatives of ADD probands. The overall rates of Antisocial Personality and Substance Use Disorders did not differ between relatives of ADD and control probands. But a subgroup of probands with Major Affective Disorder did reveal a higher risk for these disorders among their relatives (Biederman *et al.*, 1987).

Methodological Concerns

1. *Choice of control groups.* The specificity of the familial association of hyperactivity and adult psychiatric disorders reported by the early family studies was questioned because the investigators used normal rather than psychiatric controls. In subsequent studies, Stewart, DeBlois, and Cum-

Table 1. Family Studies of Childhood Hyperactivity and Attention Deficit Disorder

Study	*N* of experimental, control probands	Control probands	Classes of first-degree relatives	Blind diagnosis?	Overrepresentation[a]
Morrison & Stewart (1971)	59,41	Normal	Parents	No	M: hysteria F: HA
Cantwell (1972)	50,50	Normal	Parents	No	M: hysteria alcoholism F: HA sociopathy
Welner, Welner, Stewart, Palkes, & Wish (1977)	43,38	Normal	Siblings	No	B: HA depression
Stewart, DeBlois, & Cummings (1980)	69,57	Psychiatric	Parents	Yes	F: alcoholism[b]
Morrison (1980)	140,91	Psychiatric	Parents	No	F: sociopathy
Befera & Barkley (1985)	30,30	Normal	Mothers	Self-report	M: depression
Biederman, Munir, Knee, Habelow, Armentano, Autor, Hoge, & Waternaux (1986)	22,20	Normal	Parents and siblings	Yes	F: ADD B: ADD

[a]HA = hyperactivity (Stewart, Pitts, Craig, & Dieruf, 1966, criteria); ADD = attention deficit disorder (DSM-III criteria); M = mothers; F = fathers; B = brothers.
[b]In absence of primary diagnosis of antisocial personality.
Note. Adapted from "A Family Study of Patients with Attention Deficit Disorder and Normal Controls" by J. Biederman, K. Munir, D. Knee, W. Habelow, M. Armentano, S. Autor, S. K. Hoge, and C. Waternaux, 1986, *Journal of Psychiatric Research, 20,* p. 265. Copyright 1986 by Pergamon Journals Ltd. Adapted by permission.

mings (1980) and Morrison (1980) responded to their critics by using a highly conservative bias in choosing controls. Morrison (1980) selected a miscellany of disorders for his psychiatric control group's probands, notably affective disorder. That there was no excess of alcoholism among the parents of hyperactive children relative to the control group is not surprising given the familial association between childhood depression and adult alcoholism (see the review by Orvaschel, Weissman, & Kidd, 1980). An analogous problem arose in the Stewart *et al.* (1980) study: the psychiatric control probands included undersocialized aggressive boys, whose parents would be expected to have a high rate of alcohol abuse and antisocial personality (Cantwell, 1975).

2. *Diagnostic criteria.* The early family studies used diagnostic criteria drawn from a number of expert sources, some concordant with DSM-II criteria (cf. Morrison, 1980). Critiques of diagnostic criteria and of the practice of obtaining retrospective diagnoses in these studies are detailed by Dubey (1976) and McMahon (1980). Befera and Barkley (1985) diagnosed their ADDH probands using DSM-III criteria, and depressive symptomatology by self-report on the Beck Depression Inventory. Only the Biederman *et al.* (1986, 1987) family study diagnosed relatives by DSM-III criteria.

3. *Blind diagnosis.* Knowledge of diagnosis of the proband may unconsciously bias ratings of relatives' psychopathology. Of the studies in Table 1, only Stewart, DeBlois, and Singer, (1979) and Biederman *et al.* (1986) maintained a diagnostic blind. Befera and Barkley (1985) used self-report ratings of maternal depression.

A Half-Sibling Study

This design is a variation on the conventional family study design. Safer (1973) compared the number of times that full-sibling and half-sibling pairs were concordant for minimal brain dysfunction (MBD—a pre-DSM nosologic analogue of ADD). The half-siblings had their mother in common and were reared together. He found that 10 of the 19 full-siblings were diagnosed as MBD. Only 2 of the 22 pairs of half-siblings had this diagnosis, consistent with a genetic hypothesis.

Are ADDH and Tourette Syndrome Genetically Related?

A review of ADD family studies would be incomplete without citing this recent controversy. It began with the suggestion that Gilles de la Tourette Syndrome (TS) and ADD with hyperactivity (ADDH) may be due to the same underlying genetic factor(s); ADDH may represent a different (possibly milder) manifestation of those factors (Comings & Comings, 1984). A TS family study by Pauls, Hurst, Kruger, Leckman, Kidd, and Cohen (1986) did not confirm this hypothesis:

1. The frequency of ADDH among the relatives with both disorders (TS + ADDH) was higher than the frequency of ADDH among the relatives of those with pure TS (TS − ADDH). This suggests that TS and ADDH are genetically unrelated or that TS + ADDH and TS − ADDH are etiologically distinct syndromes.
2. There was independent segregation (random association) of ADDH and TS in the affected relatives of probands with TS + ADDH. This argues against genetic commonality.
3. There was no difference between the frequencies of ADDH among relatives of TS probands compared with the population prevalence of ADD. A genetic association would predict an increased frequency among the relatives of TS probands.

Beyond its intrinsic interest, this issue introduces a broader question: How might genetic models incorporate multiple manifestations of a single genetic entity? We will address this question shortly.

Summary of Family Studies

What overall impression of the familiality of ADD do these studies provide? Only the Welner, Welner, Stewart, Palkes, and Wish (1977) and the Biederman *et al.* (1986) studies ascertained siblings, and only the latter study utilized a diagnostic blind to group membership. Both studies support a genetic contribution to ADD. Additionally, a number of adult disorders seem to run in the families of hyperactive children, notably depression, sociopathy, alcoholism, and hysteria. The latter three disorders have been termed the "sociopathic spectrum" (Schulsinger, 1972).

Does a history of sociopathic spectrum disorders in parents of hyperactive probands predict symptomatology among the probands' siblings? August (1982) subgrouped pedigrees of hyperactive probands by a positive or negative parental history for a sociopathic spectrum disorder. Although the severity of hyperactivity was similar in the two groups of probands, the frequency of psychopathology in their siblings differed. The frequencies of hyperactivity among biological siblings of probands in the positive and negative parental-history groups were 8% and 17%, and of conduct disorder, 24% and 0%, respectively.

Do parents with sociopathic spectrum disorders genetically transmit a predisposition to ADD? This question is impossible to answer through family studies, but we will discuss some attempts to deconfound genes and environment through twin and adoption studies.

Twin Studies

Twinning allows tests of genetic hypotheses through an experiment of nature. This experiment derives from the existence of identical (monozygotic, or MZ) and fraternal (dizygotic, or DZ) twins. MZ co-twins arise from a single zygote that divides into two separate embryos early in gestation. Because only one zygote is involved, the twins are genetically identical. In contrast, DZ co-twins arise from two zygotes that are produced by fertilization of two separate ova; they have the same genetic relationship as siblings (sharing 50% of their genes). MZ twins are always of the same sex, but DZ twins may differ in gender.

A comparison of MZ and DZ co-twin concordance for ADD allows a test of a genetic hypothesis: To the extent that ADD is inherited, the concordance rate would be higher in the MZ than in the DZ co-twins. Two studies have taken this approach. Lopez (1965) reported the concordance rate for hyperactivity in MZ and DZ twins to be 100% and 17%, respectively, supporting a genetic hypothesis. But his study was flawed by the fact that, whereas all 4 MZ twin pairs were male, 4 of the 6 pairs of DZ twins were female. Given the reported gender difference for hyperactivity, failure to match for sex in the MZ and DZ twins compromised his conclusions.

In a rigorous twin study of hyperactivity, Willerman (1973) concentrated on one aspect of ADD symptomatology: locomotor hyperactivity. Mothers of twins completed questionnaires on activity level (measured on a continuous scale) and

zygosity. The intraclass correlation for activity level in the MZ twins was significantly higher than for the DZ twins, suggesting a substantial genetic contribution. Willerman then defined twins with ratings in the top 20% of the distribution as "hyperactive." There were 8 MZ and 16 DZ twin pairs with activity scores in this range. MZ twins showed a high correlation for activity, whereas DZ twins showed no correlation. A contrast of MZ and DZ concordance rates allows estimation of heritability, the proportion of the total phenotypic variance in a population attributed to genetic variance (Plomin *et al.*, 1980). Willerman (1973) found the heritability of locomotor hyperactivity to be approximately 70%.

Twin studies provide a valuable technique for evaluating genetic hypotheses. Yet, unless the twins are raised apart, gene–environment confounds still exist. Adoption studies provide the most convincing demonstration of genetic components to psychiatric disorders. These designs deconfound genetic and environmental factors common to biological families by studying genetically related individuals living apart (to test genetic influences) and genetically unrelated individuals living together (to assess environmental influences common to family members). Evidence of heritability would in no way obviate the role of environment.

Adoption Studies

Early Adoption Studies

Morrison and Stewart (1971) and Cantwell (1972), having demonstrated the familiality of hyperactivity and adult psychiatric diagnoses, mounted adoption studies to test genetic hypotheses. They used the "adoptive parents method" (the nomenclature of Rosenthal, 1971) first used by Wender, Rosenthal, and Kety (1968) in studies of schizophrenia.

The Morrison and Stewart (1973) design compared psychopathology in the adoptive and the biological parents of hyperactive probands. Three groups were studied: (1) hyperactive probands and their biological relatives, (2) adopted hyperactive probands and their adoptive relatives, and (3) normal control probands and their biological relatives. Within this design, common postnatal environment is assumed to be held constant, but genetic relatedness is varied as an independent variable; a genetic influence would predict more psychopathology in the biological (Group 1) and in the adoptive (Group 2) parents of hyperactive probands. The biological hyperactive probands (Group 1) can be further compared to the biological parents of normal probands (Group 3); this comparison constitutes a family study design (Rosenthal, 1971) identical to that employed by Morrison and Stewart (1971) and Cantwell (1972).

Morrison and Stewart (1973) found childhood hyperactivity (retrospectively diagnosed) or a sociopathic spectrum disorder more often in biological than in adoptive parents of hyperactive probands. This outcome is consistent with a genetic hypothesis. The family study contrast (Group 1 vs. Group 3 relatives) also supported a genetic hypothesis. The frequency of parental psychopathology did not differ between the control and the adoptive groups. The pattern of findings for parents was duplicated for a combined group of parents and second-degree relatives. Cantwell (1975) corroborated these results in an adoptive parents study identical in design to that of Morrison and Stewart's.

Toronto Adoption Study

The early adoption studies focussed on psychopathology in parents to the exclusion of siblings. Our study adapted the adoptive parents method to include siblings (Deutsch, 1983; Deutsch & Swanson, 1985). The pattern of findings parallels the previous two adoption studies: the biological ADD, adoptive ADD, and biological control probands ($N = 72$) were matched for age, gender, and parental socioeconomic status. We assessed a wide range of psychiatric symptomatology, with emphasis on ADD (including Residual Type). There was a higher rate of ADD symptomatology among both parents and siblings of biological ADD probands (Group 1) than parents and siblings of the adoptive ADD probands (Group 2), supporting a genetic hypothesis. The analysis of children of adopted probands' parents should be confined to biological offspring. This is because adoptees are at increased risk for ADD (see below).

The frequency of ADD in relatives of the adoptive and control group probands were not significantly different. This contrast could conceivably reveal a selection bias: adoptive agencies may select adoptive parents for absence of psychiatric diagnosis. They may be "supernormal." Supernormality would be indicated by less symptomatology in the adoptive relatives than in the control relatives. There was no evidence of supernormality in these adoption studies.

In the Toronto Adoption Study, ADD symp-

tomatolgy was assessed on a continuous scale. We also used standard (*z*-) score cutoff points to obtain discrete diagnoses. Both means of rating ADD yielded significant contrasts of Groups 1 and 2.

A family study (whose design contrasts Groups 1 and 3) is embedded within the adoption design. As in the earlier adoption studies, this contrast supported a genetic hypothesis.

Overrepresentation of Adoption among ADD Patients

Adoptive status confers a high risk for ADD (Deutsch, Swanson, Bruell, Cantwell, Weinberg, & Baren, 1982). We have found that approximately 17% of ADD children are nonrelative adoptees. This figure represents an eight-fold increase over population and control group base rates. Dalby, Fox, and Haslam (1982) and others have replicated these findings.

These figures lead one to predict a high frequency of ADD among adoptees. We tested this prediction directly by randomly ascertaining a population of nonrelative adoptees (Deutsch, in press). Indeed, there was excessive ADD symptomatology among nonrelative adoptees. Both environmental and genetic hypotheses may be invoked to explain this phenomenon (Deutsch, in press). One genetic hypothesis is suggested by a study of unwed mothers by Horn, Green, Carney, and Erickson (1975). According to the U.S. Department of Health, Education and Welfare (1970), 88% of nonrelative adoptees' biological mothers are unwed. Horn and his colleagues found that, controlling for the effects of pregnancy, unwed mothers had an excess of psychopathology (notably, antisocial personality disorder). To the extent it is heritable, the psychopathology might be transmitted to their adopted-away offspring.

The family, twin, and adoption studies provide convergent support for a genetic hypothesis for ADD symptomatology. This constitutes a first step toward understanding the genetics of the disorder. But we are left with fundamental questions: Which form(s) of ADD are heritable? What might be transmitted, and by what mode of inheritance? Heterogeneity stymies attempts to answer these questions. One solution to this problem is to seek biological correlates that reveal homogeneous subgroups (Gottesman & Shields, 1982).

One conventional technique used in this search will usually fail: to find a putative "marker" and subgroup patients by its presence or absence. These markers have proven elusive for a simple reason: Statistical power is too low to detect abnormality. This is true even if the subgroup with the abnormality comprises a substantial proportion of patients (see Monte Carlo study in Matthysse, 1985). If a laboratory were to find such an abnormality, it probably would fail to replicate.

We will outline an alternative strategy that employs biological correlates. But first we introduce the measures used in these analyses: minor physical anomalies.

Dysmorphology

An excess of minor physical anomalies (MPAs) is likely the most frequently replicated biological abnormality in ADD (for review, see Krouse & Kauffman, 1982). MPAs are dysmorphological signs that require neither medical attention nor cosmetic concern. But these minor features may be of major diagnostic significance, indicating general classes of altered morphogenesis or specific malformation syndromes (Deutsch, 1987; Pinsky, 1985; Jones, 1982).

MPAs commonly assessed include epicanthus, single palmar creases, wide-set eyes, and ear malformations. It is not unusual to have one or two anomalies, but three or more are uncommon. Waldrop and Halverson's (1971) protocol has been used almost exclusively to assess MPAs in psychiatric disorders. This scale yields a summary score that is a weighted sum of individual MPAs.

High MPA scores are by no means confined to ADD patients. Excessive scores have been reported in schizophrenia (see the reviews by Guy, Majorski, Wallace, & Guy, 1983, and Gualtieri, Adams, Shen, & Loiselle, 1982). Gualtieri *et al.* (1982) reported that alcoholic adults did not have an overall excess of MPAs. But the MPAs were distributed bimodally, suggesting a dysmorphological subgroup.

Firestone, Peters, Rivier, and Knights (1978) assessed MPAs in ADD and normal control boys and their first-degree relatives. MPAs were overrepresented in both ADD probands *and* their relatives. We have replicated this finding (Deutsch, Matthysse, Swanson, & Farkas, in press). A curious familial pattern emerged: Firestone's and our group found many dysmorphic relatives to be psychiatrically normal. There was no one-to-one correspondence between psychopathology and dysmorphology. How could this familial pattern be explained? One explanation is that there is a *single underlying factor* that produces both psycho-

pathology and dysmorphology. An individual with the factor may have ADD, MPAs, or both. In fact, this model would explain our finding that some psychiatrically normal relatives were dysmorphic, even when the ADD probands were *not* dysmorphic.

How can this pattern by modeled? Recently, a technique has been developed that can account for heterogeneous manifestations of an underlying factor (Matthysse, 1985). Further, this method can *use* heterogeneity to obtain more homogeneous subgroups. We introduce the rationale of this technique below and provide a worked example using our family data.

Genetic Latent Structure Analysis

This model assumes that (1) there is no one-to-one map between syndromes and etiologies and that (2) each syndrome expresses itself as a range of possible symptoms. We seek a transmissible underlying factor. The factor may be genetically transmitted, but we do not make that assumption.

The transmissible pathologic process could be expressed in various ways. (If the disease were genetic, these multiple manifestations would be termed "pleiotropic.") The range of symptoms is independently distributed, each symptom with its own probability ("incompletely penetrant"). Such a process is best studied in the family, where the range of symptoms can be sampled most powerfully. In fact, probands are inadequate for the study of their own disease.

Manifestations of the pathologic process ("manifest traits") are measured for each family member. The pathologic process is termed *latent* because it is unobservable; only its manifestations can be measured. We assume the latent trait to have a concrete biological meaning, even if it is concealed at the time of the analysis.

Based on the pattern of manifest traits in the family, it is possible to *infer* the latent trait. This is done by latent structure analysis, developed principally by Goodman (1974). Matthysse (1985) has incorporated this technique into pedigree analysis so that patterns of inheritance can be modelled. It has been applied to major psychoses (Matthysse, Holzman, & Lange, 1986) and ADD (Deutsch *et al.*, in press). In ADD, we postulated the latent trait to be a dysmorphic process that is manifested as central nervous system (behavioral) or craniofacial (dysmorphological) abnormalities. Employing the biological families from the Toronto Adoption Study, we studied ADD symptomatology and MPAs as dichotomous manifest traits.

Using these data, genetic models ascertaining only ADD and not MPAs did not provide a clear picture of mode of inheritance (Lange & Deutsch, unpublished data). A different pattern emerged with latent structure analysis of ADD and MPAs: the data fit an autosomal dominant model for the latent trait. The latent trait does not produce the manifest traits with certainty. Given the presence of the latent trait in an individual, the probability of ADD was estimated to be 43%, and the probability of a high MPA score was 38%. Bayesian analysis suggested that the latent trait is carried by 81% of ADD individuals. The frequency of carriers for the latent trait in the general population is estimated to be 6%. The model bore out the suggestion that phenocopies of a transmitted form of ADD exist (Rapoport, Pandoni, Renfield, Lake, & Ziegler, 1977).

This example illustrates the genetic latent structure strategy. But these data and the autosomal dominant model derived from them are not definitive: (1) they were derived from only 24 nuclear families. (2) A continuous scale would be more appropriate for assessing ADD symptomatology, as described above. Our cut-points for determining the dichotomous traits also impact on the results. (3) The catalog of MPAs with which we diagnose dysmorphology is limited.

Once the structure analysis is performed, it is possible to subgroup individuals on the basis of their latent trait status. One obtains two groups: those who probably have it and those who probably do not. This provides a rational basis for obtaining homogeneous subgroups and may even pave the way for biochemical discoveries.

Biochemical Hypotheses

Biochemical studies of ADD have focused on monoamine metabolism. The primary neurotransmitters of interest have been the catecholamines (dopamine [DA] and norepinephrine [NE]) and serotonin (5-hydroxytryptamine [5-HT]). Each neurotransmitter system has been hypothesized to be abnormal—usually underactive—in ADD.

Serotonergic Hypothesis of ADD

Until recently, there has been little support for this hypothesis (Coleman, 1971; Wender, 1969; see also the review by Ferguson & Pappas, 1979).

More recent platelet studies have rekindled interest in serotonin (Goldman, Thibert, & Rourke, 1979; Irwin, Belendiuk, McCloskey, & Freedman, 1981). Goldman *et al.* (1979) reported abnormally high platelet serotonin levels in subgroups of hyperactive children. As they pointed out, this may be construed to indicate increased serotonergic activity in the central nervous system *or* decreased activity through defective release or binding of 5-HT.

This example illustrates a broader phenomenon in biological psychiatry: An abnormal neurotransmitter level may be taken to support either an over- or underactive state. Low monoamine metabolite levels have been suggested to indicate underactive systems. But low levels may compensate for an overactive system. This absence of strong inference generalizes to discussions of receptor activity (see the review by Iverson, Iverson, & Snyder, 1983).

In any case, an underactive serotonergic system could hardly be considered specific for ADD. Perturbations of the 5-HT system have been noted in several disorders (van Praag, Kahn, Asnis, Lemus, & Brown, 1987). Low levels of 5-HT metabolite 5-hydroxyindoleacetic acid (5-HIAA) in cerebrospinal fluid (CSF) or in brain has been reported in obsessive-compulsive disorder, bulimia, sociopathy, and schizophrenia. A common thread running through all these disorders is deficient impulse control. In fact, deficient 5-HT metabolism may characterize many "disorders of constraint" (Depue & Spoont, 1986).

Catecholaminergic Hypotheses of ADD

The bulk of biochemical investigation has focused on the catecholamine metabolic pathway. These hypotheses take two basic forms: functional underactivity of the NE (Wender, 1974) or DA system (Shaywitz, Yager, & Klopper, 1976).

Many ADD patients respond favorably to CNS stimulants such as *d*-amphetamine and methylphenidate (Barkley, 1981; Kinsbourne & Swanson, 1979). Their action is partly mediated by catecholaminergic mechanisms (Coyle & Snyder, 1979; Moore, 1978). Because these stimulants agonize DA and NE action, it is hypothesized that these symptoms must be underactive in ADD.

Pharmacological Probes of ADD

But which neurotransmitter system is more important in the mediation of drug effects? Unfortunately, it is difficult to define the relative contribution of the NE and DA systems in the amelioration of ADD. For instance, amphetamines may mimic NE or DA at postsynaptic receptor sites, increase the concentration of amines by inhibiting monoamine oxidase (MAO) or catechol-*O*-methyltransferase (COMT), block reuptake of NE and DA, and potentiate their release (Moore, 1978). Given the widespread impact of CNS stimulants on catecholaminergic pathways, it is difficult to determine whether agonism of the dopaminergic or noradrenergic system would be more efficacious in treating ADD.

Drugs more specific to either the DA or NE system may allow for pharmacological probes of catecholamine function. Still, interpretation of the action of these probes can be complicated. For example, if pemoline preferentially agonizes DA rather than NE *and* is an effective treatment for ADD (Bachman, 1981; Conners & Taylor, 1980), is the ADD biochemical abnormality more dopaminergic than noradrenergic? Not necessarily. DA is immediately metabolized by dopamine-β-hydroxylase (DBH) to NE; the facilitation of DA can serve to engage NE mechanisms. The story is further complicated by the fact that DBH is not even the rate-limiting step in catecholamine production; tyrosine hydroxylase (TH) is (Cooper, Bloom, & Roth, 1982). Either DA or NE could decrease TH activity by feedback inhibition.

Could pharmacological probes test the involvement of catecholamine versus *other* neurotransmitter systems? It is difficult to alter a single neurotransmitter system with today's probes: a single neurotransmitter hypothesis for ADD is untenable (see the review by Zametkin & Rapoport, 1986). Yet pharmacological probes have the potential to reveal indirectly altered sensitivity of classes of receptors, for example, through neuroendocrine responses to drug challenges (Brown, 1977; Garfinkel, Brown, Klee, Braden, Beauchesne, & Shapiro, 1986; Greenhill *et al.*, 1984; Hunt, Cohen, Anderson, & Clark, 1984; Shaywitz *et al.*, 1982).

Medications used to treat ADD are nonspecific in their affects on neurotransmitters. But that may be one reason why they are effective: They may enhance attention by acting on one system (e.g., NE) and decrease motor activity by acting on another (e.g., 5-HT) (see the review by Teicher & Baldessarini, 1987).

Perhaps it would be instructive to explore the reciprocal balance among neurotransmitter systems. This may be possible in the catecholamine

system. The metabolism of DA into NE is mediated by DBH. Inhibiting DBH activity (e.g., by fumaric acid) would shift the equilibrium in the direction of DA.

Catecholamine Metabolites

Most tests of the catecholamine hypotheses have inferred underactivity from decreased levels of urinary metabolites (Ferguson & Pappas, 1979). However, several problems weaken this inference.

1. Only a fraction of urinary metabolites is derived from CNS pools. NE metabolism is a case in point. The major metabolite of NE in the CNS is its glycol derivative, 3-methoxy-4-hydroxyphenylethyleneglycol (MHPG) (Cooper *et al.*, 1982). The exact percentage of MHPG derived from brain NE is controversial (Warsh, Stancer, & Li, 1981), but Kopin, Jimerson, Markey, Ebert, and Polinsky (1984) estimate that less than 20% of urinary MHPG is derived from brain NE.

Compared to urinary MHPG, plasma MHPG is a superior index of catecholamine metabolism; the turnover rate of plasma MHPG accounts for over two-thirds of all catecholamines metabolized in the body. Plasma levels are highly correlated with levels in CSF, which more directly reflect brain concentrations. Normally, about 50% to 70% of MHPG in CSF is derived from the CNS (Kopin *et al.*, 1984). However, plasma MHPG is not necessarily an adequate index of central NE activity. There is a free exchange of plasma, CSF, and tissue MHPG. In fact, the covariance of plasma MHPG levels with those of CSF and brain areas is largely attributable to exchange with plasma. Knowledge of these equilibria allows adjustment of CSF MHPG levels for plasma MHPG (Jimerson, Insel, Reus, & Kopin, 1983). Thus, CSF MHPG studies testing a NE hypothesis are more interpretable if they include simultaneous measurement of plasma MHPG.

2. The disposition of DA metabolism is similarly complicated (Kopin, 1978). There may be little or no relationship between CNS dopaminergic activity and peripheral DA metabolites (Commisiong, 1985).

3. The motor restlessness present in many ADD patients may also present a confound in metabolite studies. Physical activity has been shown repeatedly to increase urine and plasma NE levels (see the review by Tang, Stancer, Takahashi, Shephard, & Warsh, 1981). However, it is unclear whether overactivity enhances peripheral MHPG production. There appears to be little effect of exercise on urinary MHPG excretion, but this finding is controversial. On the other hand, plasma levels of MHPG and VMA (vanilymandelic acid) are increased after exercise (Tang *et al.*, 1981). Thus, it may be advisable to monitor physical activity in order to partial out its effect.

4. The potential for contamination of results by lingering medication effects is great, even when drug washouts are employed. For instance, a decrease in urinary MHPG levels after exposure to dextroamphetamine persists at least two weeks after the medication has been discontinued (Zametkin, Karoum, Rapoport, Brown, & Wyatt, 1985). How long a washout is required is unclear. Thus, it is difficult to interpret reports of decreased urinary MHPG in ADD patients who have been exposed to dextroamphetamine (e.g., Shekim, Dekirmenjian, & Chapel, 1977; Shekim, Dekirmenjian, Chapel, Javaid, & Davis, 1979; Shekin, Javaid, Dans, & Bylund, 1983).

A cautious approach to drug washout does not preclude another type of confound: change in receptor sensitivity as the result of chronic stimulant medication. Conceivably, long-term exposure to catecholamine agonists may decrease receptor binding. Ideally, biochemical studies should be performed on drug-naive patients.

Urinary catecholamine metabolite studies have not pointed to clearly decreased levels in ADD (Khan & Dekirmenian, 1981; Rapoport, Mikkelson, Ebert, Brown, Weise, & Kopin, 1978; Shekim *et al.*, 1977, 1979, 1983; Shekim, Javaid, Dekirmenian, Chapel, & Davis, 1982; Wender *et al.*, 1971; Yu-cun & Yu-Feng, 1984; Zametkin, Karoum, Rapoport, Brown, & Wyatt, 1984).

More compelling are studies of CSF metabolites. Unfortunately, these suffer from small sample sizes and inadequate normal controls. Shetty and Chase (1976) did not find decreases in homovanillic acid (HVA) in CSF, but Shaywitz, Cohen, and Bowers (1977) did. Decreased HVA levels emerged only when expressed in relation to probenicid, which itself varied between groups (Shaywitz *et al.*, 1977). Reimherr, Wender, Ebert, & Wood (1984) also found a trend toward lower levels of HVA among ADD, Residual Type, patients who were favorable responders to methylphenidate.

The fact that peripheral measures of catecholamine metabolism are inadequate measures of CNS activity does not affect the validity of these findings; but further research must determine the basis of any alterations found (Kopin, 1978, 1981; Kopin *et al.*, 1984).

Catecholaminergic Enzymes

Many of the methodological problems in metabolite studies also plague enzyme studies. Do enzyme activity levels in the periphery reflect CNS levels? Recent evidence suggests not, for the most widely studied catecholaminergic enzyme, monoamine oxidase (MAO) (Young, Laws, Sharbrough, & Weinshilboum, 1986). MAO exists in two forms, A and B, both present in human brain; platelets contain only MAO B. Young and his colleagues studied both human and platelet levels of MAO A and B in epilepsy patients during neurosurgery. There was no correlation between platelet MAO B and cortical levels of MAO A and B.

Simple group comparisons between ADD and control patients have revealed no enzyme abnormalities. Three enzymes have been studied: MAO, COMT (catechol-*O*-methyltransferase), and DBH. Shekim, Davis, Bylund, Brunngraber, Fikes, and Lanham (1982) have reported platelet MAO to be decreased, but we have not been able to replicate this finding (Bowden, Deutsch, & Swanson, 1988). Plasma DBH activity appears normal (Bowden, *et al.*, Deutsch, 1983; Mikkelsen, Lacke, Brown, Ziegler, & Ebert, 1981). Gustavson, Rasmussen, Floderus, Ross, Bille, and Wetterberg (1983) have reported normal erythrocyte COMT levels in children and adults with minimal brain dysfunction.

Subgrouping ADD children by secondary diagnosis or family history of ADD may reveal differences (Bowden, Deutsch, & Swanson, 1988; Deutsch, 1983; Deutsch, Swanson, Warsh, & Farkas, 1984; Quinn & Rapoport, 1974; Rapoport *et al.*, 1977). For instance, conduct disorder has been associated with decreased plasma DBH levels (Rogeness, Hernandez, Macedo, & Mitchell, 1982; Rogeness, Hernandez, Macedo, Mitchell, Amrung, & Harris, 1984; Rogeness, Hernandez, Macedo, Amrung, & Hoppe, 1986). Our Toronto studies of ADD probands bore this out: boys with conduct disorder were found to have lower DBH levels than those without conduct disorder (Bowden *et al.*, 1988).

Animal Models

Several animal models have been developed for locomotor hyperactivity (see the review by Teicher & Baldessarini, 1987). The most widely studied model involves treating rats with 6-OH-dopamine (6-OHDA), a substance which depletes catecholamines and produces hyperactivity (Iversen & Iversen, 1975). Both methylphenidate and amphetamine reverse the behavioral effects of the chemical lesion (Shaywitz *et al.*, 1976).

Hyperactivity seems to be a frequently elicited manifestation of focal cerebral damage in rats. Robinson (1979) was able to induce hyperactivity by generating ischemia in the territory of the right middle cerebral artery, but not the left. Lesions limited to the cortex of the right hemisphere were sufficient to induce hyperactivity, as well as depletion of norepinephrine and dopamine (Pearlson, Kusos, & Robinson, 1984). Yet these rat studies were not targeted at the pathogenesis of ADD, and, in any case, evidence for a right-lateralized neuropsychological deficit in ADD is lacking.

Locomotor hyperactivity is no longer considered the core of the ADD syndrome: rather, problems in attention and behavioral inhibition are currently emphasized. Lesions in several locations (e.g., globus pallidus, substantia nigra, and pontine reticular formation) are known selectively to impair those functions in rats (Thompson, Harmon, & Yu, 1985).

Porrino and Lucignani (1987) argued that the intact rat is an acceptable model for studying the locus of stimulant action in ADD. They based this on the assumption that the effect of stimulants in ADD children is quantitatively the same as on normal children (Rapoport *et al.*, 1978). This position is defensible if one makes allowances for the effect of behavioral base-state on the degree as well as the direction of stimulant-induced behavioral change (Kinsbourne, 1985). When normal rats are given dextroamphetamine (Porrino, Lucignani, Dow-Edwards, & Sokoloff, 1984) or methylphenidate (Porrino & Lucignani, 1987), the nucleus accumbens exhibits a selective metabolic enhancement. Its connections, interposing the nucleus accumbens between cerebral and limbic afferents and efferents to motor-control centers, would seem strategic for correcting attentional/motivational imbalances, regardless of their origins.

Rodent studies have clarified aspects of the functional anatomy and ontogeny of catecholamine systems, but their relevance to ADD is controversial (Brown, Ebert, & Minichiello, 1985). An animal model is required that affords a more sophisticated behavioral repertoire for mimicking ADD symptomatology and for greater similarity between human and animal neuroanatomy. A rhesus model may have been successful in these respects. Brozoski, Brown, Ptak, and Goldman (1979) demonstrated a deficit in spatial-delayed alternation after DA depletion. They were able to reverse the

deficit by use of DA agonists. (Chemical lesions of NE and 5-HT did not produce this effect.) A lesion in the prefrontal cortex was sufficient to produce this behavioral deficit, one which may characterize both frontal lobe damage and ADD (Evans, Gualtieri, & Hicks, 1986).

Proponents of animal models of ADD rely upon the effect of stimulant drugs in correcting the behavioral abnormality that characterizes the model animal. This line of reasoning assumes that stimulants exert a specific effect upon the locus of disordered neural function. This may not be so; the drugs may exert their corrective effect on another system altogether. A recent study on frontal lobe hypofunction illustrates this point: Lou, Henriksen, and Bruhn (1984) found relatively less blood flow in the frontal regions in ADD than in control children. Although it corrected the behavioral abnormality, methylphenidate did not increase frontal blood flow; instead, it increased flow in the medial basal ganglia region.

The notion that neurotransmitter agonists control dimensions of behavior that are specific but override diagnostic categories has received recent illustration by studies of the serotonin system (van Praag *et al.*, 1987). If this notion were correct, stimulant effects might be unreliable guides to the brain basis of ADD.

Alternative Biochemical Models

No clear-cut biochemical abnormality has emerged in ADD. The catecholamine system may be implicated, but there are broad uncertainties as to how an imbalance might be manifested. The staple measures of biochemical psychiatry, metabolites and enzymes, may prove inadequate.

Direct *in vivo* imaging of neurotransmitter activity at the receptor level, now possible using positron emission tomography (Wong *et al.*, 1987), may help define the imbalance. Radioligands can be chosen to test specific hypotheses regarding the functional activity of neurotransmitter systems. Eventually, these techniques may be applied safely to children. As brain imaging becomes more resolute, it may reveal abnormal patterns of activity in ADD. More specific hypothesis of localization of function may also be tested using these techniques. It may become possible to test the frontal lobe hypofunction hypothesis (Evans *et al.*, 1986; Lou *et al.*, 1984; Mattes, 1980) with regard to specific neurotransmitter systems.

It may also be necessary to adopt alternative models for biochemical abnormalities. There may be abnormal *variability* rather than static overactivity or underactivity of a neurotransmitter system. Recent pharmacological findings suggest that ADD children suffer from a disorder of homeostasis (Hicks, Gualtieri, Mayo, Schroeder, & Lipton, 1985; Kinsbourne, 1985). Perhaps what is deficient at the biochemical level is a system of reciprocal balances. Stimulant medication may then correct the imbalance, not by changing neurotransmitter levels directly, but by facilitating stabilizing feedback mechanisms.

Given a homeostatic model, variability of catecholamine levels would be more informative than mean values. Mikkelsen *et al.* (1981) addressed the issue of variability in a study of plasma NE levels. Mean NE concentration was normal in ADDH children; but the intrasubject coefficient of variation for NE was higher than normal. Such a finding would disconfirm a traditional catecholamine hypothesis, but would support a homeostatic model for ADD. We are still faced with problems of heterogeneity. Conceivably, several homeostatic mechanisms are subject to perturbation.

Latent Structure Analysis and the Search for Biochemical Markers

As described above, subgrouping based on latent trait status may provide increased power to uncover biochemical abnormalities. This strategy may sharpen our thinking on ADD. We are compelled to consider what might be transmitted, e.g., cortical disinhibition (Conners, 1969), aberrant response to reinforcement (Wender, 1971), or imbalanced homeostatic mechanisms (Kinsbourne, 1985). And how might the latent trait set into motion its chain of consequences? Genetic latent structure analysis may provide a preliminary framework for constructing biologically interpretable models of ADD.

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CHAPTER 9

Conduct Disorders

Barclay Martin and Jeffrey A. Hoffman

Children who do not conform to society's rules and expectations have long been a concern to mental health professionals as well as to the general public. Adolescent delinquents, perhaps because their troublemaking can be particularly disturbing, have been given special attention, as witnessed by such historic events as the establishment of the Juvenile Psychopathic Institute by William Healy in 1909. In the past, such terms as *constitutional psychopathic inferiority* and *moral imbecility* have been used to refer to both adults and older children who had severe and chronic antisocial tendencies. More recently, the term *conduct problem* or *conduct disorder* has been applied to a wider range of antisocial behaviors in preadolescent as well as in adolescent children. Three current diagnostic systems include rather similar definitions.

Categorical Approaches: Formal Diagnostic Systems

The most widely used diagnostic system in the United States is the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III-R) (American Psychiatric Association, 1987). For a diagnosis of conduct disorder DSM-III-R requires a disturbance of conduct lasting at least six months, during which at least three of the following have been presented:

1. Has stolen without confrontation of a victim on more than one occasion (including forgery)
2. Has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning)
3. Often lies (other than to avoid physical or sexual abuse)
4. Has deliberately engaged in fire-setting
5. Is often truant from school (for older person, absent from work)
6. Has broken into someone else's house, building, or car
7. Has deliberately destroyed others' property (other than by fire-setting)
8. Has been physically cruel to animals
9. Has forced someone into sexual activity with him or her
10. Has used a weapon in more than one fight
11. Often initiates physical fights
12. Has stolen with confrontation of a victim (e.g., mugging, purse-snatching, extortion, armed robbery)
13. Has been physically cruel to people

Three types of conduct disorders are listed in this system:

1. *Group type*. The predominance of conduct problems occurs mainly as a group activity. Ag-

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gressive physical behavior may or may not be present. (In the DSM-III, similar to Socialized, Nonaggressive type.)

2. *Solitary aggressive type.* The predominance of aggressive physical behavior is initiated by the person, not as a group activity. (In the DSM-III, similar to Undersocialized, Aggressive type.)

3. *Undifferentiated type.* Includes a mixture of clinical features that cannot be classified as either of the above.

Two other classification systems might be briefly mentioned. The World Health Organization (WHO) Classification System (Rutter *et al.*, 1969) has only a single category for Conduct Disorder that includes some forms of illegal actions (e.g., theft or property destruction) as well as fighting, bullying, and cruelty to animals. The International Classification of Disorders (ICD-9) (an Appendix in the DSM-III) has a category called Disturbance of Conduct Not Elsewhere Classified under which four subcategories are listed: Unsocialized Disturbances of Conduct (e.g., defiance, disobedience, aggression, destructive behavior, solitary stealing, lying); Socialized Disturbance of Conduct (have acquired values of a delinquent peer group to whom they are loyal and with whom they characteristically steal, play truant, etc.); Compulsive Conduct Disorder (antisocial behavior is compulsive in nature, such as kleptomania); Mixed Disturbance of Conduct and Emotions (antisocial behavior is accompanied by considerable anxiety or misery). The first two categories clearly parallel the solitary aggressive and group types in the DSM-III-R.

Of historical interest is the classification system of the Group for the Advancement of Psychiatry (1966) which, taking a psychodynamic perspective, used the term Tension-Discharge Disorder to refer to conduct-disordered children. This category was divided into two subcategories. The first, Impulse-Ridden Personality, describes the undersocialized child who shows shallow relationships and very low frustration tolerance associated with a basic defect in the control of aggressive and sexual impulses. The Neurotic Personality Disorder describes the socialized child whose relationships are warmer and more meaningful, and whose antisocial behavior is more of an unconscious symbolic reaction to the intensification of conflict than it is a sudden discharge of impulses.

How reliably can these categories be diagnosed? The originators of the DSM-III (American Psychiatric Association, 1980) published results of preliminary field tests on 126 children and adolescents in which two clinicians evaluated the same patient (sometimes in joint interviews). For the overall category of conduct disorder, the kappa coefficient was .61. A kappa of about .70 would ordinarily be seen as a desirable level of reliability. Reliabilities for the subcategories of conduct disorder were not reported. Strober, Green, and Carlson (1981) had clinicians *jointly* interview adolescents admitted to a hospital and reported a kappa of .75 for overall conduct disorder and kappas of .86 for undersocialized aggressive disorders (similar to solitary aggressive type in DSM-III-R) and .60 for undersocialized, nonaggressive disorders (similar to group type in DSM-III-R). The largest study was performed on 195 successive admissions of children and adolescents to an inpatient unit by Werry, Methven, Fitzpatrick, and Dixon (1983). They found kappas of .53 for overall conduct disorder, .59 for the undersocialized aggressive (solitary type in DSM-III-R), and .32 for the socialized, nonaggressive subcategories (group type in DSM-III-R). These results suggest that the reliability of diagnosing the subtypes of conduct disorders is only moderate, although it is possible that the revised DSM-III-R categories may be more reliable. Gould, Shaffer, and Rutter (1984) also found relatively low levels of reliability for the subcategories of ICD-9.

Dimensional Approaches

In the categorical approach, an individual is diagnosed as either fitting a category or not, although there may be some provision for judging severity within a category. In a dimensional approach, individuals are considered to vary in a continuous way in the degree that a characteristic is present. Thus, antisocial tendencies can vary from a small to a large amount, and any cutting point on the dimension that is used to define the antisocial child can be seen as arbitrary.

One of the earliest attempts to identify dimensions of childhood psychopathology was that of Hewitt and Jenkins (1946). These authors intercorrelated 45 behaviors rated from case records on 500 children who were referred to a Chicago child-guidance clinic and who formed clusters of traits that they labeled unsocialized-aggressive, socialized delinquent, and overinhibited—the first two labels foreshadowing those used later in the DSM-III. Quay (1986a) provides an excellent summary of some 61 studies published in the last 40 years that used a variety of assessment procedures, such as behavior observations or ratings from case records

made by trained professionals, and ratings by various untrained informants, such as the self, parents, teachers, or peers. The children in these samples varied in age from 3 to 18 years and represented normal public school classes as well as various kinds of clinically defined populations. Quay included only samples of boys or mixed samples of boys and girls in which the sexes were not analyzed separately. He concluded that a dimension reasonably labeled undersocialized aggressive conduct disorder (solitary aggressive type in the DSM-III-R) appears in almost all studies. The characteristics associated with the dimension in most studies were: fighting, hitting, assaultive; disobedient, defiant; temper tantrums; destructiveness; impudent; uncooperative, stubborn; attention-seeking; bullying, threatening; disruptive; boisterous, noisy. Quay (1986a) also found strong support for a dimension labeled socialized aggressive conduct disorder, but primarily for adolescent age groups. The characteristics that were associated with this dimension across the most studies were: has "bad" companions; truant from school; truant from home; steals in company with others; and belongs to a gang.

Since Quay's (1986a) summary includes only boys or mixed-sex samples, we cannot conclude that the two dimensions are equally well confirmed for girls, although Achenbach and Edelbrock (1979) and Jenkins and Glickman (1947) found essentially the same two dimensions for samples of girls. The studies of Achenbach (1978) on 6- to 11-year-old boys and Achenbach and Edelbrock (1979) on boys 12 to 16, and girls 6 to 11 and 12 to 16, are worth special attention because they involved large groups (450 girls and 450 boys) sampled from some 25 different mental health settings in the Eastern United States, divided into the preadolescent and adolescent age groups shown above, and age-matched normal children to provide a standardization reference. Parents made the ratings. The confirmation of these two dimensions for both boys and girls and both age levels in these studies should probably be given special weight.

Cluster analytic procedures have also been applied to behavioral characteristics of children in an effort to identify groupings of children as opposed to groupings of behavioral characteristics. These typologies are similar in concept to the categorical diagnostic approach except that they ordinarily involve more objective assessment of child characteristics as well as more precisely defined types. Edelbrock and Achenbach (1980) and Lessing, Williams, and Gill (1982) have identified clusters resembling the solitary aggressive type and the group type in clinic-referred children. Edelbrock and Achenbach (1980) used the same sample of children as in the Achenbach and Edelbrock (1979) factor-analytic studies, and found these two types in both sexes and at both age levels.

In case all of this sounds too neat, some qualifications are in order. In the factor-analytic studies, the dimensions are relatively uncorrelated, which means that it is just as likely that children will be high on both solitary aggressive and group type conduct problems as low on one and high on the other. This implies that a number of children will show characteristics of both dimensions. The cluster analytically derived types do suggest some degree of differentiation between types, but even here two different types can have some characteristics in common.

The typal characteristics, or profiles, are not precisely the same in different studies or in different sex and age groupings. For example, in the group we identify in Edelbrock and Achenbach's (1980) study as corresponding to the solitary aggressive type in the 6- to 11-year-old boys, there are also peaks on depression and social withdrawal. Furthermore, the decision to cluster into many homogeneous groups or a few heterogeneous groups (with resulting great variability within a group) is relatively arbitrary. When previously identified types are applied to new samples of children, a number of children will probably not fit any of the types.

In conclusion, although factor-analytic studies suggest that children's behavior can reasonably be seen to fall along these two dimensions (and, of course, other dimensions not considered here) and cluster-analytic studies suggest that empirically derived groupings correspond roughly with two of the DSM-III-R categories of conduct disorders, these dimensions and types should probably be viewed as rough approximations, and depending on a given research or treatment issue, it may be desirable to focus on more narrowly defined syndromes or highly specific behaviors.

Relationship between Conduct Disorders and Other Disorders

Although we know in some abstract sense that these diagnostic categories rarely exist in pure form, the very existence of a categorical term, such as conduct disorder, and book chapters carrying that label, promotes a reification that few can resist. Conduct disorders may be no worse than other cate-

gories in this respect, but it is clear that other symptoms and disorders often occur simultaneously with the conduct disorder syndrome. The attention deficit disorder is a case in point. Although many factor-analytic studies have found attention deficit disorders (frequently under the older term of hyperactivity) to emerge as a factor separate from the undersocialized (solitary) aggressive dimension (e.g., Achenbach & Edelbrock, 1978; Lahey, Stempniak, Robinson, & Tyroler, 1978), conduct-disordered children, nevertheless, often manifest symptoms of the attention deficit disorder. For example, Stewart, Cummings, Singer, and DeBlois (1981) assessed consecutive admissions to a Child Psychiatry Clinic and found that 61% of the children were diagnosed as hyperactive (attention deficit), unsocialized (solitary) aggressive, or both. Three-fourths of the unsocialized (solitary) aggressive children were also diagnosed hyperactive and two-thirds of the hyperactive children were also diagnosed unsocialized (solitary) aggressive. See Chapter 7, in this volume, for further discussion of attention deficit disorders and their relation to aggressive behavior.

Other disorders may also occur with some frequency in association with conduct disorders. For example, Puig-Antich (1982) found that a third of a sample of preadolescent boys diagnosed with depressive disorders also fit the criteria for conduct disorders. Lewis, Lewis, Unger, and Goldman (1984) found a little over a half of a sample of psychiatrically hospitalized adolescents were presently or had been in the past diagnosed as conduct disordered. Of those so diagnosed, 56% had at some time been also diagnosed as psychotic, 35% as attention deficit disorder, 26% as retarded, 61% as learning disability, and 45% as neurosis or adjustment reaction. Of course, one would expect that adolescents hospitalized in a psychiatric facility would manifest disorders of these types. The point is that the characteristics we associate with conduct disorders may often occur in the context of other symptoms, especially in more severe cases A dimensional rather than a categorical conception of conduct disorders may be more useful in this respect. Certain psychosocial-biological factors can lead to varying degrees of the conduct-disorder syndrome (or one of its subtypes), which may to varying degrees be associated with other symptoms.

Prevalence

Prevalence figures will vary as a function of how the assessments are made as well as the population sampled. Lapouse and Monk (1957) found, for example, that mothers of a representative sample of 6- to 12-year-olds considered 31% of boys and 21% of girls to be behavior control problems, and Werry and Quay (1971) found that 6 kindergarten, first, and second grade teachers reported rates of 31% and 26% for fighting and disobedience, respectively, for boys, and 6% and 11% for girls on the same traits. However, when more stringent criteria are used to assess conduct disorders, the rates are considerably lower. Rutter, Cox, Tupling, Berger, and Yule (1975) and Rutter, Tizard, and Whitmore (1970) reported for 10- and 11-year-old British youths an overall rate of 4% in a rural area, and Graham (1979) reported an overall rate of 8% in an urban area. The rates for boys were about three times those for girls. In a large sample of 7-year-olds in Dunedin, New Zealand, McGee, Silva, and Williams (1984) found 9% of the boys and 4.6% of the girls to have a chronic antisocial disorder as rated by parents and teachers. No epidemiological comparisons have been made separately for the subtypes of conduct disorders. In samples limited to children seen in inpatient or outpatient clinics, conduct disorder is the most prevalent form of disorder, with about three times as many boys as girls being so diagnosed (Cerreto & Tuma, 1977; Kotsopoulos & Nandy, 1981).

Stability and Course

We are concerned here with the extent to which early manifestations of antisocial behavior continue as the child grows older, possible changes in the nature of the antisocial behavior over time, and sex differences in these variables.

Olweus (1979) reviewed research on boys that was published between 1935 and 1978 and concluded that there was evidence for considerable consistency in aggressive behavior over time intervals varying from 6 months to 21 years. The average stability correlation computed for a total of 24 stability correlations was .55. The stability correlations tended to decrease as the time interval increased and at younger ages of first assessment. For example, when age of first assessment was 9-years-old or less and the time of reassessment was 10 years or more later, the four stability correlations for these studies ranged from .26 to .48. More recently, Moskowitz, Schwartzmann, and Ledingham (1985) found stability correlations of around .50 for boys' aggressive behavior over 3-year intervals in three samples—from Grade 1 to Grade 4, Grade 4 to Grade 7, and Grade 7 to Grade 11. There

is, then, for boys some consistency in aggressive tendencies over rather long periods of time.

Aggressive behavior covers a broader range of behavior than the more narrowly defined categories of conduct disorder, and we turn now to studies that relate more specifically to the diagnosis of conduct disorder or its subtypes. Robins (1966) reassessed 500 children seen in child guidance clinics and 100 matched control children when they were adults. For males, 71% of those diagnosed antisocial as a child had been arrested as an adult for a nontraffic offense, compared with 22% of the controls; for females, the corresponding percentages were 40% and 0%. Similar studies were performed by Robins, West, and Herjanic (1975) on young black men and by Robins (1974) on Vietnam veterans. In general, Robins concluded that adults who show antisocial behavior have, in most cases, shown antisocial behavior as children, but most antisocial children do not become antisocial adults. Also she found no particular type of childhood antisocial behavior to be an especially good predictor of adult antisocial behavior. The greater the variety of childhood antisocial behavior, whatever its nature, the greater was the likelihood that the children would display antisocial behavior as adults. Social class did not contribute much to prediction in these studies.

A number of more recent studies have further substantiated the conclusion that either older children or adults diagnosed as antisocial were showing antisocial tendencies at an earlier age. Richman, Stevenson, and Graham (1982) found boys of age 8 who had been diagnosed as having a conduct disorder were more likely at age 3 to have been rated as difficult to control, overactive, and as having poor relations with siblings. Farrington (1978) followed a sample of inner-city London boys from age 8 to about age 22. For boys rated aggressive at ages 8 to 10, 14% were found among 27 violent delinquents defined on the basis of conviction records up to age 21, compared with 4.5% of those not rated aggressive at ages 8 to 10. Looking backward, 48% of the violent delinquents had been rated aggressive at ages 8 to 10 and 70% at ages 12 to 14. Roff and Wirt (1984) followed a sample of 1,224 boys and 1,228 girls and found aggression in third-grade through sixth-grade students to correlate with both adolescent delinquency and adult criminality in boys but not for girls. After reviewing research on the stability of antisocial and delinquent behavior, Loeber (1982) concluded that four factors were associated with the tendency for children who showed antisocial behavior to persist in their behavior and become delinquent or criminal at a later time: (1) extremely high rates of antisocial behavior (e.g., Craig & Glick, 1963; Mitchell & Rosa, 1981; Osborn & West, 1978; West & Farrington, 1977); (2) a display of antisocial behavior in more than one setting, for example, at home, at school, and in the community (e.g., Mitchell & Rosa, 1981; Schachar, Rutter, & Smith, 1981); (3) a variety of antisocial behavior (e.g., Loeber, Schmaling, & Patterson described in Loeber, 1982; Robins & Ratcliff, 1979); and (4) an early onset of antisocial behavior (e.g., Farrington, 1978; Hamparian, Schuster, Dinitz, & Conrad, 1978; Robins, 1966; Wolfgang, Figlio, & Sellin, 1972). Few longitudinal studies distinguish between the socialized (group) and undersocialized (solitary) aggressive types. There is some indication that preadolescent children who steal, lie, and are truant (characteristics of the socialized [group] disorder) are more likely to become adolescent delinquents than are preadolescents characterized by undersocialized (solitary) aggression (Mitchell & Rosa, 1981; Moore, Chamberlain, & Mukai, 1979). Moore *et al.* (1979), for example, studied later court-recorded offenses for three groups of adolescent boys who primarily showed aggression at home, were stealers, or were "normal." Eighty-four percent of the stealers, 24% of the aggressive boys, and 21% of the normal boys had committed serious juvenile offenses as adolescents. These results contrast with those of Henn, Bardwell, and Jenkins (1980) who used a different research strategy. They examined the adult criminal records of individuals who had previously been placed in state institutions for delinquent boys. Adult criminal convictions occurred in 62.7% of individuals previously diagnosed as undersocialized (solitary) aggressive and 42.1% for individuals previously diagnosed as socialized (group) type. They also had classified a third group as undersocialized conduct disorder-unaggressive—defined differently from the DSM-III category and resembling the neurotic delinquent category referred to previously in which a mixture of chronic disobedience and stealing in the home is accompanied by fearfulness timidity, and lonesomeness. Fifty-seven percent of these individuals had adult criminal convictions. In discussing the results of this study, Quay (1986b) suggests that the socialized group with their unimpaired cognitive skills and good social skills may be better able to profit from experience and change their behavior. The undersocialized (solitary) aggressive individuals in the preadolescent years may be troublesome to parents and schools with their fighting and verbal aggression but do not necessarily come to the attention of law-enforcement agencies at that time. However, as the years go by, their lack of

control over verbal and physical aggression may be reflected in criminal assault, resisting arrest, and other illegal activities.

Psychological Correlates

Another criteria that is used to determine the legitimacy of a diagnostic category is the relationship between the disorder and other variables that are thought to correlate with it. There are several psychological features that are considered to be associated with the conduct-disorder diagnoses on which research has been conducted. Unfortunately, because the DSM-III and the DSM-III-R subtype definitions have only recently been developed, most of the studies are based on samples of more general populations, such as conduct-disordered, aggressive, antisocial, or delinquent children and adolescents. Some of these studies distinguish between socialized (group) and undersocialized (solitary) delinquents or aggressive youth, which parallel the aggressive subtypes, but most of the studies represent a fairly broad spectrum of youth with antisocial characteristics. Nevertheless, some validity for the general category of conduct disorder may be established by examining several psychological variables that are thought to be associated with the disorder: these include intelligence and cognitive skills, perceptual processes, impulsivity, need for stimulation, empathy, moral development, and interpersonal relationships.

Intelligence and Cognitive Skills

When socioeconomic status (SES) is not controlled, delinquent (or conduct-disordered) youth are frequently found to have lower intelligence and to show poorer academic performance than nondelinquent youth (e.g., Berman & Siegal, 1975; Dishion, Loeber, Stouthamer-Loeber, & Patterson, 1984; Hirschi & Hindelang, 1977; Rutter, *et al.*, 1970). However, after reviewing the literature, Quay (1986b) concludes that "there is little reason to believe that general intelligence as measured by IQ tests is significantly lower than SES *comparable groups* with and without other disorders for either undersocialized (solitary) or socialized groups" (p. 37) (e.g., Beitchman, Patterson, Gelfand, & Minty, 1982; Rutter, 1964). The distinction between conduct disorder and attention deficit disorder may be important in this respect. McGee, Williams, and Silva (1984) found reading, spelling, and performance IQ to be related to a factor analytically derived dimension of attention deficit but not to a factor of antisocial behavior. In addition, Hogan and Quay (1984) concluded, after reviewing the appropriate literature, that controlled research on discrepancies between verbal and performance IQ scores indicated that the discrepancies were not of a magnitude to differentiate between the conduct-disorder subtypes and other disorders (e.g., Hecht & Jurkovic, 1978). With respect to academic performance, Rutter *et al.* (1970) found that low reading skills continued to distinguish conduct-disordered children with intelligence held constant by limiting the sample to children of average IQ. SES, however, was not controlled. Stevenson, Richman, and Graham (1985) found that poor language "structure" (e.g., speech limited to nouns and verbs, or only 2 or 3 words in an expression) at age 3 predicted behavioral problems at age 8 with SES controlled. However, the prediction was considerably stronger for neurotic type symptoms than for antisocial behavior. Overall, then, we must conclude that though samples of conduct-disordered youth are likely to have lower than average intelligence and academic achievement, there is not strong and consistent evidence that these differences will hold up when the conduct-disordered samples are compared to groups carefully matched on SES.

Perceptual Processes

Writing from a psychodynamic perspective, Burlingame (1973) suggested that conduct-disordered adolescents frequently use defense mechanisms of projection and denial by which they deny responsibility for their aggressive behavior by perceiving the responsibility as belonging to others. The Group for the Advancement of Psychiatry (1966) also suggested that the impulse-ridden personality exhibited the primitive defense mechanisms of denial, projection of hostile feelings, and rationalization. Although no research has been conducted pertaining directly to these psychoanalytically derived concepts, there is some research on perceptual processes that lends some empirical support to these notions.

Nasby, Hayden, and DePaulo (1980) had emotionally disturbed boys in residential treatment view filmed vignettes of a woman portraying either anger or affection and either dominance or submission. They found that more aggressive boys tended to perceive all of the presentations as reflecting hostility and dominance, even when the presentations were meant to be benign. This would suggest a tendency to overperceive aggression in others, which may partly account for their tendency to "project" the blame. Dodge and Frame (1982)

summarized three studies that consistently revealed a bias on the part of aggressive boys to overattribute hostile intentions to peers. These biased attributions were identified as direct precedents to aggressive responses. It was noted that the aggressive boys were frequently the targets of peers' aggressive behavior, which suggested some experiential basis for their biased attributions. It may be postulated that the tendency to overperceive hostility in others (placing blame) may elicit aggressive responses that reinforce the biased attribution and justify the projection of blame in the mind of the aggressor. A study by Dodge and Newman (1981) further suggests that attentional deficits may play a role in the overattribution of hostile intent. They found that aggressive boys overattributed hostility in unwarranted circumstances only when they responded very quickly, paying less attention to available social cues.

Impulsivity

A limited ability to delay impulses and tolerate frustration is another central feature considered to be related to conduct disorders (Burlingame, 1973; Group for the Advancement or Psychiatry, 1966). As stated above, quick responses were associated with the overattribution of hostility in aggressive boys (Dodge & Newman, 1981). Camp (1977) found aggressive boys to respond more quickly and impulsively on cognitive tasks than nonaggressive boys. Another study comparing delinquent to nondelinquent boys found that the delinquent boys displayed poorer performance on cognitive measures of impulsivity (Doctor & Winder, 1954). Unfortunately, there are no studies relating impulsivity to the specific diagnosis of conduct disorder or its subcategories.

Need for Stimulation

Quay (1965) has hypothesized that at the root of psychopathic behavior is an extreme need for stimulation. DeMyer-Gapin and Scott (1977) found that antisocial children displayed higher initial attention to novel stimuli than to repetitive stimuli, but in contrast to neurotic children, they habituated rapidly to both types of stimuli (photographic slides). Whitehill, DeMyer-Gapin, and Scott (1976) also found that antisocial children showed a decrement in viewing time of slides earlier than neurotic and normal children. Other studies have shown that more psychopathic delinquents have more difficulty sustaining attention and have a greater preference for novel and complex stimuli than do neurotic delinquents (Orris, 1969; Skrzypek, 1969). These findings are difficult to extrapolate to conduct-disorder subtypes, especially in light of the confounding influences of attention deficit problems, but the evidence suggests that there may be a greater need for stimulation among less socialized conduct-disordered youth.

Empathy

Empathy as a construct has not always been conceptualized or operationalized in a consistent way (Rotenberg, 1974). In most empirical research related to conduct disorders, investigators have emphasized the cognitive ability to appraise the cognitive, emotional, or motivational state of other people. Occasionally, however, the term refers to the direct experiencing of the same emotion in oneself as shown by another person. Deficits in either the cognitive or the more directly affective types of empathy have been considered important in the development of antisocial behavior, usually in terms of explaining why the antisocial person can deceive and injure others without experiencing any remorse. Research comparing undifferentiated samples of delinquents with nondelinquents has been slightly inconsistent but generally suggests that delinquents are less cognitively empathic (see Ellis, 1982, for a review). Two studies distinguished subtypes of delinquency and both found that psychopathic delinquents (comparable to the undersocialized or solitary aggressive conduct disorder) showed less cognitive empathy than either nondelinquents or subcultural delinquents (comparable to socialized or groups conduct disorder) (Ellis, 1982; Jurkovic & Prentice, 1977).

Moral Development

Research suggests that conduct-disordered and delinquent youth reason at a somewhat lower level of moral development than do other youth, generally revealing a preconventional level which focuses on an avoidance of punishment by authority figures (Bear & Richards, 1981; Jurkovic & Prentice, 1977). See Chapter 13, in this volume, for a more comprehensive discussion of morality and conduct disorders.

Interpersonal Relationships

Defective interpersonal relationships are another feature thought to characterize conduct-disordered youth. Parental rejection, inconsistent management with harsh discipline, early institutional living, and marital conflict and frequent shift-

ing of parental figures are a few of the factors listed by the DSM-III that are thought to predispose children to the undersocialized (solitary) pattern of behavior; whereas father absence and father alcoholism have been associated with the socialized type. See Chapter 14 in this volume for a more detailed review of social and familial correlates of aggressive behavior in children. Given these kinds of destructive and disruptive experiences in early family relationships, it is not surprising that these youths exhibit difficulties in relating to others.

Interpersonal skill deficits as indicated by peer rejection have been shown to be predictive of later court-reported delinquency in two studies (Roff & Sells, 1970; West & Farrington, 1977). Institutionalized delinquents (Freedman, Rosenthal, Donahue, Schlundt, & McFall, 1978) and antisocial adolescents (Dishion *et al.*, 1984) showed less interpersonal problem-solving skills on a role-play problem-solving task than comparison groups. Panella and Henggeler (1985) studied peer relations among black adolescent males classified as conduct disordered, anxious-withdrawn, and well-adjusted. They found that youths identified as conduct disordered displayed less social competence and less positive affect than the well-adjusted adolescents. They attributed the problems in friendship relations of conduct-disordered adolescents to difficulties in exchanging sensitive, responsive, and positive behaviors. Konstantareas and Homatidis (1985) studied dominance hierarchies in conduct-disordered and normal children. They found that the dominance hierarchies were less stable and associated with greater intragroup conflict for the conduct-disordered children, compared to the normal group. We have previously mentioned the work of Dodge and Frame (1982) that demonstrated the tendency for aggressive boys to overattribute hostile intentions to others—a characteristic likely to interfere with effective interpersonal relationships. In addition, Richard and Dodge (1982) found that aggressive boys verbalized fewer potential solutions to interpersonal problems than did popular boys. In conclusion, research on peer relations clearly suggests deficits in social skills in conduct-disordered children and adolescents. More research, however, would be useful in assessing the hypothesized differences in the quality of attachments between the socialized and undersocialized types.

Conclusions

The DSM-III-R diagnosis of conduct disorder can be assigned with only moderate reliability, and the subcategories with somewhat less reliability. Factor- and cluster-analytic procedures provide some support for the subdimensions or subcategories of group (socialized) and solitary (undersocialized) aggressive disorders. Other features of conduct-disordered youth are: early presence of antisocial tendencies, an overattribution of hostility to others, deficits in cognitive empathy and interpersonal relationships, and a level of moral development that focuses on an avoidance of punishment by authority figures.

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CHAPTER 10

Behavioral Genetics and Aggressive Behavior in Childhood

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Behavioral genetics has contributed to the study of psychopathology in three ways: (1) It has provided a theory of the genetic and environmental origins of individual differences in behavioral pathology, (2) devised methods that can disentangle the influence of nature and nurture, and (3) produced data that converge on the conclusion that hereditary influences are important in psychopathology. The new subdiscipline of developmental behavioral genetics is particularly relevant to the field of developmental psychopathology because its focus is on genetic change as well as continuity during development (Plomin, 1986a). The purpose of this chapter is to introduce developmental behavioral genetics, using aggressive behavior in childhood as an example of this approach.

Prior to reviewing relevant behavioral genetic research, two prerequisite issues are discussed: the normal distribution of individual differences and quantitative genetics.

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Individual Differences and the Normal Distribution

One advantage in introducing behavioral genetics to researchers who are interested in developmental psychopathology is that they are interested in individual differences, whereas most other developmentalists are not. In terms of behavioral genetics this is important because behavioral genetics is relevant only to the analysis of differences among individuals in a population. For example, the *Handbook of Child Psychology* (Mussen, 1983) presents a recent and representative sampling of theory and research in the field of developmental psychology. Of the 2,926 pages of text (excluding references, outlines, and notes), 78% of the pages are devoted predominantly (i.e., more than half the page) to group differences. Of the 48 chapters, 41 include more pages on group differences than on individual differences. Moreover, 19 chapters include not a single page on individual differences, and 4 other chapters consider individual differences on 2% or less of their pages. These chapters—primarily on perception, learning, cognition, and language—focus on universals of human development, that is, species-typical developmental patterns. Only 8 chapters in the handbook devote more than 50% of their pages to individual differences; one of these is the chapter on developmental psychopathology.

It is interesting that theories of aggression are normative theories, such as the ethological/innateness theory, drive theory, social learning theory, and the social cognition perspective (Parke & Slaby, 1983). Respectively, these theories emphasize innateness, frustration as drive, observational learning, and information processing. Only by loose association do these theories pertain to individual differences in aggressiveness. For example, from the ethological/innateness position, it could be argued that if aggression has been subject to successful selection in our species, then differences in aggression among individuals within our species are also likely to be genetic in origin. Similarly, the other theories could argue that differences in reinforcement contingencies, aggressive models, and cognitive strategies might underlie differences in aggressiveness among individuals. To the contrary, however, there is no necessary relationship between the explanation of average behavior in a species and the explanation of individual differences. The innateness hypothesis is a good example of this point. Behaviors that are subject to strong directional selection will in fact show little genetic variation; thus, differences among individuals that remain for highly selected characteristics must be primarily environmental in origin. In the same way, it is likely that human beings learn to behave aggressively by means of processes such as instrumental learning, observational learning, and social cognition; however, this does not imply that these are the mechanisms responsible for individual differences in aggressiveness. An analogy from the cognitive realm is vocabulary. Vocabulary is one of the most highly heritable cognitive tests (Plomin, 1987a). This does not mean that some individuals are born with more words than others; we learn words, but some individuals pick them up more readily and are more fluent in using them.

One other point needs to be made concerning individual differences. We believe that the most parsimonious approach to developmental psychopathology is to assume that behavioral problems represent the extremes of a normal distribution of individual differences, unless it can be shown that, descriptively or etiologically, the extremes of the distribution differ from the rest of the distribution. Indeed, we suggest that it is likely that frequently occurring behavioral problems in childhood represent the extreme ends of the normal distribution and that the causes and correlates at the tail of the distribution are likely to be more extreme versions of the etiologies of the rest of the distribution. Despite its intrinsic interest in individual differences, research in developmental psychopathology focuses primarily on average differences between affected and normal groups rather than quantitative differences between individuals, even though research in developmental psychopathology, when compared to psychiatric research, is much less reliant on categorical diagnoses based on a medical disease model. Although it is perfectly reasonable to focus on "affected" individuals, unless such problems are studied in the context of the normal distribution, it is not possible to determine the extent to which such problems represent the extreme end of the normal distribution.

Thus, although we recognize that etiologies of the tail of a distribution can differ from etiologies for the rest of the distribution, in our review we include research on the normal distribution because parsimony suggests that we assume that their etiologies are similar until evidence to the contrary is found.

Quantitative Genetics

Behavioral genetics includes chromosomal analyses as well as single-gene approaches, such as pedigree analyses that test specific models of genetic transmission and linkage analyses that attempt to localize a gene on a chromosome by assessing the co-occurrence of a particular probe whose chromosomal location is known and a disorder. A report that a high incidence of males with an extra Y chromosome was found among criminals in a maximum security prison (Jacobs, Brunton, Melville, Brittain, & McClement, 1965) implied a chromosomal basis for criminal behavior. However, the frequency of XYY males in the general population is only about one per thousand; the vast majority of criminals are not XYY individuals, and most (perhaps 98%) XYY individuals are not criminals. No link to aggressive or violent criminal behavior *per se* has been established (Schiavi, Theilgaard, Own, & White, 1984). Moreover, it has been suggested that the greater occurrence of criminal records among XYY individuals is due to lower intelligence (Witkin *et al.*, 1976). No other chromosomal anomalies other than XYY have been suggested to relate specifically to aggressive behavior or associated disorders.

Linkage analyses have been vastly improved by increasing numbers of polymorphic probes that were discovered using recombinant DNA techniques (e.g., McGuffin, 1987). Using these tech-

niques, the gene for Huntington's disease has been mapped to chromosome 4 (Gusella *et al.*, 1983) and the race is on to discover the gene itself and then, from the gene, to determine the gene product responsible for Huntington's disease. For decades, classical pedigree analyses have shown that Huntington's disease is due to a single autosomal dominant gene whose expression is scarcely affected by environmental variation. In contrast, classical pedigree analyses have been equally clear in finding no single gene effects for any area of psychopathology. A rare exception to this rule is the recent report of a linkage between manic-depressive psychosis and genetic markers on chromosome 11 (Egeland, Gerhard, Pauls, Sussex, & Kidd, 1987). This study utilized a large pedigree of the Older Order Amish; however, other studies using non-Amish pedigrees have shown that bipolar disorders are *not* linked to chromosome 11 (Detera-Wadleigh, Berrettini, Goldin, Boorman, Anderson, & Gershon, 1987; Hodgkinson, Sherrington, Gurling, Marchbanks, & Reeders, 1987). Similarly, a linkage for schizophrenia with markers on chromosome 5 has been reported in two Icelandic families with high incidence of schizophrenia (Sherrington *et al.*, 1988), but another linkage study of a large Swedish pedigree ruled out the possibility of linkage to chromosome 5 (Kennedy *et al.*, 1988). Such conflicting results indicate genetic heterogeneity. However, it is possible that by focusing on single pedigrees with a high incidence of rare disorders, linkage is found for a major mutation that does not occur in the rest of the population.

It is our belief that behavior and behavioral pathology are not substantially influenced by any single factor—whether a single chromosome, a single gene, a single neurotransmitter or hormone, or a single environmental event. Unlike Huntington's disease for which the necessary and sufficient cause is a single gene whose expression is unaffected by variation in experience, psychopathology is influenced by many genes (polygenic), each with small effects, as well as by nongenetic factors. We disagree with the view that a single gene will be found if a disorder is defined carefully enough. Behavioral genetic research, although suggesting significant genetic influence on the adult psychoses, finds no evidence for single-gene effects and, furthermore, indicates that the provenances of psychopathology include substantial nongenetic factors. One simple example makes this latter point dramatically: The concordance for schizophrenia for identical twins is about .45 (Gottesman & Shields, 1982); a large recent study in the United States suggests that identical twin concordance is closer to .30 (Kendler & Robinette, 1983). Because identical twins are genetically identical, this means that most of the reason why one person is hospitalized as schizophrenic and another is not has to do with nongenetic factors. For childhood disorders, much less is known than for adult psychoses; a first step should be to ask whether genetic influences are important and then whether evidence exists that a single gene may have a major effect (Cloninger, Reich, & Yokoyama, 1983). So far, no major effects of single genes have been found for behavioral disorders of childhood.

Understanding behavioral pathology will require approaches that are probabilistic rather than deterministic and that recognize polygenic influence as well as nongenetic factors. It is for this reason that quantitative genetic theory and methods, which form the foundation for most behavioral genetic research, represent a valuable perspective for the study of psychopathology.

Behavioral Genetic Research on Disorders Related to Aggressive Behavior

We prefer to focus on individual differences in aggressive behavior rather than on DSM-III diagnostic categories in which aggression plays a role for two reasons. First, as indicated above, we believe that dichotomous diagnostic categories are likely to be less useful than studying individual differences in behavior; second, we believe that greater progress will be made in research on more narrowly defined areas of behavior rather than on global diagnostic categories. Concerning the second point, a similar view has been expressed by Achenbach and Edelbrock (1984) specifically in relation to aggression-related diagnostic categories:

> The global distinction between broad-band undercontrolled and overcontrolled behavior, for example, may be useful for general management purposes. Narrow-band hyperactive, delinquent, aggressive, depressed, obsessive-compulsive, somatic, and anxious syndromes, by contrast, may provide a better basis for detecting specific etiologies and prescribing specific treatments. (p. 234)

Although there is no widely accepted system for classifying behavioral problems of children (e.g., Garber, 1984), undercontrolled behavior is generally accepted as a major category. Often referred to as *conduct disorders*, the category in-

cludes heterogeneous behaviors, such as fighting, temper, defiance, destructiveness, and uncooperative or inconsiderate behavior. DSM-III differentiates unsocialized aggression (in which there are disturbed interpersonal relationships) and socialized aggression (aggressive behavior in children who form adequate social attachments). As yet no behavioral genetic studies have attempted to make these diagnostic distinctions. However, some relevant research has been reported in terms of antisocial personality, delinquency, and criminal behavior, although the genetic relationship among these disorders and aggressiveness is not known.

Antisocial Personality

Antisocial personality disorder, formerly called *psychopathy*, is viewed as a psychiatric disorder that emphasizes aggression and chronic antisocial behavior. Its symptoms include adolescent problems, such as running away from home, fighting, trouble at school, and continued problems in adulthood, including criminality, excessive drug use, and failure at job and marriage. Although it is likely that aggression is more integrally involved in antisocial behavior than in criminal behavior, there are very few behavioral genetic studies of antisocial personality *per se*.

Family studies indicate that antisocial personality shows familial resemblance; they also indicate that familial factors relevant to the development of antisocial personality are similar for males and females, even though males are diagnosed about four times more frequently than are females, suggesting that females, for some reason, have a higher threshold for displaying the disorder (Cloninger, Reich, & Guze, 1975; Cloninger, Christiansen, Reich, & Gottesman, 1978). Two adoption studies suggest that familial resemblance for antisocial personality is, to some extent, hereditary. In a study of 18 adopted-away offspring of biological parents who were diagnosed as antisocial, 4 were diagnosed as antisocial; in contrast, no adoptees were diagnosed as antisocial in a matched control group of adoptees whose biological parents had no record of behavioral disorder (Cadoret, 1978). A study in Denmark began with 57 index cases of psychopathic adoptees whose average age was 36 and examined the adoptive and biological relatives of these adoptees and of a matched control group of adoptees (Schulsinger, 1972). No difference in psychopathology (including psychopathy) was found between the adoptive relatives of the psychopathic adoptees and the adoptive relatives of the control adoptees, suggesting that shared family environment is not an important factor in the etiology of psychopathy. Some evidence was found for genetic influence in that psychopathology was somewhat greater in the biological relatives (19% of 305 individuals) of the psychopathic adoptees than in the biological relatives (13% of 285 individuals) of the control adoptees. Psychopathy showed a similar trend: 3.9% of the biological relatives of the psychopathic adoptees were diagnosed as psychopathic, whereas the rate was 1.4% for the control adoptees.

In addition to suggesting some genetic influence for antisocial personality, family and adoption studies have yielded two other important pieces of information. First, it appears that Briquet's syndrome, known as somatization disorder in DSM-III, occurs more frequently in females in families with antisocial male relatives (Guze, Cloninger, Martin, & Clayton, 1986). A Swedish adoption study of somatization disorders shows a genetic link between such disorders in females and criminal behavior in males (Bohman, Cloninger, von Knorring, & Sigvardsson, 1984). None of these studies has explored the genetic concomitants of antisocial personality in childhood, an area that would seem ripe for investigation; for example, it would be interesting to assess whether young girls with antisocial male relatives report more somatic complaints. Second, family and adoption studies suggest that the link between antisocial personality and alcoholism is *not* genetic—there is no excess of alcoholics among the adopted-away children of antisocial biological parents (Bohman, Cloninger, Sigvardsson, & von Knorring, 1982).

A study of considerable relevance to aggressiveness is a small adoption study of aggressive conduct disorder in children as related to antisocial personality in parents (Jary & Stewart, 1985). Aggressive conduct disorder was diagnosed when four of five sources (intake interviewer, mothers, fathers, teachers, and referring source) indicated aggressiveness and noncompliance that persisted for more than a year. The study included 37 adoptees diagnosed as aggressive conduct disorder and 42 nonadopted children also similarly diagnosed whose average age was 11 years. Eleven (30%) of the biological fathers and 11 of the biological mothers of the adoptees were diagnosed as antisocial, whereas none of the adoptive parents were similarly diagnosed. Nonadoptive fathers of the nonadopted children with aggressive disorder also showed a high rate of antisocial personality (33%), although none of the nonadoptive mothers were diagnosed as

having antisocial personality. These results suggest significant genetic links between aggressive disorder in childhood and adult antisocial personality, and they also imply that environmental transmission between parent and child may not be necessary for the development of aggressive conduct disorder. In addition, the study also shows much greater rates of alcoholism in the nonadoptive fathers of the children with aggressive disorder (50%) than in the adoptive fathers (8%) of the adopted children with aggressive disorder; however, this relationship was much less apparent for the biological fathers of the adopted-away children, 13% of whom were diagnosed as alcoholic.

Delinquency and Criminality

Although delinquency and criminality are often considered as the adjudicated versions of conduct disorder and antisocial personality disorder, respectively, little is known about the interrelationships among these disorders or their relationship to aggressiveness. One review of six twin studies of delinquency found 87% concordance with 87 pairs of identical twins and 72% concordance for 61 pairs of fraternal twins (Gottesman, Carey, & Hanson, 1983). Although it is often concluded that delinquency shows no genetic influence, this pattern of twin resemblance in fact suggests significant genetic influence and substantial shared environmental influence. Genetic influence is suggested because the identical twin concordance is significantly greater than the fraternal twin concordance, although the relatively small difference in concordance between the two types of twins suggests that genetic influence is not substantial. The influence of shared environment is assessed as the extent to which identical twin resemblance is not explained by heredity; shared environment is substantial because identical twin resemblance is remarkably high but hereditary influence is modest. Although finding genetic influence is not unusual, finding substantial shared environmental influence—environmental influences that make children in the same family similar—is exceptional. Behavioral genetic studies have shown that environmental influence on nearly all other measures of personality and psychopathology are primarily of the nonshared variety that make children in the same family different from one another (Plomin & Daniels, 1987; Rowe & Plomin, 1981), as discussed later.

A recent study of a quantitative measure of self-reported delinquent behavior yielded correlations of .71 for 178 pairs of identical twins and .47 for 97 pairs of fraternal twins, again suggesting significant genetic influence and significant shared environmental influence (Rowe, 1983a). Additional analyses suggested that the strong shared environmental influence reflects the direct influence of one twin on the other (especially being partners in crime). It is noteworthy that this effect occurred equally for identical and fraternal twins, thus leaving the equal environments assumption of the twin method intact. Furthermore, this study and subsequent research (Rowe, 1986a) found similar parameter estimates for boys and girls. In an analysis of the correlates of delinquent behavior, Rowe (1986b) found that the major genetic correlates of delinquent behavior are deceitfulness, anger, and impulsivity, rather than other traits, such as attitudes toward school.

It is reasonable to hypothesize that twins are more likely to show substantial shared environmental influence than nontwin siblings because twins are exactly the same age and are thus more likely to affect each other's delinquency and are more likely to have the same peers. In two studies of nontwin siblings' self-reported delinquency, however, sibling correlations were .48 and .51, respectively, for high school students (Rowe, 1986b; Rowe, Rodgers, Meseck-Bushey, & St. John, 1989); it is noteworthy that correlations for opposite-sex siblings were similar (.41 and .48, respectively) to the same-sex siblings. The fact that these sibling correlations are similar to the fraternal twin correlation suggests that the finding of substantial shared environmental influence on delinquent behavior is not limited to twins. However, Rowe *et al.* also report that the sibling correlation for a sample of college students is substantially lower than for high school students—for 250 pairs of college-age siblings, the sibling correlation for retrospective reports of delinquency during adolescence was only .19. This leaves open the possibility that siblings are less similar in self-reported delinquency than are twins, although it is also possible that high school and college samples truly differ in terms of shared environmental influence relevant to delinquency.

The genetic relationship between juvenile delinquency and adult criminality is not known; however, an overview of genetic research on criminality is included because it may be relevant to studies of aggressiveness and because it yields results different from delinquency. More detailed reviews may be found elsewhere (e.g., Mednick, Moffitt, & Stack, 1987; Wilson & Herrnstein, 1985). In a summary of eight earlier studies involving 175 identical

and 214 fraternal twin pairs, the average concordances were .69 and .33, respectively, for the two types of twins (Gottesman *et al.*, 1983). The best twin study involved all male twins born on the Danish Islands from 1881 to 1910 (Christiansen, 1977). Significant and substantial genetic influence is suggested both for serious crimes against persons and for crimes against property: identical and twin concordances are 42% versus 21%, respectively, for crimes against persons and 40% and 16% for crimes against property. Analysis of twin cross-concordances suggests that the two types of crimes are due to different sets of genetic influences (Cloninger & Gottesman, 1987). If this were the case, it would suggest the hypothesis that aggressiveness might contribute to crimes against people instead of crimes against property. However, the two types of crimes appear to be highly correlated in the population; more to the point, parent–offspring adoption studies do not agree with the hypothesis that the etiologies of crimes against property and crimes against people differ, as discussed later.

Adoption studies are consistent with the hypothesis of significant genetic influence on adult criminality. For example, one of the best studies again comes from Denmark, and is based on 14,427 adoptees and their biological and adoptive parents (Mednick, Gabrielli, & Hutchings, 1984). For 2,492 adopted sons who had neither adoptive nor biological criminal parents, 14% had at least one criminal conviction. For 204 adopted sons whose adoptive (but not biological) parents are criminals, 15% had at least one conviction. If biological (but not adoptive) parents are criminal, 20% (of 1,226) adopted sons have criminal records; if both biological and adoptive parents are criminal, 25% (of 143) adopted sons are criminals. In addition, the Danish adoption study obtained data for full siblings raised apart (20% concordance), half siblings raised apart (13% concordance), and pairs of unrelated children reared together in the same adoptive families (9% concordance). Although these data suggest somewhat less genetic influence than do the twin data, they are nonetheless consistent with a genetic hypothesis. They also suggest the possibility of genotype–environment interaction in that having a criminal adoptive parent has an effect primarily for those adoptees with a genetic propensity toward criminal behavior, a finding supported in other adoption studies (Cadoret, Cain, & Crowe, 1983). Additional analyses indicated no genetic distinction between violent crimes and crimes against property; that is, adoptee crime was predicted to the same extent regardless of the type of crime committed by the biological parent.

A small adoption study in the United States (Crowe, 1975) and a large adoption study in Sweden (Bohman *et al.*, 1982) have also found significant genetic influence for criminality. The latter study adds two important pieces of information. First, alcoholism interacts with the genetic disposition toward crime in the sense that the crimes of adoptees tend to be more violent when their biological parents are both criminal and alcoholic. However, the earlier hypothesis that criminality and alcoholism are alternative manifestations of the same underlying disposition is not supported: Criminality and alcoholism are independent genetically—as are antisocial personality and alcoholism—as seen in adoption studies of alcoholics that show no elevated risk of criminality in first-degree relatives (Baker, 1986; Cloninger & Reich, 1983). Second, an earlier analysis of juvenile delinquency in adopted-away offspring of parents with criminal records indicate that the incidence of juvenile delinquency in these children was no greater than for a control group of adoptees, suggesting that adolescent delinquency is genetically distinct from adult criminality (Bohman, 1972). However, we are not aware of other attempts to test this hypothesis; the fact that most adult criminals were delinquents leads us to question the hypothesis:

1. Some genetic influence is found for adolescent delinquency; genetic influence on adult criminality is greater.
2. Some shared environmental influence is found for criminality; shared environmental influence on delinquency is substantial.
3. Delinquency appears to be related genetically to personality traits including anger and impulsiveness.
4. Etiologies of delinquency and criminality are similar for boys and girls.
5. Adult crimes against persons and crimes against property both show genetic influence.

Unresolved developmental questions include the extent to which juvenile delinquency is related genetically to adult criminality—although the single available study suggest that they are not related—and other genetic correlates of adult criminality in childhood, especially the role of aggressive behavior in childhood.

Behavioral Genetic Research on Aggressive Behavior

As indicated earlier, we believe that research in developmental psychopathology will profit from a focus on behaviors that are more narrowly defined than global diagnostic categories, such as antisocial personality or conduct disorders, or complex phenotypes, such as delinquency and criminality. Rutter and Garmezy (1983) have argued that aggressiveness may constitute a dimension of behavior that can be disentangled from delinquency, emotional disturbances, inattentiveness, and other personality characteristics. Of course, it must be acknowledged that aggression itself is complex—two of the major distinctions that have been considered are verbal versus physical aggression and instrumental versus angry aggression (e.g., Buss, 1961). Context is also important: for example, aggression toward family members may differ from aggressive behavior toward peers or adults outside the family as seen in the lack of agreement between parent and teacher ratings of aggressiveness. Aggressiveness has not been immune to the controversy in personality research concerning cross-situational generalizability (e.g., Campbell, Bibel, & Muncer, 1985; Rushton & Erdle, 1987). Suffice it to say that when behavior is sufficiently aggregated across time and situations, cross-situational consistency can be demonstrated, peer- and self-ratings correlate, and substantial longitudinal stability is found (Huesmann, Eron, Lefkowitz, & Walder, 1984; Olweus, 1979; Rushton, Brainerd, & Pressley, 1983).

Regardless of the debate about the role of aggressiveness in diagnostic categories of conduct disorders, no one would deny the importance of aggression itself. We chose to emphasize aggressiveness for three reasons. The major reason is that, despite the definitional difficulties just mentioned, aggressive behavior is more clearly circumscribed than such diagnostic categories as conduct disorder. Second, although reviews are available of behavioral genetic studies of antisocial personality (Cloninger & Gottesman, 1987; Vandenberg, Singer, & Pauls, 1986) and criminal behavior and delinquency (Mednick *et al.*, 1987), to our knowledge no review has been published specifically on behavioral genetic studies of aggressive behavior. The third reason is that aggressiveness may prove to be especially interesting from a behavioral genetic perspective, for reasons described later.

Although behavioral genetic analyses have considered diverse human behaviors, there are few studies related to aggressiveness. Although it is tempting, in a review such as this, to stretch a construct—for example, in this case, to include studies of anger, dominance, and assertiveness—we chose to exclude studies that did not measure the expression of aggressive behavior.

Twin studies of aggressive behavior are summarized in Table 1. These results are unusual in the area of personality. Self-report personality questionnaires so typically yield correlations of about .50 for identical twins and about .30 for fraternal twins that one of the major questions now is whether there is any evidence for differential heritability of personality traits (e.g., Loehlin, 1982). For comparison purposes, shyness is another personality trait with implications for psychopathology; without exception, 18 twin studies of shyness yield evidence for substantial genetic influence in infancy, childhood, adolescence, and adulthood (Plomin & Daniels, 1986).

In contrast, for five twin studies involving 1,170 identical and 850 fraternal twin pairs, self-report aggression data yield average weighted correlations of .32 for identical twins and .14 for fraternal twins, suggesting lower heritability than for other personality traits. Moreover, two of the large studies (Bruun, Markkanen, & Partanen, 1966; Loehlin & Nichols, 1976) yielded very similar correlations, .25 for identical twins and .17 for fraternal twins, suggesting heritabilities of about 15% rather than the usual finding of 40% heritability.

In one of the large studies of adults that found substantial differences between identical and fraternal twin correlations (Rushton, Fulker, Neale, Nias, & Eysenck, 1986), the aggression measure appears to assess anger or hostility: Sample aggression items include "Some people think I have a violent temper" and "I try not to give people a hard time." Concerning the other study of adults (Tellegen, Lykken, Bouchard, Wilcox, Segal, & Rich, 1988), the Differential Personality Questionnaire (DPQ) manual indicates that its aggression scale assesses individuals who "hurt others for own advantage; is physically aggressive; is vindictive; likes to frighten and discomfit others; likes violent scenes" (Tellegen, 1982, p. 7). This 20-item scale shows the lowest internal consistency of the 11 primary scales of the DPQ and includes items as diverse as "I enjoy violent moves," "When I have to stand in line I never try to get ahead of others" (also reversed), "I get a kick out of really frightening

Table 1. Twin Studies of Aggressiveness

Reference	Age	*N*/Pairs	Measure	Results[a]
Ghodsian-Carpey & Baker (1987)	4–7 years	21 MZ 17 DZ	Mothers' Observations Checklist	r_{MZ} = .65 r_{DZ} = .35
			Aggressive scale of Child Behavior Checklist	r_{MZ} = .78 r_{DZ} = .31
Owen & Sines (1970)	6–14 years	18 MZ 24 DZ	Projective measure of aggression from Missouri Children's Picture Series	r_{MZ} = .09 r_{DZ} = .24
Scarr (1966)	6–10 years	24 MZ 28 DZ	Need for Aggression scale from Gough's Adjective Check List	r_{MZ} = .35 r_{DZ} = −.08
O'Connor, Foch, Sherry, & Plomin (1980)	7.6 years	52 MZ 32 DZ	Bullying scale from revised Parent Symptom Rating	r_{MZ} = .72 r_{DZ} = .42
Plomin, Foch, & Rowe (1981)	7.6 years	54 MZ 33 DZ	Videotape observations of modeled aggression:	
			Number of hits	r_{MZ} = .42 r_{DZ} = .42
			Intensity of hits	r_{MZ} = .39 r_{DZ} = .47
Vandenberg (1967)	High school	50 MZ 38 DZ	Aggression scale of Stern's Activities Index	$r_{MZ} = r_{DZ}$[b]
Canter (1973)	Adolescents and adults	39 MZ 44 DZ	Acting Out Hostility Scale of Foulds Hostility Scale	r_{MZ} = .14 r_{DZ} = .30
Rushton, Fulker, Neale, Nias, & Eysenck (1986)	Adults	296 MZ 179 DZ	Aggressiveness scale from the Interpersonal Behavior Survey	r_{MZ} = .40 r_{DZ} = .04
Bruun, Markkanen, & Partanen (1966)	Adults	157 MZ 189 DZ	Aggression scale	r_{MZ} = .25 r_{DZ} = .16
Loehlin & Nichols (1976) reported by Plomin, Foch, & Rowe (1981)	High school	504 MZ 328 DZ	Aggression scale	r_{MZ} = .25 r_{DZ} = .17
Tellegen, Lykken, Bouchard, Wilcox, Segal, & Rich (1988)	Adults	217 MZ 114 DZ 44 MZA 27 DZA	Aggression scale from the Multidimensional Personality Questionnaire	r_{MZ} = .43 r_{DZ} = .14 r_{MZA} = .46 r_{DZA} = .06

[a]MZ refers to identical (monozygotic) and DZ to fraternal (dizygotic) twins reared together; MZA and DZA refer to identical and fraternal twins reared apart; *r* indicates a twin intraclass correlation.
[b]Correlations not computed; analysis of differences within MZ and DZ shows no significant difference.

someone," and "I sometimes tease people rather mercilessly." Although the DPQ scale of aggression appears to be one of the best available questionnaire measures of aggression, it is clearly a complex dimension with a strong flavor of vindictiveness. In both of these studies, the fraternal twin correlations are much lower than expected on the basis of an additive genetic model (.04 and .14), correlations which in themselves suggest low heritability, although these low fraternal twin correlations result in a large difference between identical and fraternal twin correlations. Even so, in both studies, the aggression scale yielded one of the lowest heritabilities for the various personality scales included in the studies.

Tellegen *et al.* (1988) also reported results for 44 pairs of identical twins who were reared apart. The twin correlation for the DPQ aggression scale is .46, a correlation as great as the correlation for identical twins who were reared together. This cor-

relation suggests a heritability that is twice as great as the other twin studies. Although the sample is small and the correlation may be inflated by selective placement, it is interesting to consider the possibility that twins reared together interact in terms of their aggressiveness in a manner that lowers their resemblance. However, the small sample of fraternal twins reared apart is consistent with this hypothesis.

Thus, no clear conclusions emerge from twin studies of self-reported aggressiveness in adolescents and adults, except that aggressiveness, unlike most personality traits, shows no consistent pattern of genetic influence. However, it should be noted that no two studies used the same measure of aggression; as discussed later, more attention needs to be paid to the measurement of aggression and its components. Studies of aggression in childhood are even more diverse, both in terms of measures and results. The measures include a projective test (Owen & Sines, 1970), parental ratings on an adjective checklist (Scarr, 1966), maternal observations (Ghodsian-Carpey & Baker, 1989), parental ratings on a behavioral problems questionnaire (O'Connor, Foch, Sherry, & Plomin, 1980), and an observational study of modeled aggression (Plomin, Foch, & Rowe, 1981). The first study found a fraternal twin correlation that was greater than the identical twin correlation; the second study yielded an identical twin correlation greater than the fraternal twin correlation but the fraternal twin correlation was negative, which makes no genetic sense; the third study produced evidence for substantial heritability; and the fourth study showed no heritability.

Behavioral genetic research in the area of personality relies heavily on paper-and-pencil questionnaires, typically self-report and parental ratings. Because of the possibility that attributions and other cognitive complexities might affect responses on questionnaires, an observational study is especially valuable. For this reason, the sole observational study of aggressive behavior in childhood will be discussed in greater detail. In a study of 216 twin children whose average age was 7.6 years, each child was videotaped through a one-way mirror, playing with a 5-foot, inflated clownlike figure, which is weighted at the bottom so that it rights itself after being knocked down (Plomin *et al.*, 1981). The experimenter hit the clown five times (using a jablike punch in the nose), saying "Take that, and that, and that, and that, and that" and then said to the child, "Now you play with it." Number and intensity of hits were recorded from the videotapes, measures which yielded rater reliabilities of .94 and .74, respectively, and 2-month, test-retest reliabilities of .44 and .75. The so-called Bobo clown—developed originally by Bandura, Ross, and Ross (1961) for their studies of modeling of aggression—is the most widely used laboratory measure of aggression in children. The measure predicts aggressive behavior outside the laboratory. For example, aggressive behavior toward the Bobo clown correlates .76 with peer ratings of aggression and .57 with teacher ratings of aggression (Johnston, DeLuca, Murtaugh, & Diener, 1977). In the twin study, testing was alternated so that one twin received cognitive tests in another room while the co-twin was tested for aggression; thus, each twin was tested individually.

The results of this twin study are striking in that they are so different from behavioral genetic results for other dimensions of personality. Although the twin correlations in this study were similar to other studies in suggesting substantial hereditary influence on height and weight, the twin correlations for number and intensity of hits suggest *no* genetic influence. This is one of the only examples in the behavioral genetics literature showing zero heritability for personality measures known to be reliable.

The zero heritability estimate is not the only surprise. As mentioned earlier, one of the most important findings in human behavioral genetic research is that shared environmental influences are of little importance for personality and psychopathology (e.g., Plomin & Daniels, 1987); earlier, we saw one of the rare exceptions, juvenile delinquency, although it has been suggested that shared environmental influence is artifactual in this case. However, aggressive behavior in the observational study of aggressive behavior in childhood suggests substantial influence of shared environment: The twin correlations for aggressive behavior toward the Bobo clown are substantial for both types of twins, indicating that most of the reliable variance for these measures is due to shared environmental factors.

Although these results may be sample specific, age specific, or specific to the Bobo clown measure, it would be an important set of findings if individual differences in aggressive behavior are not influenced by hereditary difference among children and are determined solely by experiential differences shared by two children in the same family. Heritable traits are by no means immutable (Plomin, 1987b); however, traits that show no heritable variation are particularly promising in terms of prevention and intervention. The search

for specific environmental factors responsible for the environmentally induced variation would be aided by knowing that these factors are shared by children in the same family. One of the most well-documented facts about aggression is that it runs in families (Parke & Slaby, 1983); results such as these would suggest that aggression runs in families for environmental rather than genetic reasons.

Other Behavioral Genetic Issues concerning Aggressive Behavior

The bottom line of this review is the usual one that more research is needed; in this case, however, more research is needed before we can even answer the most basic question of behavioral genetics: To what extent are individual differences in children's aggressive behavior due to genetic and experiential differences among them? However, compared to results for other personality traits, twin results for aggressiveness show a much less consistent pattern of genetic influence; moreover, the only observational study shows no genetic influence and substantial shared environmental influence (Plomin *et al.*, 1981). For these reasons, aggressiveness may prove to be a particularly interesting trait.

A basic limitation of this research involves measurement, and behavioral genetic research can be no better than the measures it uses. Because aggressive behavior is likely to be heterogeneous and influenced by the context of assessment, we need multivariate measures that differentiate types and levels of aggressive behavior and multimethod approaches that consider and compare interviews and questionnaires for self-report, parental ratings, teacher ratings, and peer ratings. Additional observational studies are especially needed in light of the striking results found in the only behavioral genetic study of aggressive behavior. A second methodological limitation is the reliance on the twin design. Other behavioral genetic designs, such as family studies and especially adoption studies, are needed because of the possibility that twin results may not generalize to the rest of the population because of the unique relationship of twins.

One contribution to these issues consists of ongoing analyses of data from the Colorado Adoption Project, a longitudinal, prospective adoption study of 245 adoptive families and 245 nonadoptive families in which biological and adoptive parents of adopted children and parents of nonadoptive children have been studied and the children and their younger adoptive and nonadoptive siblings are studied at 1, 2, 3, 4, 7, 9, 10, and 11 years of age (Plomin & DeFries, 1985; Plomin, DeFries, & Fulker, 1988). At the visit to the homes of the adopted and nonadopted children at 3 years of age, videotaped observations of modeled aggression are obtained as in the twin study described earlier (Plomin *et al.*, 1981). In addition, when the children are 7, 8, 9, 10, and 11 years, parents and teachers complete the Child Behavior Checklist (Achenbach & Edelbrock, 1983), a widely used measure of developmental psychopathology, which includes items assessing aggressive behavior.

Asking the extent to which genetic and environmental factors are important in the origins of individual differences in aggressive behavior is only the first step in behavioral genetic research. As indicated earlier, if genetic influence is found, we can proceed to ask whether any major effects of single genes can be detected; if so, the science fiction-like tools of the new genetics can be applied to map the gene to a particular chromosome, isolate the gene, clone it, sequence it, and determine the gene's product. However, it is our bet that behavioral problems of childhood are highly polygenic and will continue to require quantitative genetic approaches.

Two directions for quantitative genetic research are multivariate analyses and longitudinal analyses. Multivariate analysis of the covariance among traits as compared to the traditional analyses of the variance of each trait considered one at a time is one of the major developments in behavioral genetics during the past decade (e.g., DeFries & Fulker, 1986). A multivariate approach is important because it is extremely unlikely that completely different genes affect different components of behavioral problems in childhood. Twin and adoption methods that are used to analyze the variance of single traits can be readily extended to analyses of covariance among traits to assess the extent to which genetic effects on one trait can also affect other traits (e.g., Plomin & DeFries, 1979). Two multivariate approaches were mentioned in this chapter: analyses of cross-twin sums and differences indicating that delinquent behavior is correlated genetically with personality variables, such as deceitfulness, anger, and impulsivity (Rowe, 1986b), and the analysis of cross-twin concordances for crimes of violence and property crimes which suggested that these two categories of crime might be independent genetically (Cloninger & Gottesman, 1987).

The same approach to the analysis of covariance can be applied to longitudinal data to

assess age-to-age change and continuity during development (Plomin, 1986b). For example, although juvenile delinquency shows some genetic variance and adult criminality shows substantial genetic influence, this does not imply that the genes that affect delinquency also affect criminality. The power of assessing age-to-age genetic continuity and change during development justifies the effort and expense of conducting longitudinal studies. In addition, parent–offspring designs can be used to screen childhood behaviors for their ability to predict problem behavior in adulthood as an "instant" longitudinal study from childhood to adulthood (Plomin, 1986a). An adoption analysis mentioned earlier (Bohman, 1972) is implicitly relevant to this approach in that it found that criminality of biological parents is not genetically related to juvenile delinquency in their adopted-away offspring. In the same vein, prospective high-risk studies of aggressiveness are possible to explore the developmental unfolding of aggressive behavior before social interactions are impaired—such work would be much easier than in the dozens of high-risk studies of schizophrenia because aggressive behavior is much more common and appears much earlier in development.

Although these additional genetic tacks will be important, we predict that behavioral genetic research will make its major advances in developmental psychopathology in terms of nurture rather than nature; not only is psychopathology polygenic, it is also "polyenvironmental." For this reason, quantitative genetic research that recognizes both polygenic and polyenvironmental sources of variance are likely to advance our understanding of nature, nurture, and the nature–nurture interface. Two examples were mentioned in this chapter. First, behavioral genetic data provide the best available evidence for the importance of the environment, an obvious point that must not be overlooked in the current zeitgeist in psychopathology, which is beginning to lean dangerously toward biological determinism. Second, behavioral genetic research converges on the conclusion that, for personality and psychopathology, nearly all of the environmental variance that affects these domains is of the nonshared variety, making two children in the same family no more similar than are pairs of children picked at random from the population (Plomin & Daniels, 1987; Rowe & Plomin, 1981). An interesting facet of research on delinquency—and possibly on aggression—is that it represents an exception to this rule, showing substantial shared environmental influence.

Another environmental direction for behavioral genetic research is the exploration of genetic influence on ostensibly environmental measures: environmental measures can be treated as phenotypes and can be submitted to quantitative genetic analysis (see the review by Plomin, 1986a). For example, in two twin studies, Rowe (1981, 1983b) found that adolescents' perceptions of their parents' acceptance are influenced by genetic differences among the adolescents, whereas their perceptions of parental control are not affected by genetic factors. A recent study employing the powerful design of identical and fraternal twins reared apart and matched samples of twins reared together found substantial genetic influence on perceptions of one's childhood family environment, viewed retrospectively some 50 years later; again, genetic influence was found for warmth/acceptance but not for control (Plomin, McClearn, Pedersen, Nesselroade, & Bergeman, 1988). It is possible, for example, that parents' self-reports of their physical punitiveness or children's perceptions of their parents' punitiveness are mediated by genetic factors.

As intriguing as it is to find genetic influence on ostensibly environmental measures, the real importance of this issue is the possibility that genetic influences on environmental measures are translated into genetic effects on the relationship between environmental measures and the development of aggressive behavior. Many environmental factors have been implicated in the development of aggression; the most well-documented factor is intrafamilial aggression, between spouses, parents and children, and siblings (Parke & Slaby, 1983). This finding is usually interpreted in terms of social learning. From a behavioral genetics perspective, however, the possibility looms large that such familial resemblance may be hereditary in origin: It is possible that relationships between parental punitiveness, inconsistency, and coercion and children's aggressiveness may be mediated by heredity if, as seems reasonable, aggressive parents are more punitive, inconsistent, and coercive. In infancy, it has been shown that associations between family environment measures and infant development are often substantially mediated genetically, as shown in comparisons between environment–development associations in nonadoptive and adoptive families (Plomin, Loehlin, & DeFries, 1985). In adoptive families, such associations cannot be mediated by heredity, whereas in nonadoptive families, heredity can play a role. Thus, if heredity mediates associations between measures of the family environment and aggressiveness of children,

the associations will be stronger in nonadoptive families than in adoptive families. No research of this type has yet been reported for the relationship between measures of the family environment and aggression, although we are currently analyzing data from the Colorado Adoption Project in this manner, comparing environment–development relationships in nonadoptive and adoptive families using parent and teacher ratings of aggressiveness on the Child Behavior Checklist.

Finally, the concepts of genotype–environment interaction and correlation (Plomin, DeFries, & Loehlin, 1977) can guide new ways of thinking about the nature–nurture interface in developmental psychopathology. Genotype–environment interaction and correlation need to be distinguished from the mistaken view called *interactionism*, that is, that the separate effects of genotype and environment cannot be analyzed because they interact. It is certainly true that for *an individual* there can be no behavior without both environment and genes; however, as discussed earlier, behavioral genetics does not address the causes of behavior of a single individual but rather the causes of differences among individuals in a population. If interactionism were to be believed, it would imply that main effects cannot be found because everything interacts with everything else; if this view were taken seriously, it would imply that one cannot study environmental influences because they are inexorably intertwined with genetic influences.

Genotype–environment interaction refers to the differential effect of environmental factors on individuals of different genotypes; for example, permissive parenting techniques might be less effective for aggressive children, as Patterson's (1982) work suggests. Earlier in this chapter, a possible example of genotype–environment interaction was mentioned: having a criminal adoptive parent has an effect primarily for those adoptees with a genetic propensity toward criminal behavior (Mednick *et al.*, 1984). Adoption studies that include measures of the family environment are particularly valuable for studying genotype–environment interaction.

Genotype–environment correlation refers to the differential exposure of individuals to environments; for example, children with a genetic tilt toward aggressiveness tend to seek out situations that reinforce their aggressiveness. Three types of genotype–environment correlation—passive, reactive, and active—have been proposed and methods for assessing them have been discussed (Plomin *et al.*, 1977). *Passive* refers to a child passively receiving from genetically related family members environments correlated with the child's genetic propensities; *reactive* implies that people other than family members can react to gene-based differences among children; and *active* denotes the likelihood that children will seek out and create environments correlated with their genetic propensities. A general theory of development fashioned around these types of genotype–environment correlation essentially posits a shift from the passive to the reactive and active varieties during childhood (Scarr & McCartney, 1983). Although genotype–environment correlation appears to account for a substantial amount of variance in IQ scores (Loehlin & DeFries, 1987), its effect on personality is not known.

In summary, behavioral genetics offers powerful tools for understanding the etiology of developmental psychopathology. The goal of this concluding section was to indicate that behavioral genetic methods can accomplish much more than estimation of the relative contributions of genetic and environmental influences. If the promise of this approach is to be realized, it will require the use of these tools by developmental psychopathologists who are not behavioral geneticists. There are not many behavioral geneticists and very few of them study developmental psychopathology; furthermore, only a handful of universities teach behavioral genetics at the graduate level so that no cadre of young researchers is waiting to fill the void. For this reason, it is our hope that some of this research agenda can be accomplished "on the side" by developmental psychopathologists who use behavioral genetic designs in studies aimed at whatever issues the researchers planned to study. For example, one can often add siblings or parents to a study of developmental psychopathology; twins are surprisingly accessible to researchers; and the current high divorce rate results in large numbers of stepparents who rear children who are not genetically related to them and in half-siblings who share only one parent in common (Plomin, 1986c). By adding a behavioral genetic dimension to their research designs, developmental psychopathologists can do what they were going to do anyway and then collect the bonus of exploring their topic from the perspective of behavioral genetics.

ACKNOWLEDGMENTS

Preparation of this chapter was supported in part by grants from the National Science Foundation (BNS-86-43938 and 88-06598), from the Na-

tional Institute of Child Health and Human Development (HD-10333 and HD-18426), and from the John D. and Catherine T. MacArthur Foundation.

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CHAPTER 11

Learning of Aggression

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Aggression—behavior aimed at harming another person—has environmental and biological determinants. Environmental factors include the degree to which the environment provides aggressive models, reinforces aggression, and frustrates and victimizes the child. Biological factors include the child's temperament, hormones, and physique. Interplays between heredity and environment are also influential. For example, children born with irritating, hard-to-handle temperaments are especially at risk for eliciting the rejecting, punitive parental reactions that are conducive to aggressive development. This chapter focuses on environmental bases of aggression. Special attention is paid to how environmental factors interact with the child's cognitions and behaviors to influence the development of aggression.

The chapter begins with a synopsis of the dominant learning-theory position on aggressive development. Next we describe the social cognitive deficits found in aggressive children. We assume in this section that aggressive children develop habitual ways of processing social information (such as a tendency to attribute hostile intentions to others) that consistently lead them to respond aggressively in certain situations. The rest of the chapter is devoted to how these social cognitive deficits and biases are acquired. We stress the roles of the family, the peer group, and the mass media.

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Social Learning Theory

According to Bandura's (1986) social learning theory, it is how the child's transactions with the social environment become represented in the child's mind that determines the child's behavior and development. In this theory, much aggression is acquired through observing the aggressive behavior of others, by encoding these aggressive action sequences into memory, and by using these action sequences to generate more abstract rules for conduct (e.g., the only way to get what you want is physical force). By watching the behavior of playground bullies, television villains, and even their own parents acting as disciplinarians, children learn how to engage in a wide variety of destructive and harmful acts.

Although most children know how to perform a large variety of aggressive acts, they are likely to behave aggressively only when they expect a positive outcome for it. Children learn the typical consequences for aggressive acts partly through observation and partly through personal experience. For example, if children see others rewarded for

aggression, they may infer that they, too, would be rewarded for similar action. Direct personal experience also contributes. Children who have had repeated success in eliminating frustration with aggression, for example, should develop more confident beliefs that aggression will pay off.

A major feature of social learning theory is that aggression comes under the control of internal self-regulatory processes. Once children discern which behaviors society deems appropriate and inappropriate for them to perform, they tend to adopt these standards of conduct as internalized guides for behavior. They feel good about themselves and reward themselves when their behavior matches these standards, but they feel guilty and dissatisfied when they perform behaviors that they know are wrong. If children see that certain forms of aggression in certain situations and toward certain targets are inappropriate (e.g., physical aggression toward females, or aggression against someone whose frustrating behavior is not intentional), they may avoid acting aggressively under these circumstances for fear of self-censure. Not all children learn the same rules. Members of delinquent gangs may internalize the norm that violence and destruction are worthy of self-praise rather than self-blame. The kinds of models and social support systems that surround children influence the shape their rules for self-regulation take.

Social learning theorists shun the concept of aggression as a reflexive reaction to frustration, but this is not to say that learning theorists deny the importance of frustrating and other aversive events in the etiology of aggression. On the contrary, learning theorists stress that aggression is often a response children learn to make when deprived, threatened, or hurting, especially if the children find the aggression to be successful at improving their plight. But learning theorists regard aggression only as one possible learned consequence of aversive experience, not as a reflexively triggered outcome.

Social learning theorists stress that reactions to frustration and other arousing events are cognitively mediated, or depend on the individual's cognitive evaluation of the arousing event. For example, children who develop a style of blaming others for their mishaps and believe aggression is an effective way to eradicate frustrations should be more likely to develop aggressive habits than children who do not blame others for their misery or can think of effective, nonaggressive solutions to conflicts. In the next section, we examine the social cognitive processes and biases of the aggressive child.

Social Cognition and Aggression

Aggression can result from breakdowns or deficits in the way children process social information. Suppose a boy is hit in the back by a ball thrown by a playmate. Even if the event is purely accidental, the boy may react aggressively if he impulsively assumes the ball-thrower was deliberately trying to hurt him, if hitting is the only response that comes to the boy's mind, or if the boy fails to consider the negative consequences (e.g., pain to the playmate, punishment from a teacher) that might result from acting aggressively. Highly aggressive children possess deficits and biases in the way they react to certain social situations, and these deficits and biases lead them to respond aggressively.

A Social Cognitive Model of Competent Responding

Dodge (1986) has proposed a social information-processing model of competent social responding. His model describes a sequence of five cognitive steps that are thought to be necessary for a child to react appropriately and competently (i.e., nonaggressively) to a social situation or stimulus. Each step is a necessary, but insufficient, part of appropriate responding.

The first step is the *encoding* of social cues, which involves searching for and focusing attention on relevant social information (e.g., attending to a playmate's face in order to gather information about the peer's intentions). Second is the *interpretation* step, which involves giving meaning to the cues attended (e.g., correctly reading a provocateur's actions as accidental, hostile, or well-intended). Third is *response search,* or the generating of various possible behavioral responses to the situation (e.g., thinking of prosocial or assertive as well as aggressive solutions to a problem situation). The fourth step is *response decision,* which involves choosing a response after evaluating the potential consequences of each possible response (e.g., selecting a response that promises positive outcomes for both the self and other). The final step is *enactment,* which is behavioral performance of the chosen response (e.g., adhering to a mental script for the desired response).

Social Cognitive Deficits of Aggressive Children

Many aggressive children possess deficiencies at one or more of the steps in Dodge's model. Here we review some of the deficiencies.

Encoding Deficits

Aggressive children search for fewer cues than nonaggressive children before reaching a decision about another child's intentions. In one study, aggressive and nonaggressive children were told a story about a provoked character and then were asked what they thought the character intended to do. Before responding, the children could listen to as much or as little tape-recorded evidence about the character as they wanted. Aggressive children listened to less testimony than nonaggressive children before making a decision about the character's intentions (Dodge & Newman, 1981). Slaby and Guerra (1988) also found that aggressive adolescents sought less information than nonaggressive peers before deciding that someone was "out to get them."

Interpretation Deficits

Aggression is more common in response to frustration and other aversive stimuli if the provoked person attributes his or her plight to intentionally provocative behavior on the part of someone else. Aggressive children possess a bias toward perceiving hostile intent in others that leads them to behave aggressively in situations where others would not. Dodge (1980; Dodge & Frame, 1982) examined the reactions of aggressive and nonaggressive boys to a frustrating situation (a peer destroyed a puzzle the boys were completing, thereby preventing the boys from earning a reward). For some children the destruction was rigged to look clearly accidental, for other children it looked clearly deliberate, and for still other children it was ambiguously motivated (it was left unclear whether the peer had intended to destroy the puzzle or not). When the frustration was clearly accidental, both aggressive and nonaggressive boys correctly perceived the frustration as accidental and responded with little aggression. When the frustration was clearly deliberate, both groups of boys again reacted similarly, correctly perceiving the provocation as deliberate and reacting with a fair degree of aggression. The interesting finding came when the provocation had been ambiguously motivated. Here, nonaggressive boys reacted nonaggressively (giving the peer the benefit of the doubt), whereas aggressive boys interpreted the ambiguously motivated frustration as if it had been hostilely intended and responded aggressively. This tendency to perceive hostile motivation even when it may not exist is called "hostile attributional bias." Slaby and Guerra (1988) confirmed the existence of hostile attributional bias among aggressive adolescents.

Dodge (1986; in press) has suggested several possible explanations for the hostile attributional bias of aggressive boys. Perhaps most important is the fact that many aggressive children have histories of actual victimization and aversive treatment at the hands of their parents and peers. When confronted with a new aversive stimulus that is ambiguously motivated, the aggressive child may simply ascribe the stimulus a meaning that is consistent with a preexisting expectation of hostility.

Response Search Deficits

When children are asked to generate as many solutions as they can to hypothetical story conflicts, aggressive children generate fewer solutions than nonaggressive children. Furthermore, aggressive children are more likely to generate aggressive solutions and are less likely to think of prosocial and cooperative solutions than nonaggressive children (Deluty, 1981; Dodge, 1986; Richard & Dodge, 1982; Slaby & Guerra, 1988; Spivack & Shure, 1974).

Response Decision Deficits

Not only do aggressive children generate more aggressive solutions to conflicts but they also evaluate aggressive reactions more favorably than their nonaggressive peers. Part of the reason aggressive children evaluate aggressive responses more favorably is that they expect more payoffs for behaving aggressively than do less aggressive children. As Bandura's (1986) social learning theory predicts, aggressive children are especially confident that aggression will yield tangible rewards (such as a disputed toy or other resource), and they believe that aggression will be successful in terminating aversive behavior directed toward them by noxious others (Perry, Perry, & Rasmussen, 1986). Moreover, compared to nonaggressive peers, aggressive children attach more value, or importance, to certain of

the rewards that aggression offers (such as gaining control over the victim), and they attach less importance to the negative outcomes of aggression (such as causing suffering in the victim or being rejected by the peer group) that serve to inhibit aggression in most children (Boldizar, Perry, & Perry, 1989). The thought of causing suffering in their victim, then, does not discourage many aggressive children from aggression (Perry & Perry, 1974); in fact, highly aggressive children sometimes reward themselves for injuring others (Perry & Bussey, 1977).

Aggressive children's desire for control over people and resources leads them to attack certain children more than others. Aggressive children do not distribute their attacks evenly across all available peer targets, but instead selectively direct their attacks toward a minority of peers who consistently serve as victims of peer abuse (Olweus, 1978; Perry, Kusel, & Perry, 1988). These highly victimized children are the ones who reward their attackers by relinquishing resources, by showing signs of submission and pain, and by failing to fight back (Olweus, 1978; Patterson, Littman, & Bricker, 1967; Perry *et al.*, 1988).

Enactment Deficits

Even if a child generates a nonaggressive, prosocial solution to a conflict and realizes that this solution will benefit everyone concerned, the child cannot perform the behavior if he or she lacks the requisite skills. Aggressive children do lack many of the skills (both academic and social) necessary to gain what they want through peaceful, cooperative, and prosocial means. Many aggressive children have poor homework habits, have trouble making friends, and experience difficulty in routine social situations with peers (e.g., in conversing, asking appropriate questions, suggesting joint play activities, nondisruptively joining a group of playing children) (Asher, Renshaw, & Geraci, 1980; Dishion, Loeber, Stouthamer-Loeber, & Patterson, 1984).

Some of the social cognitive deficits of aggressive children can be ameliorated by therapy designed to curb impulsive responding. For example, in Camp's "Think Aloud" program, children role-play nonaggressive responses to staged annoyances and accusations while verbalizing constructive thought processes (e.g., they verbalize various response options and their consequences) (Camp, Blom, Herbert, & Van Doornick, 1977).

Family Influences on Aggression

Many aggressive children come from homes in which the parents (and siblings) make the child feel insecure and rejected, bombard the child with aversive stimuli (commands, taunts, threats), teach the child that force is the only way to escape such aversive treatment, and fail to teach the child that aggression is unacceptable. Here, we see how aggression may evolve out of the child's early feelings of insecurity and how family interaction patterns may promote or discourage aggressive development.

Attachment Security and Aggression

According to the theories of Ainsworth (1979) and Sroufe (1983), infants and toddlers differ in the degree of security they feel in their attachment relationships with their primary caregivers. Securely attached children are those who have developed conceptions of their caregivers (and other people) as trustworthy, reliable, and available for assistance, if needed. Secure feelings are presumably acquired through interaction with a caregiver who responds reliably and readily to the infant's cries and bids for attention, is sensitive to the infant's needs and signals, and is consistently affectionate. Infants reveal a secure attachment by using their attachment figure as a secure base from which to explore novel surroundings, by showing moderate distress upon separation from their caregiver, and by allowing their caregiver to comfort them upon reunion.

In contrast, insecurely or anxiously attached children lack confidence in the accessibility of a caring person in times of distress, presumably because they have experienced histories of inconsistent and insensitive caregiving. Two patterns of anxious attachment have been identified—resistant and avoidant. Resistant infants cling to their caregivers rather than use them as a base for exploration, become extremely upset by separation, and do not let their caregivers calm them upon reunion. Avoidant infants appear undisturbed by separation, and they avoid their caregivers before and after separations. Compared to secure infants, anxious ones (of both types) are less cooperative and compliant with their mothers as well as with other adults, whine more, and are more prone to temper tantrums (Bates, Maslin, & Frankel, 1985; Londerville & Main, 1981; Matas, Arend, & Sroufe, 1978). These findings are important because children with a history of early oppositional and noncompliant behav-

ior at home stand an increased risk of developing serious behavior problems later on (Patterson, 1986).

In several studies, securely and insecurely attached infants have been observed a few years later in the preschool setting. Children with histories of secure attachment reveal better social skills, are more popular among their peers, display more positive and less negative affect, are more empathic, and are more cooperative and compliant with adults (Arend, Gove, & Sroufe, 1979; Sroufe, 1983; Waters, Wippman, & Sroufe, 1979). Insecurely attached children are more prone toward social isolation, irritability, restlessness, aggression toward peers and teachers, disobedience, depression, and deficiencies in imaginary play (Erickson, Sroufe, & Egelund, 1985; Lewis, Feiring, McGuffog, & Jaskir, 1984; Sroufe, 1983). There also exist differences between the two types of anxiously attached children in the way they express aggression. For anxious resistant children, aggression is more often physical, directly confrontational, disruptive, impulsive, and accompanied by tantrums. For anxious avoidant children, aggression more often takes the form of subtle noncompliance and attempts to stay out of others' control (Sroufe, 1983).

In summary, the research suggests that infants who enjoy trusting, mutually satisfying relations with their caregivers are more likely to develop prosocial, cooperative styles of interpersonal influence that obviate the need for aggression. However, the negative outcomes associated with insecure attachments are not inevitable. In fact, the ill effects of insecure attachments appear to be most likely for infants who come from severely disadvantaged homes. Among brighter, middle-class children, the potential ill effects of an insecure attachment can be offset by the more stimulating physical and social environment provided by well-educated and affluent parents (Bates *et al.*, 1985; Erickson *et al.*, 1985).

Family Management Practices and Aggression

Comparisons of the child-rearing environments of aggressive and nonaggressive children reveal several differences in parental attitudes and practices. Here, we review the major parenting dimensions for which reliable differences exist.

Monitoring

Compared to parents of nonaggressive youngsters, parents of aggressive and delinquent children are less aware of their children's whereabouts, activities, and social contacts (Loeber & Dishion, 1983; McCord, 1979; Patterson & Stouthamer-Loeber, 1984). Parental indifference and lack of supervision mean that aggressive children receive less parental pressure for appropriate behavior.

Parental Aggression

Many aggressive children come from homes in which at least one parent is exceptionally violent—toward the spouse, toward the child, toward the siblings, or toward others outside the home (McCord, 1979). George and Main (1979) reported that toddlers who were physically abused by their parents at home were assaultive toward their peers and hostile toward their caregivers in day care. McCord (1979) found that adult men convicted of crimes often had parents who were given to losses of temper, smashing things, deviance (alcoholism or criminality), and abusive discipline. Other studies confirm that parents who erratically inflict harsh physical punishment are more likely to have aggressive children (Eron & Huesmann, 1984; Olweus, 1980). These correlations are usually interpreted as support for the hypothesis that children learn aggression by imitating their parents. However, aggressive, defiant children also elicit harsher discipline from adults (Bell, 1968; Trickett & Kuczynski, 1986).

Permissiveness

Parents of aggressive children frequently decline to set limits on their children's behavior and are ineffective at stopping their children's deviant behavior (Baumrind, 1973; Loeber & Dishion, 1983; Olweus, 1980; Patterson & Stouthamer-Loeber, 1984). In discipline confrontations, the parents of problem children have been shown to threaten, nag, scold, and bluster, but they seldom follow through on their requests (Patterson, 1986). Paradoxically, sometimes it is the parents' very permissiveness that makes them resort to assaultive behavior when they do discipline their children. Parents who are ineffective at nipping deviant behavior in the bud sometimes find themselves becoming more and more exasperated as their child's deviant behavior escalates. They may suddenly explode with anger and assault the child.

Inconsistency

Both interparent and intraparent inconsistency in disciplinary practices have been implicated in aggressive development (Glueck & Glueck, 1950; McCord, McCord, & Howard, 1961). Interparent inconsistency involves disagreement and inconsistency between the parents in disciplinary practices. Intraparent inconsistency involves the failure of a given parent to discipline a child consistently for an infraction each time it occurs. When deviant child behavior is punished on some occasions but rewarded or ignored on other occasions, it can prove very resistant to extinction or punishment (Katz, 1971; Martin, 1975; Sawin & Parke, 1979).

Rejection

Highly aggressive individuals often have a history of parental rejection (McCord, 1979; Olweus, 1980). The hostility and negativism of rejecting parents may contribute to aggressive development, but parental rejection may also be a reaction to having an antisocial child (Patterson, 1986).

Absence of Techniques for Dealing with Family Problems

The families of aggressive children often lack techniques for dealing with crises or problems (Patterson & Stouthamer-Loeber, 1984). Verbal communication is low in these families, and discussion geared to solve problems is rare. Parents of aggressive children often avoid the use of reasoning during disciplinary encounters (Becker, 1964; Martin, 1975; Perry & Bussey, 1984).

Patterson's Theory of Aggressive Development

Patterson (1980, 1986) has proposed a powerful and comprehensive theory of aggressive development in the family. In his view, several of the deficiencies in parenting that we have already seen to be associated with aggressive development—lack of monitoring, hostility, permissiveness, inconsistency—can combine to create a home environment that is conducive to the learning of vicious cycles of aggression among family members. Patterson proposes that many aggressive acts represent high-powered attempts by children to "turn off" irritants that are supplied by parents and siblings who are threatening, aversive, and lacking in social skills. Many of these irritants are seemingly trivial and mild, such as failing to give a child attention, ignoring him, commanding him to stop doing something, expressing disapproval of him, or teasing or laughing at him, but when presented in a home atmosphere that is barren of positive stimuli, these irritants may be especially aversive and provocative. Aggression can result from children's attempts to turn off the noxious stimuli emanating from other people; it is used to demand attention, to stop the teasing, to stop being frustrated, or, in some cases, to interrupt the boredom. The aggressive behavior of these children is called "coercive" because, in effect, the children are saying, "You had better stop frustrating and irritating me and give me what I want or else I will escalate my attack until you do!"

Aggressive habits are most likely to develop, Patterson contends, if the family members who provoke the child at least intermittently reinforce the child's counterattacks by terminating their aversive behavior and yielding to the child's demands. This occasional reinforcement makes it likely that the child will attempt aggression the next time an irritant is encountered. Furthermore, because aggressive children are confident that aggression will ultimately prove successful in stopping the flow of aversive stimuli from other people, they will often continue, or even escalate, their aggressive behavior in the face of preliminary obstacles, such as intensified aversive stimuli or threats of punishment (Bandura, 1986). Because aversive consequences can trigger further aggression, parents and therapists must be careful about how they choose to modify aggressive behavior. Punishment, the threat of punishment, and other aversive controls can increase rather than deter aggression (Perry & Bussey, 1984). Patterson's treatment program for families with a hyperaggressive child involves a dual strategy: (1) teaching family members the social skills necessary to interact in positive, mutually reinforcing ways rather than aversively; (2) teaching the deviant child that coercion will not be tolerated, by refusing to comply with coercive behavior and subjecting the child to a time-out procedure when aggression occurs.

Integrity of the Family Unit

Disruption of the family unit (through divorce, separation, or death) as well as internal and external stresses on the marriage and family can foster conditions that are conducive to aggressive development (e.g., Hetherington, 1979). Consistent application of firm but reasoned discipline may be

more difficult to achieve in broken or severely stressed homes. Parke and Slaby (1983) pointed out that there is an almost infinite variety of routes by which family disharmony can stimulate aggressive behavior. In one family, the father may behave in a demanding and abusive fashion toward the mother, the child may imitate the father by also behaving aggressively toward the mother, and the mother, in turn, may reinforce the child's coercive demands. In another family, a mother may verbally discipline a child who fails to comply; the child's defiance of the mother may anger the father who then intervenes with harsh discipline. In still another family, the mother may respond negatively to her child in part because of an unsatisfactory relationship with her husband. Although both parents clearly can contribute to development of aggression in the child, mothers' childrearing practices (negativism, power assertion, yielding to the child's coercion) correlate more strongly with children's aggression than do fathers' childrearing practices (Olweus, 1984; Patterson, 1980). Mothers, it seems, are more likely than fathers to become involved in the vicious cycles of escalating aggression in which the parent incites and then inadvertently rewards the child's coercion. However, the degree of support the husband supplies the mother in her role as parent influences the likelihood of the mother and child developing vicious cycles of aggression.

Peer Influences on Aggression

Peer interaction is valuable in teaching children to control their aggressive impulses and to limit their expression of aggression to "appropriate" times and places. However, peers can also be instrumental in teaching each other new aggressive habits. We review these processes and discuss how the reputations children acquire among their peers for behaving aggressively or nonaggressively can have important consequences for the children's development.

Peer Play and the Regulation of Aggression

Research with monkeys indicates that the opportunity to play with one's peers may be necessary for the young of some species to learn how to express aggression in adaptive ways (Suomi & Harlow, 1978). Aggression in monkeys (threatening, biting, wrestling, and chasing) is probably biological in origin, but how the aggression becomes integrated into the developing monkey's behavioral system depends on the nature of the social ties the monkey forms. For monkeys who have formed close emotional ties to playmates, aggression is gradually integrated into patterns of nonserious social play. Aggressive contacts are very rough, incorporating vigorous wrestling, threats, and harmless biting, but the aggression is not destructive and rarely results in physical injury to the participants. It seems that the monkeys are learning to make sure that the aggression they direct toward friendly peers is playful and nonserious; serious aggression becomes reserved for strangers. In contrast, the aggression of monkeys who are deprived of peer attachments is inappropriate. When these monkeys are joined with a larger community of monkeys, they are hyperaggressive. They foolishly attack larger, dominant monkeys, and they aggress inappropriately against younger, defenseless animals—something almost never seen in normally reared animals. They tend to be social isolates and avoid social play. Research is needed with human children to establish whether engaging in rough-and-tumble play with close friends helps children learn to distinguish between serious and nonserious aggression and to avoid maladaptive forms of aggression.

Peer interaction also allows children to learn their place in the dominance hierarchy, and this learning has the net effect of reducing aggression among children (Blurton-Jones, 1972). Dominance hierarchies in children's play groups usually gel after an initial period in which children resolve disputes through overt aggression. For example, for the first few weeks of school, children test each other's limits, with many disputes coming to blows and resulting in a winner and a loser. As children learn who can beat whom, hierarchies result from this process. Once group members know everyone's place in the hierarchy, within-group aggression is minimized. When disputes arise, children can exchange signals of dominance and submission that remind each other of their places in the hierarchy. The dominant member's threatening gestures and facial expression remind the less dominant member of who is likely to win if they come to blows; the less dominant member acknowledges probable defeat with a submissive signal, thus bringing the episode to an end before a physical fight ensues (Strayer & Strayer, 1976; Zivin, 1977).

Intensive peer contacts do not always guarantee reduced aggression, however. Very young children (infants and toddlers), for example, may not be

able to decipher and remember a dominance hierarchy, and for these children extensive experience with peer conflicts may promote rather than control aggression. Sometimes day-care infants are more aggressive than home-reared infants (Belsky & Steinberg, 1978; Haskins, 1985). In the next section, we review some of the ways children teach each other aggression.

Peer Training of Aggression

In discussing the impact of the family on aggressive development, we noted that children who are subjected to high densities of aversive stimuli, who use physical coercion to eliminate the unpleasant behavior of others, and who are least intermittently reinforced for these aggressive counterattacks often develop habits of aggression. Similar processes operate in the school setting to make children aggressive. Patterson *et al.* (1967) observed children interacting in the nursery school setting and recorded each act of peer-directed aggression. The observers also noted how the victim of each attack behaved. Children who were passive and nonaggressive at the start of the school year were found to be frequently initiating aggressive interactions by spring, provided that during the year these children (1) had been frequently victimized by more aggressive children, (2) had eventually tried to defend themselves with aggressive counterattacks, and (3) had found these counterattacks to be successful.

Members of deviant subcultures, such as delinquent gangs, often reward each other for aggressive behavior and even make status in the group contingent upon adopting various injurious and destructive behaviors. In one study of institutionalized delinquent girls, the girls enthusiastically reinforced acts of aggression (Buehler, Patterson, & Furniss, 1966). The juvenile offender's association with delinquent peers is in fact a powerful predictor of continued antisocial behavior (Hanson, Henggeler, Haefele, & Rodick, 1984).

Peer Reputations and Aggression

We noted earlier that hyperaggressive children are paranoid in the sense that they possess a readiness to attribute hostile intentions to other people. This paranoia leads them to engage in inappropriate aggression. Dodge (in press) proposes that the hostile attributional bias of aggressive children may be reinforced by the rejection, hostility, and suspicion that aggressive children elicit from their peers. Once aggressive children sense their rejection, they may come to expect hostile intent even when it is not there. This may lead them to engage in further unwarranted aggression, causing their peers to become still more rejecting and hostile, and strengthening the aggressive children's paranoia in a vicious circle.

Evidence supports these speculations. Aggressive children do elicit dislike from their peers (Coie & Kupersmidt, 1983; Dodge, 1983). Not only do children dislike, distrust, and expect the worst of their aggressive peers (Dodge, 1980; Perry *et al.*, 1986), they also treat them with open hostility (Dodge & Frame, 1982). Children are particularly critical of peers who engage in unprovoked or unwarranted aggression (Lesser, 1959; Rule, Nesdale, & McAra, 1974). It seems likely, then, that rejection by the peer group would strengthen aggressive children's expectation that other people are treating them with hostile intent. Peer rejection may also force aggressive children to turn for companionship to children who accept and perhaps even encourage aggressive behavior (Patterson, 1986).

The foregoing analysis helps us understand how negative peer reputations can reinforce aggressive children's hostile attributional bias, but it does not explain how the bias comes about in the first place. Some children may develop expectations of hostility through interactions with negative family members. For other children, the bias may originate in the peer group. Dodge (in press) has suggested that some children take a first step toward acquiring a hostile attributional bias when they interpret an ambiguously motivated provocation as hostile and respond aggressively. If the children's peers have viewed the stimulus as benign, they may interpret the aggression as unwarranted, label the aggressing children as deviant, and reject them. The basis for the vicious cycle of attributional bias, aggression, and peer rejection is established.

Media Influences on Aggression

Effects of Viewing Television Violence

Children with strong preferences for viewing violent television programming are more aggressive than their peers (Eron, 1963), and this relation holds across diverse cultures (Eron, 1982). Experiments carried out in the laboratory and in the field confirm that watching violence can have a causal effect on children's aggression. For example, when preschoolers are exposed to a daily diet of

aggressive cartoons, they increase their spontaneous expressions of aggression—hitting, kicking, and throwing things—within the preschool setting (Friedrich & Stein, 1973; Steuer, Applefield, & Smith, 1971). Longitudinal research also shows that children who prefer violent television programs during elementary school engage in more violent and criminal activity as adults (Eron & Huesmann, 1984).

Bandura (1979) reviewed several effects of viewing violence. First, television violence teaches children aggressive styles of conduct as well as the general lesson that conflicts are settled through force. Second, witnessing violence alters restraints over aggression. Seeing others perform aggressively makes one feel that aggression is commonplace and acceptable and reduces anticipatory self-censure for behaving aggressively. Third, repeated viewing of violence desensitizes and habituates people to violence and suffering (Cline, Croft & Courrier, 1973; Thomas, Horton, Lippincott, & Drabman, 1977). A reduced empathic sensitivity decreases the chance that a person will come to the aid of distressed others or curtail aggression against suffering others (e.g., Drabman & Thomas, 1974). Fourth, viewing violence shapes people's images of reality. Heavy television users tend to view the world as a hostile place. This fact may also contribute to the hostile attributional bias of aggressive individuals.

Although television violence is one cause of aggression, it is also likely that aggressive children prefer to watch more violent television. As Eron (1982) commented,

> the process is very likely circular. As we have seen, aggressive children are unpopular, and because their relations with their peers tend to be unsatisfying, they spend more time watching television than their more popular peers. The violence that they see on television reassures them that their own behavior is appropriate while teaching them new coercive techniques that they then attempt to use in their interaction with others, which in turn makes them more unpopular and drives them back to television, and the circle continues. (p. 210)

Of course, a similar process can operate in the family. Partly because social interaction with family members is so aversive for aggressive children, they may spend long periods of time watching television. When they imitate the aggression, they elicit even more rejection and hostility from other family members, causing them to watch television even more.

Factors Affecting Imitation of Aggression

People are more likely to imitate media aggression if it is depicted as rewarded or as unpunished than if it is shown to be unsuccessful or punished (Bandura, 1965). Aggression that is justified (e.g., directed toward a villainous character) is imitated more than unjustified aggression (Berkowitz & Rawlings, 1963). Realistic aggression is more readily imitated than fictional violence (Meyer, 1972). However, people who view criticized, unjustified, or fictional violence are still more likely to respond aggressively than people who do not view aggression at all.

Characteristics of the viewer influence the likelihood of imitating media violence. Because young children sometimes fail to see a connection between aggression and the negative consequences that follow it, they imitate the aggression when older persons do not (Collins, 1973). Children with positive attitudes toward aggression, with aggressive fantasies or daydreams, or with behavioral tendencies toward aggression more readily imitate violence than children who lack these qualities (Eron, 1982; Hicks, 1971). Children who identify with aggressive television characters (who perceive themselves to be similar to the characters) or who believe that what they see on television is an accurate depiction of real life are also more likely to imitate television aggression (Eron, 1982; Huesmann, Eron, Klein, Brice, & Fischer, 1983; Huesmann, Lagerspetz, & Eron, 1984). Finally, people who are angry at the time they view aggression or who are encountering in their own lives stressors similar to those triggering aggression in the television characters are especially likely to respond aggressively (Geen & Stonner, 1973).

Mitigating the Imitation of Aggression

Adults can reduce the likelihood of children imitating aggression if they criticize televised aggression while children watch it (Grusec, 1973; Hicks, 1968). Huesmann *et al.* (1983) conducted an ambitious project in which children, over the course of several years, participated in periodic sessions that were designed to teach them three lessons: (1) that television is an unrealistic portrayal of the real world; (2) that aggressive behaviors are not as universal or as accepted in the real world as they are on television; and (3) that it is not good to behave like the aggressive characters on television. For example, children were asked to prepare a speech on why television is unlike real life and why it is bad to

emulate televised aggression; they were videotaped making their speeches and were told the tapes would be shown to other children. Compared to children in a control group who did not participate in such sessions, the experimental group of children actually declined in aggression directed toward classmates.

Conclusions

The evidence reviewed indicates that environmental conditions, cognitive factors, and the child's own behavior all play roles in the learning of aggression. Prominent among environmental factors is the degree to which the child is subjected to hostile environments and is reinforced for responding coercively. Important cognitive factors are a hostile attributional bias and the belief that aggression is an appropriate and effective way to gain what one wants. Children's own behavior also helps to create their learning environments. For example, by seeking out violent television programming, children acquire new aggressive responses.

It is fruitless to consider any one of these three classes of determinants as more basic or important than the other ones. Variables of each sort influence and are influenced by variables of the other two sorts, in the manner described by Bandura's (1986) theory of reciprocal determinism. In this chapter, we have seen numerous examples of how environmental, cognitive, and behavioral variables influence one another and how causal chains involving these three kinds of variables can create and perpetuate aggressive habits. For example, an infant with a difficult, hard-to-soothe style of interaction may elicit parental impatience and rejection; learning to expect hostility, the child may attribute hostile intentions to playmates and engage in acts of unwarranted aggression toward peers; the unjustified aggression may elicit rejection from the peer group, reinforcing the child's hostile attributional bias and driving the child to watch television or to consort with peers who condone aggression. Advances in understanding and preventing aggressive development will come not from debate over the "primary" causes of aggression but rather from charting the typical sequences of interplays among environmental, cognitive, and behavioral variables that are associated with aggressive development.

ACKNOWLEDGMENTS

The authors express their appreciation to Beth Halleck, Sarah Kusel, Ellen Milmed, and Jean Williard for their comments on a previous draft. Preparation of this chapter was assisted by National Science Foundation Grant BNS-8507000.

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CHAPTER 12

The Stability of Aggressive Behavior—Even unto the Third Generation

Leonard D. Eron and L. Rowell Huesmann

In young adults, delinquency and criminal behavior are problems of which dimensions in terms of cost to life and property are obviously staggering. Decades of research into the root causes of delinquency and criminality have revealed numerous variables that are correlated with the ontogeny of different aspects of criminal behavior. Yet there is little sign that, based on these findings, the responses of the criminal justice or of the psychological establishments have had much impact. It now seems clear that a major reason for such failures is that the aggressive style of responding, underlying most delinquent and criminal behavior, generally emerges very early in life but also appears to be a relatively stable, self-perpetuating behavior that is not readily amenable to change by the time it usually comes to the attention of society. Hence, it is imperative for researchers to examine the foundations of delinquent and criminal behavior in young children, and not just in adolescents or in young adults. Preventive programs based on such findings with young children would have a more reasonable chance for success than traditional correctional programs with older individuals. By the time a youngster is in middle childhood, characteristic ways of behaving aggressively or nonaggressively in interpersonal and other problem-solving situations have become so well practiced that they are highly resistant to change. Further, these early aggressive behaviors, which often may appear to some to reflect no more perhaps than "boisterousness and incivility" (Cook, Kendzierski, & Thomas, 1983), are predictive of future serious antisocial behavior of the type that brings the individual into contact with the law (Farrington, 1985; Huesmann, Eron, Lefkowitz, & Walder, 1984; Magnusson, Stattin, & Duner, 1983).

What are the root causes of this type of destructive behavior that persists very often despite severe negative sanctions? Serious antisocial aggression clearly seems to be a multiply determined behavior. A number of interrelated factors must converge for aggressive behavior to appear and become primary in an individual's repertoire of possible responses (Eron, 1982).

Genetic, physiological, and other constitutional factors undoubtedly contribute to antisocial aggression in many cases, but they cannot account for the extreme individual differences that one finds in aggressiveness among humans. It is the rare case of aggressive behavior that can be traced to central nervous system abnormalities. Hormonal varia-

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tions, particularly in testosterone, may also explain some differences, but more differences remain unexplained by physiological factors. The innate aggressive responses one finds in lower animals cannot usually be demonstrated in humans. Despite some ethologists' claims, there is little evidence to support the concept of an innate aggressive drive in human beings. Rather, the mounting evidence suggests that aggression is learned to a great extent. For most children, aggressiveness seems to be determined most by the extent to which their environment reinforces aggression, provides aggressive models, and frustrates or victimizes the child, thus instigating aggression. An extensive array of environmental, familial, and child characteristics have been shown to be weakly predictive of which child will be more aggressive; yet, none could be called a sufficient or necessary condition for the development of antisocial aggression. Severe antisocial, aggressive behavior seems to occur only when there is a convergence of a number of these factors during the child's development (Eron, 1982). However, this does not mean that aggressive behavior cannot be predicted or explained. Research that we and others have been doing would indicate that aggressive behavior is consistent over time and across situations despite the fact that a number of factors contribute to the behavior in varying degrees.

In this chapter, we present evidence from a 22-year longitudinal study that shows future antisocial aggression to be quite predictable from the aggressive behavior displayed by children in their everyday behavior in the school setting. We also refer to research indicating stability over 3 years among elementary school children in 5 different countries (Huesmann & Eron, 1986). Other studies also demonstrate stability of criminal behavior (e.g., Farrington, 1985; Glueck & Glueck, 1930; Magnusson *et al.*, 1983; Robins, 1966), but few have demonstrated such consistency among an unselected "normal" group of children (Olweus, 1979).

Stability of Aggression over 22 Years

In 1960, we completed a survey of all the third-grade school children in a semirural county of New York State. This included 875 youngsters whose modal age at the time was 8 years. We also interviewed 75% of their mothers and fathers. The focus of our procedures was the aggressive behavior of the subjects as it was manifested in school. Our purpose was to delineate the learning conditions for aggression. We wanted to know how children learn to be aggressive; therefore, we collected information about the presumed psychological and social antecedents and correlates of aggressive behavior. Most of the findings of this phase of the study have been published and indicate that many children do learn to be more or less aggressive from their interactions with the environment (Eron, Walder, & Lefkowitz, 1971).

Ten years later, in 1970, we reinterviewed 427 of the original subjects (modal age, 19). One of the most impressive findings was the stability of aggressive behavior over time and across situations using a variety of measurement operations (Lefkowitz, Eron, Walder, & Huesmann, 1977). Those children who were more aggressive in school in 1960 were more aggressive in a variety of situations in 1970.

In 1981, we again interviewed 295 of the original subjects individually and another 114 by a combined mail and telephone interview (modal age, 30). In addition, we obtained data about these subjects and 223 others of our original subjects from the New York State Division of Criminal Justice Services and the Division of Motor Vehicles. Thus, we had some follow-up data on over 600 of the original subjects. We also were successful in obtaining interviews with the spouses of 165 subjects, and 82 of the subjects' own children who, at the time, were approximately the same age as the subjects were when first seen. Thus, there are data from three generations of informants—the subjects, their parents, and their children.

In this chapter, we will be concerned primarily with how early indications of aggression at age 8 relate to later indicators of aggression at age 30. For our purposes, aggression was defined as "an act which injures or irritates another person." This definition is limited to hostile, interpersonal, extrapunitive aggressive behavior. It excludes behaviors many persons might call aggressive; for example, assertiveness, ambition, and striving. By this definition, the so-called aggressive salesman would only be assertive.

In 1960, child aggression was measured by a peer-nomination technique (Walder, Abelson, Eron, Banta, & Laulicht, 1961). In this procedure, every child in a class rates every other child on a series of 10 specific items of aggressive behavior (e.g., Who starts a fight over nothing? Who pushes or shoves other children? Who says mean things? Who takes other children's things without asking?). The reliability and validity of this measure have

been extensively documented. Over the course of 25 years, this measure has been used in at least 10 countries in over 50 different studies with consistent success (Eron, Walder, & Lefkowitz, 1971; Huesmann & Eron, 1986).

Indications of the subjects' aggression 22 years later at age 30 were derived from self-ratings by the subject, ratings of the subject by the spouse, and citations of offenses by the New York State Divisions of Criminal Justice and Motor Vehicles. Self-ratings included the sum of the Minnesota Multiphasic Personality Inventory (MMPI) scales 4, 9, and F, which previous research (Huesmann, Lefkowitz, & Eron, 1978) has indicated is a reliable and valid measure of overt aggression, as well as indications of past use of physical aggression by the subject. Ratings by the spouse of the subject's aggression included behavior directed toward him or her by the subject. These items came from the Straus Home Violence Questionnaire (1979). The Criminal Justice scores were the total number of convictions in New York State in the previous 10 years and a rating of the seriousness of the corresponding offenses. The latter system is used by the New York State Criminal Justice Division, and requires that each type of offense be assigned a specific seriousness score (Rossi, Bose, & Berk, 1974). The number of convictions for traffic offenses, including drunken driving, were obtained from the New York State Traffic Division. For those subjects who had children, there were also ratings of how severely the subject punished the child as well as self-ratings of aggression by the child. Reference will also be made to another punishment score reflecting how severely the subjects themselves were punished by their own parents. This measure was obtained from parent interviews during the first wave of the study in 1960 (Eron *et al.*, 1971).

Correlations between the early measure of aggression and later measures of aggression are shown in Table 1. It is apparent that over 22 years, there is moderately good predictability from early aggression to later aggression, especially in the case of males, and the relation holds up across method, informant, and situation as well as time. Because a disproportionate number of the original subjects who moved out of the state subsequent to the original testing were from high aggressive groups (Lefkowitz *et al.*, 1977), the range of aggression scores has been truncated, and the correlations are probably a minimal estimate of the relation between aggression at age 8 and later antisocial behavior of the type that brings individuals into contact with the law. Also, the 1960 aggression score of males who were not interviewed was significantly higher than the aggression score of those males who were interviewed, thus restricting further the range of scores (Huesmann *et al.*, 1984). A more representative demonstration of the relation can be obtained by dividing the subjects into low, medium, and high groups according to the original peer-nomination measure and calculating mean scores on each of the criterion variables separately for each of the three groups. These relations are depicted in Figures 1 through 5. When tested by analysis of variance, the differences among the means on each of the criterion variables are highly significant, especially in the case of males.

Perhaps the most impressive aspects of the longitudinal correlations and of the bar graphs are the relations between peer-nominated aggressive behavior at age 8 and adult criminality. The children who are nominated as more aggressive by their third-grade classmates commit, on the average, more serious crimes as adults. In Table 2, the exact conviction rate is shown for each group of males

Table 1. Correlations between Peer-Nominated Aggression at Age 8 and Various Measures of Aggression at Age 30

	Males		Females	
	N	*r*	*N*	*r*
MMPI scales F + 4 + 9	190	.30***	209	.16*
Punishment of child by subject	63	.24[a]	96	.24*
Spouse abuse	88	.27**	74	—
Criminal justice convictions	335	.24***	207	—
Seriousness of criminal acts	332	.21***	207	—
Moving traffic violations	322	.21***	201	—
Driving while intoxicated	322	.29***	201	—
Self-rating of physical aggression	193	.25***	209	—

[a] $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$.

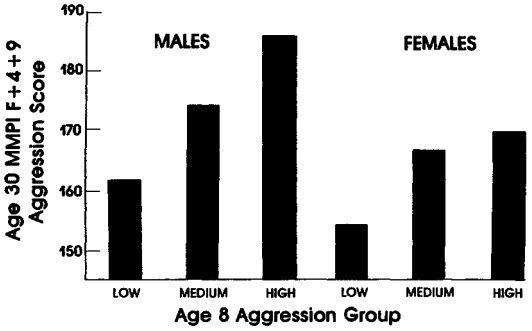

Figure 1. Relation of aggression at age 8 to aggression at age 30.

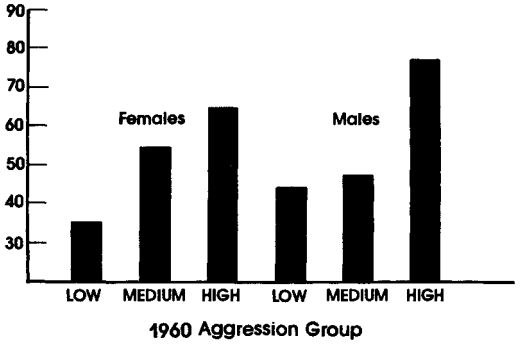

Figure 2. Mean punishment of child scores in 1981 according to subjects' aggression score in 1960.

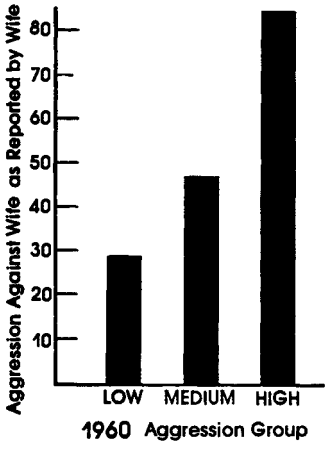

Figure 3. Relation of aggression at age 8 to wife abuse at age 30.

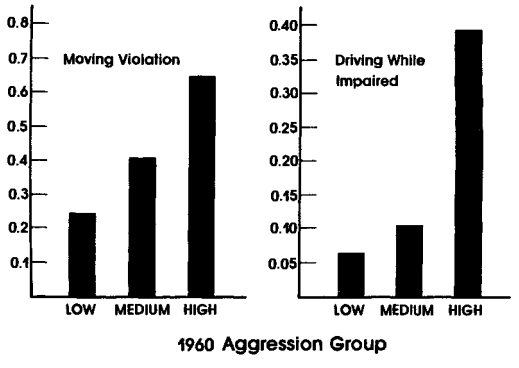

Figure 4. Mean number of traffic violations in New York State until 1981 according to male subjects' aggression score in 1960.

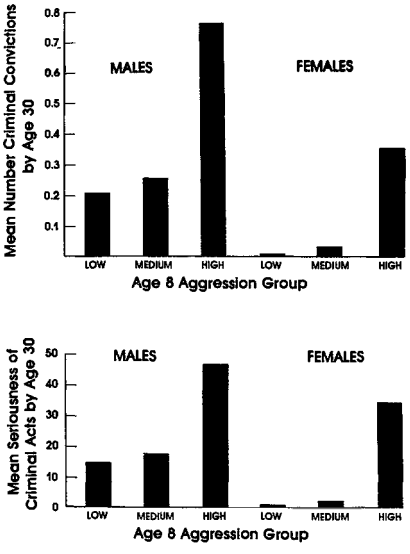

Figure 5. Relation of aggression at age 8 to criminal behavior at age 30.

and females. These data convincingly indicate that the peer nominations we obtained when these subjects were 8-years old are measuring more than transient "boisterousness and incivility" (Cook *et al.*, 1983). These peer nominations measure a characteristic aggressiveness in many children that evidences itself in severe antisocial behavior as a young adult.

There are more significant predictions from 1960 aggression to 1981 aggression for males than for females (7 vs. 2). It is interesting that the one aggression area at the later period, which is predicted quite successfully for females from early aggression, is punishment of the subject's child. Child punishment is probably the only arena in which a female can express aggression without fear of social censure or retaliation. In the other arenas, aggression toward spouse, criminal offenses, and moving traffic violations, there is such a low frequency for females that successful prediction from earlier indications of aggression is very unlikely.

Table 2. Proportion of Subjects Convicted for a Crime in New York State before Age 30 according to Gender and Aggression at Age 8

	Age 8 Peer-nominated aggression		
Gender	Low	Medium	High
Males	8	24	19
	— = 9.0%	— = 15%	— = 23%
	89	161	82
Females	0	2	3
	— = 0.0%	— = 1.8%	— = 6.3%
	49	110	48

Intergenerational Transmission of Aggression

Our data, which were collected over three generations, indicate further that aggression as a characteristic behavior seems to be transmitted from parent to child. It should be noted that genetic transmission is not necessarily implied here. Over and above whatever equipment and tendencies the child is born with, there are many ways in which parents can teach children aggression, primarily by the models of behavior they provide and the reinforcements they provide for aggressive behavior. The effect of these learning interactions across generations can be seen in the correlations among measures of parental, subject, and child aggression. Because the sample of subjects for whom all such data are available is limited by the number of subjects' children on whom there were data ($n = 82$), the data were not analyzed separately by gender. In 1960, there was a correlation of .25 between how aggressive these subjects were in school, as nominated by their peers, and how severely they were punished for aggression by their parents at home. When the subjects were 19 years of age, they were asked to imagine how they would respond to their

child's aggression if they had an 8-year-old child. The correlation was .24 between their peer-nominated aggression score in 1960 and their hypothetical response in 1970, and .31 between their hypothetical response in 1970 and their own parents' actual responses in 1960. In 1981, those subjects who now had children between 6 and 12 years were asked the same questions and the correlation between their earlier peer-nominated aggression and how severely they reported punishing their own child for aggression was similar (.31). Furthermore, the subjects' peer-nominated aggression score obtained in 1960 correlated even higher with their children's self-rated aggression in 1981 (.29) than did the subjects' self-rated aggression in 1981 ($r = .26$). This is especially interesting because the self-rating items used for the children were the same 10 items that comprised the peer nominations on which their parents had been rated by their peers 20 years earlier. Even more surprising, the child's current aggressiveness and the viewing of television violence correlate significantly with the subjects' preference for TV violence 22 years earlier when the subject was only 8 years old. In fact, the correlation of .41 between the child's self-rating of aggression and the subjects' viewing of violence 22 years earlier is one of the highest reported in the study (Huesmann, Eron, Dubow, & Seebauer, submitted). It might be maintained that this intergenerational stability could be explained by consistencies across generations in social class and intelligence. However, the correlations do not change substantially when the parents' social class and the child's IQ at age 8 are partialled out, indicating that the intergenerational stability is independent of these variables.

Although we should not be too quick to attribute causality to the circumstance that abusing parents were themselves abused as children (Belsky, 1980), the dramatic findings we have uncovered over three generations cannot be ignored. Certainly, there are many things going on—the correlations are only in the range of .20 to .35, and the univariate correlations cannot account for more than a small portion of the variance. However, many of the variables and processes can be specified,and together they can probably account for a major portion of this variance. With our recent advances in computer technology, this multiplicity of variables can be synthesized into complex, comprehensive process models that can then describe specific children at risk for developing the kind of behavior which we have been discussing. Thus, we are not limited to aggregate statistics that tell us very little about individual children. Finally, the whole process can be simulated. And, indeed, we find with Lisrel analysis that there are coefficients of approximately .26 over three generations. A model based on such analysis is seen in Figure 6.

A Cross-National Study

Earlier in this chapter we mentioned the possibility that the remarkable stability that has been demonstrated in aggressive behavior over the years might be an artifact due to social class and/or intelligence. This possibility was discounted when it was shown that controlling for these factors did not reduce the correlations appreciably. Another limiting factor might be geographical area. The 22-year study was conducted with subjects growing up and living in a semirural area of New York State. Therefore, starting in 1977, a 3-year longitudinal study was conducted in a heterogeneous urban suburb, Oak Park, Illinois, of a large metropolitan area, Chicago. The purpose of the study was to check the boundary conditions of the television violence/aggression relation obtained in the 22-year study (Huesmann & Eron, 1986). Over 700 children, half of whom were in the first grade and half in the third grade at the beginning of the study, were followed for 3 years. It was found that although aggression, as measured by the peer-nomination procedure, increased as children progressed from Grade 1 through Grade 5, there was substantial correlation between the subject's aggression scores at time 1 and at time 3. The identical study was then repeated in four other countries. As shown in Table 3, the correlation coefficients in the other countries are as large or higher than in the United States. Thus, this stability transcends cultural and national differences and is not peculiar to the semirural population of northeastern United States.

Implications of Stability of Aggression

In summary, then, the degree of stability that we have seen in aggressive behavior of individuals over 3 years as well as over a 22-year span, reaching back to a previous generation and ahead to a future one, is impressive. The stability holds up across situations, measures, and informants as well as time. The frightening implication of this intractable consistency is that aggression is not situation specific or determined solely by the contingencies. The individual carries around something inside that im-

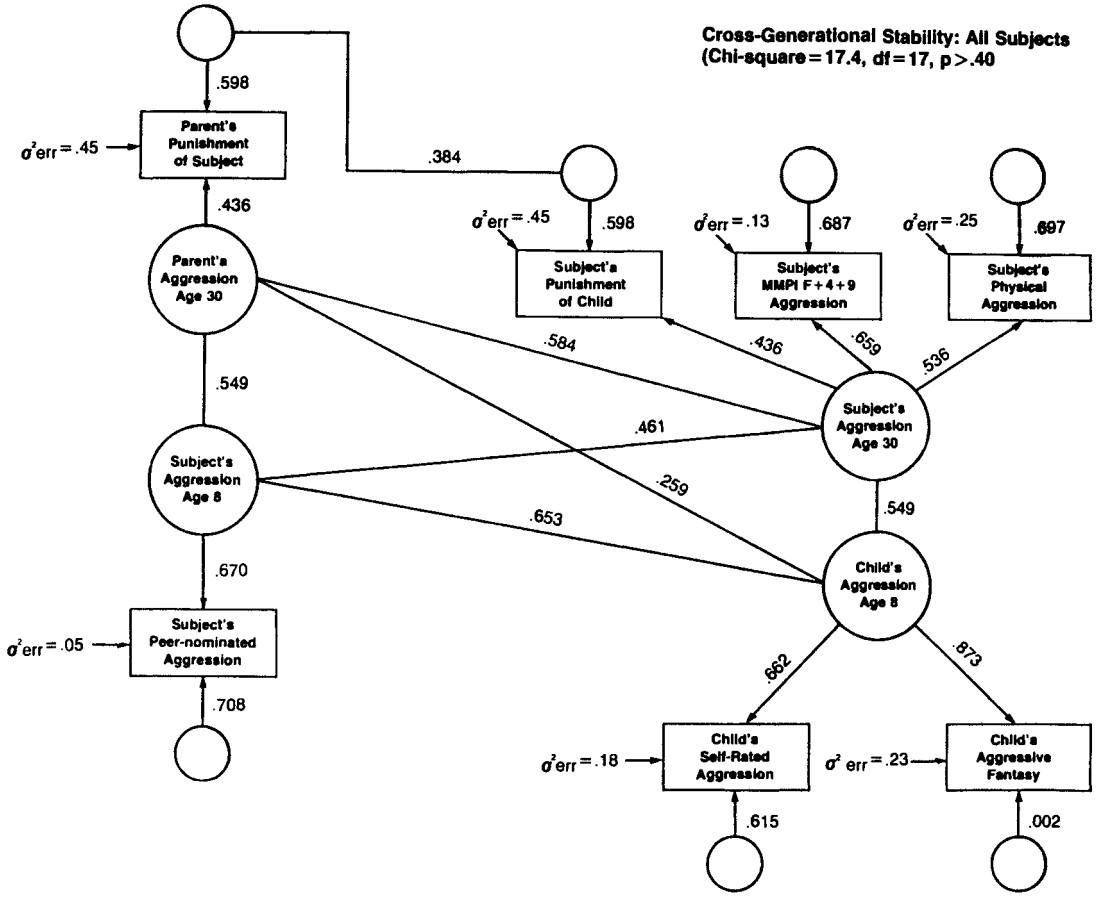

Figure 6. Process model of the development of aggression over three generations. Cross-generational stability: all subjects (χ^2 = 17.4, df = 17, p > .40).

pels him or her to act in a characteristically aggressive or nonaggressive way. Again, we do not maintain that this is genetic or constitutional, although there may well be some such basis for this type of behavior (Mednick, Gabrielli, & Hutchings, 1984). Much of this behavior is learned, and it is probably the memory traces in the brain that are activated when the individual is in a situation with some similarities to the one in which the aggressive sequence was originally encoded that probably account for much of the consistency.

Table 3. Stability of Aggression over Three Years

	Males		Females	
Country	*N*	*r*	*N*	*r*
United States	200	.53	221	.71
Finland	93	.77	85	.74
Australia	116	.54	107	.65
Poland	107	.62	96	.80
Israel kibbutz	32	.63	41	.73
Israel city	46	.75	39	.80

We hypothesize that social behavior is controlled to a great extent by cognitive scripts, schemes, and strategies that have been stored in memory, and that are rehearsed, stored, and retrieved in much the same way as are other strategies for intellectual behaviors. These strategies might be closely associated with specific cues in the encoding context, or might be abstractions unconnected to specific cues. By encoding, we mean the "formation of a representation of an external stimulus in the memory system" (Kintsch, 1977, p. 485). Under this view, an aggressive strategy must be encoded, retained in memory, and retrieved later on in

order to influence the child's behavior. A number of situational and interpersonal factors could influence each of these three processes. To encode an aggressive response, a child must attend to the behavior and must not reject the behavior as completely inappropriate. To maintain the encoded strategy in memory, the child must rehearse it. Through "elaborative" rehearsal, the child may develop abstractions of the aggressive strategies. Finally, to retrieve the strategy, the child must be able to access it in memory. According to the encoding specificity principle, the presence of cues that were also present at encoding time facilitates such retrieval.

Fortunately, children have as many or more opportunities to learn nonaggressive or prosocial strategies for solving interpersonal problems. Unfortunately, however, children who learn aggressive problem-solving strategies generally do not learn prosocial strategies. Indeed, it has been demonstrated that aggression and prosocial behavior are at opposite ends of a continuum (Eron & Huesmann, 1984). Further, prosocial behavior at age 8, as seen in a concern over interpersonal relations and a reluctance to harm others, predicted prosocial behavior 22 years later, as measured by occupational and educational status, as well as low aggression, social success, and good mental health. On the other hand, aggression at age 8 predicted social failure, psychopathology, aggression, and low educational and occupational success (Eron & Huesmann, 1984).

Several conclusions about the development of aggression are suggested from this perspective. First, we would expect each child to develop a characteristic style of aggressive or nonaggressive behavior that would become relatively stable across time and situations as the child matures. This does not mean that situational factors would be unimportant. The stability would be a stability of relative position in the population. Situational factors would be very important in cueing the retrieval of certain strategies and in continuing the learning process. However, once a schema of strategies for social behavior has been firmly established, it would probably be very resistant to change. Second, the information-processing styles that facilitated the acquisition of the aggressive or prosocial strategies originally are likely to persist. Thus, the more aggressive child becomes the more aggressive adult. Under this model, aggression can become self-perpetuating despite severe negative reinforcements that accrue to the child. The more the child behaves aggressively, the more the child is exposed to aggressive scenarios to be encoded. The more scenarios the child has encoded, the more scenarios are available to be rehearsed. The more the scenarios are rehearsed, the more likely are they to be retrieved when a social problem arises. The more likely aggressive strategies are to be retrieved, the less likely are other strategies to be retrieved. The child behaves aggressively, and the cycle continues.

Because aggression has these traitlike characteristics and because it is a behavior that develops early in life, it is also a behavior that is not easy to change. In order to change this characteristic way of behaving and solving problems, it will take all the knowledge, ingenuity, talent, and persistence we can muster. We have to know specifically which variables require change and concentrate on them. Until we know the causes of behavior deviance, we cannot institute appropriate change. And the cause is a process—there are many variables, each impinging on the other in reciprocal loops. If we are lucky, we can break into these loops by identifying specific processes and attempting to change them. But we must start early in the lives of developing youngsters. By the time a child is 8-years-old, characteristic ways of behaving aggressively or nonaggressively have already been set down. Aggression, as a problem-solving behavior, is learned very early in life, and it is learned very well; the payoff is tremendous. The inducement to change must be made equally attractive.

If aggression were mostly situationally determined, one would certainly not expect stability coefficients of the magnitude we have reported. At the same time, the stability that we have described is a stability of individual differences, that is, of relative position in a population, not a stability of absolute level. Characteristic forms and amounts of aggression change dramatically with age (Eron, Huesmann, Brice, Fischer, & Mermelstein, 1983) and situation. Some situations are much more likely to elicit aggressive behaviors than others. What remains reasonably constant is the aggressiveness of an individual relative to the population. Some situations are much more likely to elicit aggressive behaviors than are others. What remains reasonably constant is the aggressiveness of an individual relative to the population. The child who is at the top of the distribution for 8-year-olds is likely to be near the top of the distribution for 30-year-olds two decades later in many situations in which aggression is displayed.

It is apparent that many individuals are characterized by a propensity to respond in an aggressive manner to a variety of interpersonal situations. This

propensity or disposition becomes apparent *early on* in their development and continues to characterize their behavior as they grow into adulthood. The development of aggressive behavior, at least in this sample of subjects, is testimony more to constancy than to change in human development (Brim & Kagan, 1980). Where do the roots of this constancy lie? How does the aggressive disposition take hold and persist across varied environmental conditions?

Although this discussion is not directed at elaborating the causes of aggression, one cannot discuss the stability of aggression without some attention to this issue. The data reported here, showing predictability of aggression across three generations as well as from childhood to adulthood within a single life span, cannot discriminate between genetic, constitutional, and/or environmental explanations. Reports of our previous findings (e.g., Eron *et al.*, 1971; Lefklowitz *et al.*, 1977) have stressed the importance of learning conditions for aggression in a child's development. The current results are not inconsistent with such a view. Aggressive dispositions can be learned. Youngsters are often directly reinforced for acting aggressively by increased attention as well as by the material and psychological rewards that are often the consequence of such behavior. Examples of aggressive behavior are abundantly available in the media as well as at home, at school, and in the neighborhood. The learning of aggressive attitudes is facilitated when respected and glorified figures, for example, professional athletes, movie stars, and political figures, engage in various kinds of aggressive behavior for which they reap many rewards. It is easy for a youngster to justify aggressive solutions to problems by referring to norms for such behavior that are promulgated by the media and others. As stated previously (Eron *et al.*, 1983), aggression is usually learned early in life and may be learned very well, generalizing to many situations. Aggressive individuals actively seek out and create situations in which they can often be observed (Bowers, 1973; Olweus, 1978), and because of their past conditioning histories, they may be exquisitely sensitive to aggressive cues in the environment (Berkowitz, 1962). Thus, though the individual's environment and learning conditions may change radically, aggression can persist.

Quite probably, the impressive stability of aggressive behavior across time and generation is a product of the continuity of constitutional factors and the continuity of environmental factors. Certainly, constitutional characteristics, including genetic factors (Christiansen, 1977), hormonal factors (Kreuz & Rose; 1972 Rada, Kellner, & Winslow, 1976) and neurological trauma (Mark & Ervin, 1970) play some role in aggressive behavior. Just as certainly, a person's environment and learning history strongly influence his or her aggressive tendencies (Bandura, 1973; Berkowitz, 1962; Eron *et al.*, 1971). The relative importance of these factors is arguable. What is not arguable is that aggressive behavior, however engendered, once established remains remarkably stable across time, situations, and even generations within a family.

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CHAPTER 13

Morality and Conduct Disorders

Judith G. Smetana

Conduct disturbances, as indicated by temper tantrums, aggression, destructiveness, defiance, and inconsiderate or uncooperative behavior, constitute one of the most frequent causes of concern regarding children's behavior (Rutter & Garmezy, 1983), and there has been long-standing speculation that such behavior may be related to an inadequacy in such children's capacity to make moral judgments. Piaget (1932) initially suggested that developmental analysis of "difficult children," "youthful offenders," and "young ruffians" might be fruitful. Several decades later, Anthony (1956) hypothesized that children who repeatedly or seriously violate societal laws may suffer developmental delays in moral reasoning (Jurkovic, 1980). Since then, numerous studies have examined the developmental maturity of moral reasoning among behavioral disordered children.

This chapter considers the available evidence on the relationship between disturbances of conduct, particularly in juvenile delinquents, and moral judgment. I will begin with a review of prominent structural-developmental theories of moral development and their measurement. Then the empirical literature on relationships between conduct disorders and moral development will be examined, focusing first on comparisons between delinquents and nondelinquents and then on attempts to facilitate the developmental maturity of delinquents' moral judgments.

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Theories of Moral Development

Developmental research on children's moral judgments has its origins in Jean Piaget's early work (1932). Based on his interviews with children, Piaget developed a two-stage scheme of the development of moral reasoning. According to Piaget, the young child's reasoning, which he referred to as *heteronomous,* is characterized by unilateral respect for adult authority and the view that all rules are sacred and unchangeable because they stem from adults. Through experiences of cooperation with peers, children's reasoning gradually becomes transformed into a morality of justice and reciprocity, in which prescriptive morality is seen as obligatory, based on cooperation, and differentiated from the merely customary or conventional.

Piaget described 11 dimensions that he felt characterized the shift from heteronomous to autonomous moral reasoning. A great deal of subsequent research has been devoted to examining these dimensions. Most frequently, children are asked to choose between story pairs (for instance, a child who intentionally breaks a few cups versus a child who unintentionally breaks a greater number of cups); their choice is seen to reflect the structure of their underlying reasoning.

Kohlberg (1958, 1969, 1978b) expanded Piaget's two-level scheme into a much more com-

prehensive model of moral development. In his scheme, moral development progresses through three levels, encompassing six sequential stages of logically more adequate concepts of justice. These are summarized in Table 1. According to Kohlberg, morality for the young child is seen as external to the self and is oriented toward power, punishment, and physical consequences. At this level, children first make judgments on the basis of physical consequences, and then on the basis of the concrete exchange of favors and gratification of needs. Kohlberg (1958, 1978a) has hypothesized that this early level characterizes the moral orientation of juvenile delinquents. The moral orientation at the second level, including Stages 3 and 4, is toward maintaining or conforming to the rules of society, first, in terms of significant others and interpersonal relationships, and later, in terms of relationships to the wider social order. At the highest level, referred to as principled morality, moral judgments are au-

Table 1. Kohlberg's Stages of Moral Judgment

Level 1

At this level, the child is responsive to cultural rules and labels of good and bad, but interprets them in terms of physical consequences (e.g., reward, punishment, or exchange of favors) or in terms of the physical power of those in authority.

Stage 1

The punishment and obedience orientation. Right action is determined by the physical consequences of actions. Avoidance of punishment and unquestioning deference to power are valued in their own right.

Stage 2

The instrumental-relativist orientation. Right action consists of that which instrumentally satisfies one's own needs. Human relations are viewed in terms of the marketplace. Elements of fairness, reciprocity, and equal sharing are interpreted in a physically pragmatic way, such as "you scratch my back and I'll scratch yours."

Level 2

At this level, maintaining the expectations of individual's family, group, or nation is perceived as valuable in its own right. The attitude is not only one of conformity of personal expectations and social order but loyalty to it, of actively maintaining, supporting, and justifying the social order.

Stage 3

The interpersonal concordance or "good-boy/nice-girl" orientation. Good behavior is that which pleases or helps others and is approved by them. There is much conformity to stereotypical images of what is majority or "natural" behavior.

Stage 4

The "law-and-order" orientation. The orientation is towards authority, fixed rules, and maintenance of the social order. Right action consists of doing one's duty, showing respect for authority, and maintaining the given social order for its own sake.

Level 3

At this level, there is a clear effort to define moral values and principles that have validity and application apart from the authority of the group or persons holding these principles.

Stage 5

The social-contract orientation (with utilitarian overtones). Right action is defined in terms of general individual rights and standards that have been critically examined and agreed upon by the whole society. There is a clear awareness of the relativism of personal values and opinions and a corresponding emphasis upon procedural rules for reaching consensus.

Stage 6

The universal ethical principle orientation. Right is defined by the decision of conscience in accord with self-chosen ethical principles appealing to logical comprehensiveness, universality, and consistency. These principles are abstract and ethical (the Golden Rule, the categorical imperative), such as universal principles of justice, the reciprocity and equality of human rights, and respect for the dignity of human beings as individual persons.

tonomous and are based on internal principles, such as the universal principle of justice or reciprocity.

According to Kohlberg, concepts at each stage are logically necessary for development at the next, and therefore the stages are seen to follow a sequential, invariant, and irreversible sequence. It is assumed that the process is universal and that thus all individuals will progress through the same sequence of stages, although the rate and endpoint may vary. Moreover, because the stages are described as structured wholes, individuals should reason about all moral problems at the same stage.

Semistructured clinical interviews, which assess the structure or organization of thinking rather than particular conclusions or attitudes held (Piaget, 1960), are used to measure the developmental maturity of moral reasoning. In these interviews, individuals respond to hypothetical dilemmas opposing different values, for instance, the value of life versus property, and responses are then coded in terms of moral stages (ranging from 1 to 6) and moral maturity scores (ranging from 100 to 600).

Since his initial work (Kohlberg, 1958), the Kohlberg scoring system has undergone several revisions, varying in their descriptions of the basic unit of analysis for scoring (sentences, global ratings, and the like), as well as the fundamental description of the stages. Although this has lead to criticism of Kohlberg's methods (Kurtines & Greif, 1974), successive revisions have more precisely distinguished the content from the structure of the stages. Recent data show excellent reliability and validity for the newest scoring system (Colby, 1978). Nevertheless, Kohlberg's system is time-consuming to administer and difficult to score, with intense training required for both.

These difficulties have lead other researchers to develop objective measures based on Kohlberg's theory. One of the most widely used is Rest's Defining Issues Test (Rest, 1979; Rest, Cooper, Coder, Masanz, & Anderson, 1974). In this recognition measure of moral reasoning, subjects are presented with 6 moral dilemmas and, for each, are asked to rate and rank the importance of 12 issue-statements pertaining to what ought to be done. Although a variety of scores can be derived, the *P*-index, or the relative importance that a subject gives to Stages 5 and 6 items, is one of the most frequently reported scores.

The validity of this measure as a structural-developmental assessment has been questioned (Loevinger & Wessler, 1970). First, it is questionable whether a standardized objective test that prestructures subjects' responses into prototypical statements can adequately assess the underlying structure of subjects' moral reasoning, because subjects are not allowed to explain and elaborate their thinking. Second, the scores are expressed as continuous variables (*P*, *D*, and *M* scores), rather than in terms of stages of reasoning. Thus, though relying upon structural-developmental theory for its inspiration and formulation, the theory is ignored in the scoring. Finally, and a serious concern with regard to research on juvenile delinquency, the DIT cannot be used with young subjects or subjects having less than a 12-year-old reading level (Rest, 1983).

More recently, it has been proposed that in Piaget's and in Kohlberg's model, morality and conventionality have been confused (Turiel, 1975, 1979, 1983). Although they differ as to the ages at which differentiation is thought to occur, both Piaget and Kohlberg describe principled justice as emerging from a global confusion of moral and nonmoral concepts. In contrast, Turiel has proposed that children at all ages distinguish morality, or acts pertaining to others' rights, welfare, or trust, from conventionality, or the behavioral uniformities that structure social interactions of individuals within different social systems. Morality is seen to be prescriptive, obligatory, universalizable, impersonal, ahistorical, and independent of rules and sanctions, whereas conventionality is seen to be changeable, contextually relative, and contingent on rules and authority sanctions.

According to this model, although there may be coordinations or intersections across the domains, children's reasoning would be expected to follow different sequential courses of development. Developmental transformations in children's thinking about social conventions have been described, but parallel research has not been conducted in the moral domain, although the structure of young children's reasoning about some narrowly defined aspects of justice has been examined (Damon, 1977, 1980; Irwin & Moore, 1971). One implication of the domain approach is that Kohlberg has defined the moral domain too broadly by confusing morality and conventionality at the lower stages (e.g., Stages 1 through 4). An adequate account of moral development would thus entail distinguishing concepts of social organization and social convention from concepts of justice, particularly at Kohlberg's Stages 3 and 4. Recent research (Berkowitz & Nucci, 1986) indicates that, when this is done, a core of reasoning pertaining more narrowly to justice can be identified.

In the definition of conduct disorders, moral and conventional behaviors are implied as repetitive and persistent patterns of conduct in which either the basic rights of others (moral issues of rights) or major age-appropriate societal norms or rules (social conventions) are violated. Generally, the conduct is seen as more serious than the ordinary mischief and pranks of children and adolescents (American Psychiatric Association, 1987). Later, the relevance of the moral/conventional distinction for research on moral reasoning and conduct disorders will be discussed.

With this background, I will now turn to the empirical research. First, research on the relationship between moral reasoning and delinquency and/or conduct disorders will be examined, followed by a review of the efficacy of intervention studies that have attempted to change delinquents' moral reasoning.

Relations between Moral Judgment and Delinquency

Only studies that included some measure of moral reasoning, that is, in which subjects were asked to justify or give reasons for their moral decisions, are reviewed. Unpublished material, such as doctoral dissertations, were included to be as comprehensive as possible. In the studies summarized, Kohlberg's measure of moral reasoning, Rest's Defining Issues Test, and measures based on Piaget's theory were most frequently used.

Table 2 summarizes the results of 35 studies examining relationships between moral reasoning and conduct disorders and/or delinquency. Most frequently, these studies tested the hypothesis that juvenile delinquents function at lower levels of moral reasoning than matched comparison groups of nondelinquents. It is important to note that, with few exceptions, factors that might affect moral reasoning, such as socioeconomic status, age, sex, ethnicity, and intelligence, were controlled. Thus, we can be reasonably confidant that differences observed between delinquent and nondelinquent groups reflect differences in their capacity to make moral judgments, rather than more general intellectual differences that may be reflected in measures of moral reasoning.

All but five of the studies used measures derived from Kohlberg's or Piaget's theories. Twelve of the 17 comparative studies using Kohlberg's measure show support for this hypothesis. One study (Bear & Richards, 1981) demonstrates positive and significant relationships between behavior disorders, as measured on Quay's Behavior Problem Checklist, and moral reasoning, but one study (Lee & Prentice, 1988), finds no such relationship. Only 3 of the 17 studies using Kohlberg's measure (Jurkovic & Prentice, 1974, 1977; Ruma, 1967) showed no support for the hypothesized relationship. An additional study (Petronio, 1980) found, contrary to hypotheses, that a repeat delinquent group had higher levels of moral reasoning than nonrepeating delinquents, and one other study (Ruma & Mosher, 1967) examined differences among a delinquent group but had no comparison group of nondelinquents.

Similarly, the results of studies utilizing objective measures derived from Kohlberg's theory are also overwhelmingly supportive of hypothesized relationships between juvenile delinquency and moral reasoning. Eight of the 9 studies using Rest's Defining Issues Test (including McColgan, Rest, & Pruitt's, 1983, two samples) presented in Table 2 indicate that delinquents' level of moral reasoning is lower than matched comparison groups, although the use of *P* scores make conclusions about delinquents' modal stage of moral reasoning more difficult to ascertain. Two other studies using another objective measure based upon Kohlberg's model also showed differences in the maturity of delinquents' versus nondelinquents' moral reasoning with age, IQ, and socioeconomic status controlled (Gavaghan, Arnold, & Gibbs, 1983; Gibbs, Arnold, Morgan, Schwartz, Gavaghan, & Tappan, 1984).

The results of the three studies utilizing Piagetian measures are more equivocal, although difficult to compare, because the studies examined different developmental dimensions or did not have comparable groups. For instance, one study (Eshel, Kugelmass, & Breznitz, 1968) found the predicted developmental differences between delinquents and nondelinquents on intentionality and moral realism, whereas another study (Haviland, 1977) found no differences in punitive versus restitutive reasoning. A third study (Miller, Zumoff, & Stephens, 1974), which reported differences between delinquents and retardates in their reasoning regarding intentionality and no differences between delinquents and normals in different Piagetian moral judgments, is of dubious value because of the uncontrolled differences in IQ between the three groups.

Thus, 22 out of the 28 studies relying upon

Table 2. Studies That Relate Moral Judgment and Delinquency or Conduct Disorders

Study	Subjects	Delinquency/nondelinquency	Basis for matching	Measure of moral judgment	Relation tested	Outcome
Bear & Richards (1981)	32 boys, 28 girls; ages 11–14	Behavior disorders: teacher ratings on Quay's Behavior Problem Checklist	Age, IQ, SES, sex, geographic area	Subjects selected on Kohlberg, written, group administered; final sample given individual Kohlberg Scores: modal stage MMS Stage: 2 as average	Conduct disorder and MJ stage with SES, IQ, and sex controlled	+
Betke (1944)	50 del, 50 nondel; ages 11–16	Del: court conviction, institutionalization Offenses: truancy, larceny, theft Nondel: NI	Age, IQ, SES, geographic area	Constructed by author, 25 social-moral problems, individual administration, scoring for correct logic and moral criteria (ethical vs. empathic vs. pragmatic)	Delinquency and correctness in logic Delinquency and use of ethical criteria Delinquency and use of empathic criteria Delinquency and use of pragmatic criteria	? + 0 +
Campagna & Harter (1975)	21 sociopaths, 23 normals; ages 10 & 13	Sociopaths: institutionalized; selected by Robin's criteria as evident in case history Normals: school records, counselors' reports, authors' classroom behavior checklist	Age, IQ, SES, geographic area	Kohlberg, interview, 4 dilemmas, global scoring, blind Score: Moral Maturity (MMS) Sociopathic stage: 2(1) as average	Sociopathy and MMS	+
Eisikovits & Sagi (1982)	26 del, 40 nondel, boys and girls; mean age 16.75 (Israel)	Del: institutionalized because of del acts Offenses: NI Nondel: NI	Age, SES, residential area, father's education, ethnic group	Ziv's morality measure, 7 situations, multiple-choice judgments, scored for Bull's 4 stages (anomic, heteronomic, socionomic, autonomic)	Delinquency and moral stage Moral stage and gender Delinquency, fathers' use of induction and moral stage	+ + +

(*continued*)

Table 2. ***(Continued)***

Study	Subjects	Delinquency/nondelinquency	Basis for matching	Measure of moral judgment	Relation tested	Outcome
Emler, Heather, & Winton (1978)	34 del, 34 nondel; ages 16–18 (Scotland)	Del: institutionalization; no serious disciplinary problem as indicated by type of facility Offenses: NI Nondel: high school boys or boys who had just left school	Age, SES, sex, urban residence	Rest's Defining Issues Test (DIT)	Delinquency and DIT Self-report of delinquency (seriousness and frequency) and DIT	+ 0 0
Eshel, Kugelmass, & Breznitz (1968)	68 del, 98 nondel; mean age approximately 13 (Israel)	Del: institutionalization Offenses: NI Nondel: from underprivileged schools	Age, parents' education, SES, ethnicity, immigrant status	Questionnaire constructed by authors based on Piaget; 12 transgressions, 2 of Piaget's stories, responses scored for intentionality, moral realism	Delinquency and intentionality Delinquency and moral realism	+ +
Fodor (1972)	40 del, 40 nondel; ages 14–17	Del: court referral Offenses: from petty larceny to attempted homocide Nondel: NI	Age, race, verbal IQ, mother's education	Kohlberg, written, 9 dilemmas, issue scoring, blind Score: MMS Del stages: approximately 3 as average	Delinquency and MMS Delinquency and social influence	+ +
Fodor (1973)	30 psychopathic del, 30 nonpsychopathic del; ages 14–17	Del: institutionalization Psychopathy: counselor's judgment according to Cleckley's criteria	Age, race, IQ, mother's education	Kohlberg, written, 9 dilemmas, issue scoring, blind Score: modal stage Del stages: 1–4	Psychopathy and MJ (Stages 1 & 2 vs. 3 & 4)	+
Gavaghan, Arnold, &Gibbs (1983)	60 del, 73 nondel, males and females; ages 14–18	Del: court conviction, institutionalization Offenses: from status offenders to homocide Nondel: high school students, NI	Age	Sociomoral Reflection Measure (SRM), written; Sociomoral Reflection Objective Measure (SROM), based on Kohlberg's stages Del stage: 2(3) as average on SRM	Delinquency and SRM Delinquency and SRM with age, SES, IQ controlled Delinquency and SROM Delinquency and SROM with age and SES controlled Delinquency and SROM with age, SES, IQ controlled	+ + + + 0

Gibbs, Arnold, Morgan, Schwartz, Gavaghan, & Tappan (1984)	6 samples, ages 11–65, including 60 male and female del, ages 14–18	Del: court conviction, institutionalization Offenses: NI Nondel: NI	None	Gibbs *et al.*'s SROM, SRM (see above); Kohlberg, 3 dilemmas, blind Scores: Kohlberg MMS, global stage scores; SRM modal stage, maturity score; SROM mean stage Del stage: average 2(3) on Kohlberg; modal stage 2 (SRM)	Delinquency and SROM Delinquency and SROM with age and SES controlled Delinquency and SROM with age, SES, IQ, controlled Delinquency and SRM with IQ, SES, age controlled	+ + 0 +
Hains (1984)	16 del, 18 nondel; ages 10–11 & 14–15	Older del: residential group homes; younger del: school authorities Offenses: runaway, incorrigibility, burglary, theft, overt aggressive behavior Nondel: schools	SES, age, geographic area	Rest's DIT, 3 dilemmas, 2 conditions: normal and with subject role-taking as policeman Scores: *P* index.	Delinquency and DIT Delinquency and normal vs. role-taking condition	+ 0
Hains & Miller (1980)	42 del, 54 nondel, boys & girls; ages 10–11, 12–13, 14–16	Del: Contact with juvenile authorities Offenses: status offenses, misdemeanors, felonies Nondel: NI	Age, SES, attendance at same school	Rest's DIT: 5 dilemmas, group administered, read to all delinquents and youngest nondel Scores: *D* index	Delinquency and DIT Delinquency, age, and DIT	+ +
Hains & Ryan (1983)	20 del, 20 nondel, ages 14–15; 14 del, 20 nondel, ages 10–11	Del: institutionalization Offenses: from status offenses to criminal acts Nondel: NI	Age, race	Rest's DIT: 5 dilemmas Eisenberg's prosocial dilemmas, 4 stories Scores: *D* index (DIT), concrete vs. abstract (prosocial)	Delinquency and DIT Delinquency and prosocial reasoning	0 0
Hanson & Mullis (1984)	43 male and 28 female del; 27 male and 27 female nondel; mean age 14.68	Del: institutionalization Offenses: considered serious offender (NI) Nondel: no criminal history, performing adequately within school system	Age, sex	Rest's DIT, group administered Scores: *D* index	Delinquency and DIT	+

(*continued*)

Table 2. (*Continued*)

Study	Subjects	Delinquency/nondelinquency	Basis for matching	Measure of moral judgment	Relation tested	Outcome
Haviland (1977)	22 del, 22 nondel; mean age 16.2	Del: institutionalization Nondel: high school students, NI	Age, IQ, same or similar communities	Three stories of theft, based on Piaget Score: punitive or restitutive reasoning	Delinquency and punitive vs. restitutive reasoning	0
Hawk & Peterson (1974)	15 del, ages 14–17; 196 nondel, ages 17–32; 15 nondel in therapy, ages 23–33	Del: voluntarily institutionalized by parents Offenses: ranging from running away from home to assault and battery Therapy group: membership in therapy collective	None	Kohlberg, written, 4 stories, global scoring Scores: MMS Del stage: approximately 3–4 as average	Delinquency and MMS Delinquency, education, and MMS	+ +
Hudgins & Prentice (1973)	10 del, 10 nondel; ages 14–16	Del: court convictions Offenses: auto theft or burglary Nondel: local high school, NI	Age, IQ, SES, race, geographic area	Kohlberg, written, 4 dilemmas, issue scoring Scores: MMS, global stage Del stages: 1–3	Delinquency and MMS Delinquency and MJ (Stages 1 & 2 vs. 3 & 4)	+ +
Jurkovic & Prentice (1974)	8 del, 8 nondel; mean age 15.0	Del: court conviciton, probation Offenses: car theft, burglary Nondel: school, court, counselors', parents' records; self-reports	Age, IQ, race, SES, geographic area, education, mother's education, number of siblings	Kohlberg, written, issue scoring, blind Scores: MMS, modal stage Del stages: approximately 2–3 as average	Delinquency and MMS	0
Jurkovic & Prentice (1977)	12 psychopathic, 12 neurotic, and 12 subcultural del; 12 nondel, approximate age 15	Del: institutionalization Offenses: burglary, theft, forgery, runaway, destruction of property, etc. Del subgroups: according to Quay's Behavior Problem Checklist Nondel: counselors' records	Age, race, SES	Kohlberg, 3 dilemmas, issue scoring, blind Scores: MMS, modal stage Del stages: 2 as average for psychopaths; 2(3) as average for neurotics; 2–3 average for subcultural delinquents	Psychopathic vs. neurotic delinquency and MMS Psychopathic vs. subcultural delinquency and MMS Psychopathic delinquents vs. nondelinquents and MMS Differences among other groups	+ + + 0

Kanter (1976)	159 del, divided into 5 groups on basis of DIT stage	Del: maximum security prison 4 categories of offenses: noneconomic violence, economic violence, economic violence, drugs	Groups (by stage) matched on age, IQ, race	Rest's DIT Del stages: 2 to principled	Higher vs. lower MJ stage and type of offense	+
Kohlberg (1958)	12 del, 12 nondel; age about 16	Del: awaiting trial, institutionalization Offenses: car theft, burglary, robbery by assault Nondel: NI	Age, IQ, SES	Kohlberg, interview, 9 dilemmas, global scoring, not blind Score: MMS Del stages: 2 as average	Delinquency and MJ	+
Kohlberg & Freundlich in Kohlberg (1978a)	20 del, 13 nondel; mean ages 19.3 & 19.9	Del: court conviction, institutionalization Offenses: felonious burglary, assault	Age, IQ, SES	Kohlberg, interview, 4 dilemmas, issue scoring, not blind Score: MMS	Delinquency and MMS Delinquency and percentage stage usage: Stages 2 vs. 3 & higher; Stage 4 vs. lower	+ +
Study 2		Nondel: school records		Del stages: approximately 2–3 as average		
Study 3	13 del, 13 nondel; mean ages 15.5 & 14.9 (Scotland)	Del: probation, institutionalization Nondel: in institution because home was unfit	Age, race, SES	Kohlberg, interview, 9 dilemmas, global scoring, blind Score: MMS Del stages: 2 as average	Delinquency and MMS Delinquency and stage usage: Stages 1 & 2 vs. 3 & 4	+ 0
Study 4	8 del, 10 nondel; ages 12–13; 7 del, 10 nondel; average age 15 (England)	Del: institutionalization Offenses: larceny Nondel: NI	Age, IQ, SES	Kohlberg, interview, 9 dilemmas, sentence scoring, blind Del stages: 2 as average	Delinquency and MMS Delinquency and Stages 1 & 2 vs. 3 & 4	+ +

(*continued*)

Table 2. ***(Continued)***

Study	Subjects	Delinquency/nondelinquency	Basis for matching	Measure of moral judgment	Relation tested	Outcome
Lee & Prentice (1988)	12 psychopathic, 12 neurotic, & 12 subcultural del, 18 nondel, approximately age 15	Del: Institutionalization; Offenses: burglary, theft, truancy, assault, illegal trespass or drug possession Del subgroups: according to Quay's Behavior Problem Checklist Nondel: School records	Age, ethnic group, IQ	Kohlberg, 2 dilemmas, issue scoring, blind Scores: MMS, modal stage Del stages: 2 as average for psychopathic, neurotic and subcultural groups	Delinquents vs. nondelinquents & MMS Psychopathic vs. subcultural delinquents & MMS Psychopathic vs. neurotic delinquents & MMS Neurotic vs. subcultural delinquents & MMS	+ 0 0 0
McColgan, Rest, & Pruitt (1983)	26 antisocial pre-delinquents, 26 nondel; age approximately 14	Predel: identified by Community Corrections Agency & Teachers and assigned to treatment program Offenses: disruptive and antisocial behavior Nondel: not in treatment program or regarded as flagrantly antisocial (NI)	Age, IQ, SES, race, sex, neighborhood and schools, parents' marital status, school grades	Rest's DIT, 6 dilemmas; Kohlberg, 3 dilemmas, issue scoring, blind Scores: *P* index on DIT; MMS Predel stage: 2 as average	Predelinquency and DIT Predelinquency and MMS	+ 0
Pilot study	29 del, mean age 16; 41 nondel, mean age 15	Del: institutionalization Offenses: NI Nondel: NI	Age, IQ, SES, race, region	Kohlberg, interview, 4 dilemmas, issue scoring, blind; Rest's DIT Scores: MMS, *P* index on DIT Del stages: 2 as average	Delinquency and DIT Delinquency and MMS Delinquency vs. predelinquency vs. control on DIT with IQ SES, age controlled Delinquency vs. predelinquency vs. control on MMS with IQ, SES, age controlled	+ + + +
Miller, Zumoff, & Stephens (1974)	16 del girls, 16 female retardates, IQ 50–75; 15 normal girls, IQ 90–110; ages 13–16	Del: convicted by courts, institutionalization Offenses: promiscuity, incorrigibility, truancy, running away, thievery, overtly aggressive acts Normals: public schools	Age, sex, SES	Constructed by author, based on Piaget, intentionality, retributive vs. restitutive justice, subjective vs. objective responsibility	Delinquents vs. retardates on intentionality Delinquents vs. normals on all moral judgments	+ 0

Nucci & Herman (1982)	20 behavior disordered, 22 normals; ages 9–11	BD: special education diagnosticians; teachers' ratings on Children's Psychiatric Rating form Offenses: acting out but little depression, hallucinations, or withdrawal NonBD: NI	IQ, SES, schools	Reasoning regarding 15 moral, conventional, and personal acts, as defined by Turiel; scored in justification categories	BD and reasoning regarding justice BD and reasoning regarding personal jurisdiction	+ +
Petronio (1980)	38 del boys; 19 repeaters and 19 nonrepeaters; ages 13–17	Del: on probation, under supervision of juvenile court Repeaters: returned to court at least once within 2 years of being placed on probation Nonrepeaters: had not been returned to court within same period	Age, IQ, SES	Kohlberg, interview, 5 dilemmas, "thought unit" scoring Score: MMS Del stages: M = not given	Repeat delinquency and Kohlberg MMS	−
Ruma (1967)	30 del, 30 nondel; ages 15–17	Del: court convictions, institutionalization Offenses: vandalism, theft, assault Nondel: members of Boys' Clubs	Age, IQ, SES, geographical area	(a) Kohlberg, interview, 9 dilemmas, global scoring, not blind Score: modal stage or stage mixture Del stages: 1–5. (b) Relevant situation MJ; procedure as in (a) with everyday situations	Delinquency and Kohlberg MJ Delinquency and relevant situation	0 0
Ruma & Mosher (1967)	36 del; ages 15–17	Del: court conviction, institutionalization Offenses: car theft, assault, incorrigible behavior problems	None	Kohlberg, interview, 6 dilemmas Score: Modal stage Del stages: 1–3	MJ stage & guilt (Mosher Guilt Scale) MJ stage and intensity of guilt (clinical ratings) MJ stage and internalized moral standards	+ + +
Ryan (1985)	88 del, 88 nondel	Del: institutionalization Offenses: NI Nondel: NI	NI	Rest's DIT, short form Del stages: 3 &4	Delinquency and moral stage	0

(continued)

Table 2. (*Continued*)

Study	Subjects	Delinquency/nondelinquency	Basis for matching	Measure of moral judgment	Relation tested	Outcome
Sagi & Eisikovits (1981)	103 male and female del, 146 nondel; ages 14, 16, 18 (Israel)	Del: court convictions, institutionalization Offenses: NI Nondel: NI	SES, education, age, sex, neighborhood	Ziv's morality test, 7 situations, multiple-choice judgments fitting Bull's stages (anomic, heteronomic, socionomic, autonomic)	Delinquency and moral stage	+
Schmidlin (1977)	30 institutionalized del, 30 community-placed del, 30 nondel; ages 14–17	Del: institutionalization (past or present) Offenses: runaway, theft, drugs, arson, armed robbery Nondel: court and school records, self-reports	Age, IQ, SES, race, geographic area, type of offense (for del groups)	Maitland & Goldman's (1974) Moral Judgment Scale, based on Kohlberg's theory; 15 multiple-choice items, each alternative representing a stage Del stages: 3–4	Institutionalized del vs. community-placed del vs. nondel and MJ	0

Note: del = delinquent; nondel = nondelinquent; BD = behaviorally disordered; nonBD = not behaviorally disordered; NI = no information; SES = socioeconomic status; MMS = Moral Maturity Score on Kohlberg's scale; MJ = moral judgment; DIT = Defining Issues Test. All subjects were male unless otherwise noted. A + denotes support for a positive relation between delinquency or conduct disorders and moral reasoning; − indicates a negative relation between delinquency or conduct disorders and moral reasoning; a 0 indicates no support for such a relation; a ? indicates that the results are ambiguous. Based upon Blasi (1980).

Kohlberg's model (i.e., those using his measure, Rest's measure, and one other objective measure) show support for the hypothesis that juvenile delinquents are more developmentally immature in their moral reasoning than nondelinquents. These results are impressive; indeed, as one author (Blasi, 1980) noted, research on juvenile delinquents appears to yield the strongest support of any area for a relationship between moral judgment and action. And yet, this conclusion obscures the considerable variability in these findings.

For instance, although delinquents were, on average, found to be functioning primarily at an instrumental relativistic (Stage 2) level, as Kohlberg (1958, 1978a) predicted, a number of investigators (Fodor, 1972, 1973; Hudgins & Prentice, 1973; Jurkovic & Prentice, 1974; Kohlberg, 1978a; Ruma, 1967; Ruma & Mosher, 1967; Ryan, 1985) found delinquents reasoning at Stage 3 or even found Stage 3 to be the delinquents' dominant moral stage. Furthermore, older delinquent samples were sometimes found to exhibit more Stage 3 reasoning than younger delinquent samples, suggesting that delinquents' moral reasoning develops in adolescence, as does the moral reasoning of nondelinquents. Therefore, delinquents' reasoning is more variable than has been hypothesized, and their behavior cannot be attributed entirely to the immaturity of their moral judgments.

There is also considerable conceptual confusion in the findings. For instance, the definition of delinquency as it pertains conceptually to morality is problematic. Whereas conduct disorders represent a psychiatric classification, delinquency is a legal term referring to a juvenile (usually under 18) who is brought to the attention of the justice system by virtue of his or her committing a criminal act or displaying a variety of other behaviors not specified under criminal law (e.g., truancy or curfew violations). In most of the studies described in Table 2, delinquency was defined in terms of trial and conviction in court, institutionalization in a juvenile facility, or both. There are some problems and pitfalls in using this definition. For instance, it is not clear how delinquents who are caught and adjudicated differ from those who are not. This concern is relevant in selecting the delinquent sample as well as the appropriate comparison groups. Although most studies have been quite rigorous in controlling for factors that may confound moral reasoning scores, very few studies have taken adequate precautions to determine whether subjects in their nondelinquent comparison groups have engaged in criminal acts (Blasi, 1980). We found only 7 studies (Campagna & Harter, 1975; Hanson & Mullis, 1984; Kohlberg & Freundlich, Study 2, noted in Kohlberg, 1978a; Jurkovic, 1976; Jurkovic & Prentice, 1974; McColgan *et. al.*, 1983; Schmidlin, 1977) that described precautions taken (typically, school records or parents' or counselors' reports) to remove this possible source of confounding.

Complementarily, only one study (McColgan *et al.*, 1983) bears on whether institutionalization may represent a confounding factor when examining delinquents' moral reasoning. In their study, a sample of predelinquent boys drawn from a contingency management classroom was compared to a group of very carefully matched nondelinquents attending the same school. The *P* index of the Defining Issues Test (DIT) was found to discriminate the groups, but scores on the Kohlberg measure did not. Since the DIT is a recognition task, the authors argue that perhaps the tacit awareness of higher levels of moral thinking may organize behavior. Of course, it is also possible that there is no difference between nonadjudicated and nonincarcerated delinquents and matched comparison groups in their moral reasoning and that the differences obtained on the DIT were due to some uncontrolled source of covariation. Therefore, it is not clear what effect, if any, institutionalization has on children's moral reasoning. However, Kohlberg, Scharf, and Hickey (1973) have asserted that prison inmates at different levels of moral reasoning perceive the "moral atmosphere" of prisons to be punitive-coercive (Stage 1) or instrumental-relativistic (Stage 2) and that prisons may thus elicit greater instrumental relativism among their inmates than would otherwise be obtained.

A second major problem with these conclusions is that juvenile delinquents form a very heterogeneous group. Indeed, there have been a variety of attempts to subclassify juvenile delinquency (Hewitt & Jenkins, 1946; Jenkins, 1973; Quay, 1964, 1979), and four subtypes are currently recognized in the American Psychiatric Association's (1987) definition of conduct disorders. Consistent with these approaches, several studies have attempted to distinguish delinquents on psychological criteria. Three of the four studies (Campagna & Harter, 1975; Fodor, 1973; Jurkovic & Prentice, 1977) that employed a personality approach were successful in finding differences in subgroups of delinquents. Campagna and Harter (1975) found that delinquents classified as sociopathic according to Robin's (1966) criteria displayed significantly more Stage 2 moral reasoning than an appropriately matched comparison group of nondelinquents.

Fodor (1973) found that delinquents who were classified as psychopathic according to Cleckley's (1955) criteria scored lower on Kohlberg's measure than a group of nonpsychopathic delinquents. They reasoned at primarily (80%) Stages 1 and 2, whereas the nonpsychopathic delinquents were found to reason at the first two levels (Stages 1 and 2 vs. 3 and 4) at nearly even frequencies. Finally, two studies (Jurkovic & Prentice, 1977; Lee & Prentice, 1988) differentiated between homogeneous groups of unsocialized psychopathic, neurotic disturbed, and socialized subcultural delinquents on the basis of Quay's Behavior Problem Checklist and compared their moral reasoning to a matched group of nondelinquents. Consistent with the aforementioned study, Jurkovic and Prentice (1977) found that the moral judgments of the psychopathic delinquents on Kohlberg's measure were almost entirely at Stages 1 and 2, whereas the average level of each of the other groups approached Stage 3. However, Lee and Prentice (1988) found no relationship between delinquent subgroup and moral reasoning on Kohlberg's measure.

Juvenile delinquents have also committed a variety of offenses varying considerably both across and within studies. They range from truancy, drug use, car theft, and serious arson to aggression against persons and attempted homicide. The offenses can be seen to vary in seriousness and also—to return to a distinction made earlier—in conceptual domain. That is, some offenses, such as truancy, running away, and drug-related offenses, are primarily social-conventional in nature, whereas others are moral in that they entail violations of others' welfare or rights. It can be hypothesized that for those who commit conventional offenses, the appropriate domain for comparison may be the conventional, and not the moral domain. That is, although there may be deficits in the developmental maturity of conventional offenders' conventional reasoning, their moral reasoning may be no less mature than that of nonoffenders (see Turiel & Davidson, 1986, for an elaboration of the general argument).

Only one study (Kantner, 1976) directly attempted a behavioral rather than a psychological classification of juvenile delinquents. In this study, variation in moral reasoning was examined as a function of the type of offense. He grouped the offenses into four categories: violence not related to property (i.e., toward people), violence related to property, offenses against property without violence, and drug-related offenses. According to Turiel's (1983) definition of the domains, the first three can be seen as primarily moral offenses, whereas the latter may be primarily conventional (although details of the specific crimes were not provided). He found no differences between the groups, although the delinquent sample was not compared with a nondelinquent group.

A similar analysis was performed by Kohlberg (1978a). Noting that, in contrast to his expectations, a sizeable proportion of one of his delinquent samples was at the Stage 3 level of judgment, he examined the relationship between moral orientation and drug use. The sample was classified as to whether they had a hard drug habit (which was supported by illegal activities) or no drug habit. Half of the 40 inmates in the sample were classified as drug-users; of these, 70% were found to be at Stage 3, as compared to only 35% of the nondrug-users. These data are consistent with the proposition that delinquents who commit nonmoral (albeit illegal) offenses are more mature moral reasoners than those who commit crimes that are moral in nature. These data are only suggestive, however, because the exact nature of the drug-related offenses was not provided.

Another way of examining the potential confounding of conceptual domain, although also quite speculative, is to consider the data on sex differences in delinquents' moral reasoning. Epidemiological studies indicate that disorders of conduct are more common among boys than girls; criminal statistics and self-report data similarly indicate that the preponderance of juvenile delinquents are male (Rutter & Garmezy, 1983). Interestingly, the offenses for which girls are convicted are characteristically different from those for boys. Girls are much more likely to be found guilty of sexual misdemeanors (for instance, promiscuity), truancy, or incorrigibility (Cowie, Cowie, & Slater, 1968). Thus, girls are more likely to be convicted and institutionalized for conventional offenses than are boys. This suggests that female delinquents may be at higher developmental levels of moral maturity than male delinquents. (Of course, the respondent's sex provides only a very crude approximation of domain, because females can and do commit moral offenses.)

Most of the studies reviewed in Table 2 consisted of male samples. Only eight studies had samples consisting of male and female delinquents, and one study (Miller *et al.*, 1974) had an all-female sample. In most, no information was provided about the type of offenses committed or the relative frequencies of different kinds of offenses committed by boys and girls.

Of these nine studies, three (Eisikovits & Sagi, 1982; Hains & Miller, 1980; Hawk & Peterson, 1974) either did not examine or provided no information on sex differences in moral maturity. Of the remaining five comparative studies, two reported no sex differences among delinquents (Gavaghan *et al.*, 1983; Gibbs, Arnold, Morgan, Schwartz, Gavaghan, & Tappan, 1984), and three reported some evidence consistent with the hypothesis that female delinquents may be at developmentally higher levels of moral maturity than males (Bear & Richards, 1981; Hanson & Mullis, 1984; Sagi & Eisikovits, 1981). The one study comparing female delinquents and nondelinquents found no differences in Piagetian measures of moral maturity (Miller *et al.*, 1974), as might be expected if females' offenses are of a conventional rather than moral nature. Thus, there is some evidence to suggest that the conceptual domain of the offense (moral vs. conventional) is confounded in these studies and that a careful separation of delinquent subgroups on the basis of a conceptual domain analysis would yield a more precise indicator of the relationship between moral reasoning and juvenile delinquency.

Nevertheless, the results of these studies do suggest that juvenile delinquents generally function at a lower level of moral maturity than matched comparison groups of nondelinquents. These conclusions have lead a number of investigators to attempt to modify delinquents' moral reasoning through training or the facilitation of moral judgment, with the assumption that the development of Stage 3 moral reasoning on Kohlberg's scale will reduce the frequency of delinquent behavior (although only a few studies actually tested that hypothesis). In the next section, the effectiveness of these interventions is discussed.

Modification of Juvenile Delinquents' Moral Reasoning

Table 3 presents the results of studies attempting to raise the developmental level of delinquents' moral reasoning. Of the 12 studies described in Table 3, 11 were interventions with juvenile delinquents, and one intervened with behavioral disordered adolescents (Arbuthnot & Gordon, 1986). As with the comparative studies, the measures were primarily Kohlberg's or Rest's, or were based upon Piaget's work. Most of the interventions were based upon Blatt and Kohlberg's (1975) finding that group discussion of moral dilemmas facilitates the development of moral reasoning.

Three studies compared the effectiveness of different types of interventions. Gibbs, Arnold, Ahlborn, and Cheesman (1984) examined the effectiveness of weekly discussion groups in which the participants were either required or not required to reach consensus on the best decision for discussed dilemmas and a control group which did not meet for discussions. They found no differences in the efficacy of the two types of discussion groups on Gibbs's Sociomoral Reflection Measure (SRM). However, for subjects who scored at Stage 2 on the SRM at the pretest, they found significant pretest–posttest gains of one stage in moral maturity. Subjects who scored at Stage 3 at the pretest showed no change in moral reasoning.

Similarly, Jennings and Kohlberg (1983) compared the effectiveness of a "Just Community" approach, in which participants establish and regulate the rules of their community, with two behavior modification programs and a transactional analysis program in raising inmates' moral reasoning. The Just Community approach was found to be more effective than the other two types of programs, with inmates participating in the Just Community gaining one-half of a moral judgment stage.

Comparing the effectiveness of weekly discussion groups that focused on interaction and consensus with values clarification sessions using the same dilemmas, and a control group that received no treatment, Niles (1986) found significant shifts of about one-third of a stage on Kohlberg's measure for the discussion group subjects but not for the control or the values clarification subjects. Shifts occurred from Stage 1 to Stage 2, but Stage 2 subjects generally did not move.

The remaining studies primarily assessed the effectiveness of weekly discussions of moral dilemmas, as compared to control adolescents who did not participate in discussion, in facilitating moral reasoning. Arbuthnot and Gordon (1986), for instance, found that weekly discussions geared at one stage above the participants' level of moral reasoning (a "+1 induction," according to Blatt & Kohlberg, 1975) significantly raised behavioral disordered adolescents' moral reasoning. Both Hickey (1972) and Ventis (1976) similarly found that sessions focusing on the discussion of moral conflict situations lead to increases of one-fifth of a stage (in the former study) or one-fourth of a stage (in the latter study) on Kohlberg's measure (although Hickey, 1972, did not examine differences between the experimental and the control groups).

Table 3. Moral Judgment Interventions with Delinquents or Conduct-Disordered Adolescents

Study	Subjects	Delinquency (BD)	Measure of moral judgment	Intervention	Relation tested	Outcome
Arbuthnot & Gordon (1986)	35 male, 13 female BD; ages 13–17	BD: Teacher's nominations; ratings on School Adjustment Index Offenses: unruliness, aggressiveness, lying, impulsivity, disruptiveness, stealing, vandalism, fire-setting Assignment to treatment vs. control: random	Kohlberg, blind Pretest: 3 dilemmas Posttest: 3 dilemmas Scores: MMS, global BD Pretest scores: 2 as average	Treatment groups: moral dilemma discussion sections 45 min/week for 16 weeks; Blatt & Kohlberg "+1" stage induction Control groups: no discussion groups Assessment: week 3 (pre), week 16 (post), 12 months after posttest (followup)	Pre-post for treatment group on MMS (+1/2 stage) Pre-post for control group on MMS (−1/2) Treatment vs. control on MMS at follow-up Post-follow-up, treatment vs. control on MMS	+ − + +
Fleetwood & Parish (1976)	13 female, 16 male del; ages 16–17	Del: adjudged by county youth services program Offenses: excessive truancy, drug abuse, sex offenses, robbery Assignment to treatment vs. control: nonrandom (worst offenders in treatment group)	Rest's DIT given at pre-, posttest, 6 stories Scores: *P* index, modal stage Discussion groups: 6 dilemmas, constructed by author, based on Biblical stories Pretest stages: 3 as average for treatment; 5 as average for control	Treatment group: moral dilemma discussion groups 1.5 hrs/week for 5 weeks; 5-phase format: present conflict situation, list solutions, elaborate consequences, researcher support, group consensus of opinion Control group: no group discussion Assessment: week 1 (pre), week 5 (post)	Pre-post for treatment group on *P* scores Pre-post for treatment group on modal stage Pre-post for control on *P* scores, modal stage Treatment vs. control on *P* scores, modal stage	+ + 0 +
Gibbs, Arnold, Ahlborn, & Cheesman (1984)	30 male, 30 female del; ages 14–18	Del: court convictions, institutionalization; males classified as serious offenders Offenses: NI Assignment to consensus dilemma, nonconsensus dilemma, no discussion	Gibb's SRM, blind Dilemma Decisions Survey (DDS), 10 dilemmas, both pre-post Scores: modal stage, maturity scores (SRMS); yes/no judgments (DDS)	Treatment groups: moral discussion (2 types), 40 min/week for 8 weeks Consensus dilemma discussion: no agreement on best decision; nonconsensus dilemma discussion: not required to	Pre-post for Stage 2 exptl subjects on modal stage SRMS (+1 change) Pre-post for control on modal stage, SRMS: ns Pre-post for Stage 3 exptl subjects on modal stage SRMS (0 change)	+ 0 0

		groups: triadically grouped according to SRM scores, then randomly assigned	Pretest stage: 2(3) as average, range 2 to 3	reach agreement Control: no discussions Assessment: week 1 (pre), week 9 (post)	Consensus dilemma discussion vs. nonconsensus discussion, ns	0
Hickey (1972)	40 del; ages 16–22	Del: institutionalization Offenses: NI Assignment to treatment group: random	Kohlberg, blind Scores: MMS	Treatment group: discussion of moral conflict situations, 36 1.5hr sessions; 8 inmates: 72 more sessions Controls: no discussion	Pre-post for treatment on MMS (+1/5 stage) Pre-post for control on MMS Pretest follow-up for exptl group on MMS	+ 0 +
Jennings & Kohlberg (1983)	48 del; age approximately 15	Del: residence in 3 unlocked residential homes; institutionalization in locked facility Offenses: NI (1 program), serious behavior problems or problems seen as modifiable (2 programs); character disordered as diagnosed by staff (4th program) Assignment to treatment group: by facility; nonrandom	Kohlberg, 3 dilemmas; Ethnographic Moral Atmosphere Interview Scores: global stage scores, MMS (Kohlberg) Pretest stage: 2 as average	Just Community: weekly community meetings Open Behavior Modification: rewards & punishments for appropriate behavior Secure Behavior Modification program: same Transactional Analysis program: verbal transactions examined Pre-post assessment: initially and then after 8 months in each program	Pre-post for Just Community on MMS (+1/2) Pre-post for transactional analysis, behavior modification on MMS, ns Change scores, Just Community vs. behavior modification, transactional analysis Change scores, Just Community vs. behavior modification, transactional analysis with pretest scores controlled	+ 0 + +
McCann & Prentice (1973)	12 del; average age 12.6	Del: referred to juvenile court Offenses: burglary, theft, glue sniffing Assignment to treatment group: random	Intentionality: two 12-item pairs (Bandura & McDonald) Pretest scores: lacking intentionality	Treatment: Given additional items and promised $.10 for responses that match (hypothetical) teachers Control: given additional items Pre-post assessment: one session (pre, exptl induction, post)	Pre-post for exptl group on intentionality Pre-post for control group on intentionality Exptl vs. control at pre Exptl vs. control at post	+ 0 0 +

(*continued*)

Table 3. (*Continued*)

Study	Subjects	Delinquency (BD)	Measure of moral judgment	Intervention	Relation tested	Outcome
Niles (1986)	59 del and predel boys; ages 13–15	Del: enrollment in institutional or day school special education program; adjudicated youthful offender status or had persons-in-need-of-supervision; petition against them by parents Offenses: purse-snatching to burglary Assignment to treatment group: matched by MJ score within school	Kohlberg, Standard Form scoring Pretest: Stage 2 as average Scores: MMS	Treatment group: moral discussion, focusing on interaction and resolution of dilemmas, twice a week for 16 weeks Placebo group: Same materials, used values clarification rather than interaction or consensus Control group: no treatment Assessment: pretest 1 week prior to moral discussion group; posttest 1 week following last discussion	Pre-post for moral discussion group on MMS (+1/3 stage) Pre-post for values clarification or control on MMS Moral discussion vs. placebo or control at post	+ 0 +
Prentice (1972)	36 del; ages 13	Del: minimum of 3 del offenses Offenses: burglary, theft, runaway, arson, assault, glue sniffing Assignment to treatment groups: random	Intentionality as measured by 48 story pairs; moral relativism using Moral Realism scale; two 12-item forms Pretest scores: del selected for lack of intentionality	Treatment groups: intentionality responses presented by live or symbolic modeling Control: no modeling Pre-post assessment: intervention within 24 hours of pretest, followed by posttest within 24 hrs; follow-up 9 months after posttest	Pre-post on intentionality for exptl groups Exptl vs. control groups on intentionality Live vs. symbolic models Exptl vs. control groups on moral realism Pre-post on realism Exptl vs. control groups at follow-up on number or type of offenses	+ + 0 0 − 0
Rosenkoetter, Landman, & Mazak (1980)	13 male, 6 female del: ages 13–16	Del: probation Offenses: incorrigible behavior, theft, assault Assignment to treatment group: random	Kohlberg, 3 dilemmas blind Scores: MMS Pretest stage: 2 as average	Treatment groups: moral discussion groups 1.5 hrs/week for 7 weeks Assessment: 3 weeks preceding and following intervention No control group	Pre-post on MMS (+1/5 stage)	+

Schmidlin (1977)	30 institutionalized del, 30 community-placed del; 30 nondel; ages 14–17	Del: institutionalized (past or present) Offenses: runaway, theft, drugs, arson, armed robbery Nondel: court and school records, self-reports	Maitland & Goldman's (1974) Moral Judgment Scale, based on Kohlberg's theory; 15 multiple choice items, each alternative representing a stage Pretest del stages: 3–4	Treatment: Peer group discussion (NI)	Pre-post for treatment group on Moral Judgment Scale	0
Smith (1985)	23 black and 19 white del	Del: institutionalized Offenses: NI Assignment to treatment groups; NI	Rest's DIT	Treatment: guided moral discussion groups Control: activity control groups	Treatment vs. control groups on DIT	0
Ventis (1976)	57 del, 19 nondel; ages 13–16	Del: convicted; 6 months left on probation Offenses: NI (not hard drug offenders) Nondel: school system Assignment to del treatment groups: matched for race, age, offenses, IQ, then random	Kohlberg 3 dilemmas, blind Scores: MMS Pretest del stages (exptl and control): 2	Treatment: moral discussion groups for 12 weeks, 40 sessions Assessment: 1 month interval preceding intervention, 5–6 months between pre- and posttesting Del and nondel control groups Controls: no discussion	Exptl vs. control on MMS at pretest Pre-post for exptl del group on MMS (+1/3 stage) Pre-post for del controls (+16 points on MMS) Pre-post for nondel controls (+1/4 stage on MMS) Exptl vs. control at post ($p < .15$)	0 + 0 0 +
Ventis (1980)	16 white and 7 black del; ages 13–16	Del: institutionalized Offenses: ranged from status offenders to felonies Assignment to treatment groups: by facility; no differences in age, IQ, number of offenses; differed by race & severity of offenses	Kohlberg, 3 dilemmas, blind Scores: MMS	Treatment group: small-group discussions of hypothetical dilemmas, 1 hr 2 times/week for 7 weeks Assessment: week 1 (pre), week 9 (post) Controls: no discussion	Pre-post for exptl group Pre-post for control group on MMS Exptl vs. control at pre on MMS Exptl vs. control at post on MMS (+1/5 stage)	0 0 0 +

Note: del = delinquent; nondel = nondelinquent; BD = behaviorally disordered; nonBD = nonbehaviorally disordered; NI = no information was provided; MMS = Moral Maturity Score on Kohlberg's scale; DIT = Defining Issues Test; exptl = experimental group; pre = pretest; post = posttest; ns = not significant. All subjects were males unless otherwise noted. A + denotes positive support for the effect of the intervention on moral reasoning; a 0 indicates no support for such an effect; and a − indicates a negative relation between the intervention and moral reasoning. Based upon Blasi (1980).

In three other studies (Fleetwood & Parish, 1976; Rosenkoetter, Landman, & Mazak, 1980; Ventis, 1980), moral discussion groups were found to raise significantly delinquents' moral reasoning on Rest's and on Kohlberg's measures, but the lack of random assignment to treatment versus control groups in the Fleetwood and Parish (1976) and the Ventis (1980) studies and the lack of a control group in Rosenkoetter *et al.* (1980) seriously limit the generalizability of these findings. Only two studies (Schmidlin, 1977; Smith, 1985) reported no significant gains with this type of intervention.

Prentice (1972) examined the effects of live versus symbolic modeling on delinquents' reasoning regarding intentionality. Following a single-session intervention, delinquents in both modeling conditions showed significant gains in reasoning, as compared to a control group. The ease at which the expressed moral orientations were enhanced led to speculation that the more mature responses may already have been acquired, and that the changes may have been due to response set. Thus, McCaan and Prentice (1973) subsequently attempted to modify delinquents' reasoning about intentionality by altering their response set. Delinquents selected on the basis of their low pretest scores on intentionality were given incentives to change their moral orientation. They were told that for every response that matched a hypothetical teacher's response, they would be given a reward. This inducement lead to a significant change in moral orientation. However, research (extensively reviewed in Keasey, 1979) on the aspect of intentionality examined in these studies (e.g., material damage) has not supported the developmental progression proposed by Piaget (1932). Thus, it is unlikely that these training procedures actually facilitated developmental changes in the structure of delinquents' moral reasoning.

Overall, these results suggest that structured opportunities to engage in discussion of moral dilemmas produce some statistically significant gains in delinquents' or behavioral disordered youths' moral reasoning, but are the changes produced through such interventions maintained over time? Only one study (Arbuthnot & Gordon, 1986) addressed this question by conducting a long-term follow-up of moral reasoning. Their results are encouraging. They found that behavioral disordered adolescents who had participated in moral dilemma discussion groups still reasoned at significantly higher levels one year after the posttest than those who had not. Furthermore, over time, the two groups continued to diverge in their level of moral reasoning. These results suggest that interventions may produce changes in moral maturity that are maintained or even enhanced over time.

The developmental changes in moral orientation produced by these intervention programs typically are not large, however, and range from one-fifth to one stage. One way of examining their pragmatic benefit is to examine whether changes in moral reasoning are accompanied by changes in behavior. Although almost all investigators raised this question, only two studies (Arbuthnot & Gordon, 1986; Prentice, 1972) directly examined this issue, and a third (Jennings & Kohlberg, 1983) examined it indirectly. The results are equivocal. Arbuthnot and Gordon (1986) reported that one year after the posttest, behavioral disordered adolescents participating in a moral reasoning intervention had lower rates of absenteeism and tardiness and higher grades than did control adolescents. There were no differences between the groups in recorded police or court contacts, with both groups showing no contacts. Prentice (1972) reported no differences between their experimental and control group delinquents in the number or type of criminal offenses at the 9-month follow-up, although questions about whether their intervention actually produced developmental changes make the value of these findings questionable. Finally, Jennings and Kohlberg (1983) reported perceived changes in Just Community participants' morally relevant actions toward other persons. Because the interviewer was the program director, however, reported changes in moral atmosphere may have been due to social desirability.

In summary, attempts to enhance behavioral disordered or delinquents' moral reasoning appear to produce small but significant changes in moral maturity. The results of one study suggest that such gains may be maintained over time, but further research on this issue is needed. It is as yet unclear whether developmental changes in moral reasoning are accompanied by changes in delinquents' or in behavioral disordered adolescents' behavior.

Conclusions

Piaget's (1932) early speculation about the utility of a developmental analysis in understanding conduct-disordered children has proven fruitful. A variety of studies indicate that with intelligence and other factors controlled, antisocial children reason at lower levels of moral maturity than their nondisturbed counterparts. These findings still leave a number of

unanswered questions. Primary among these is a developmental explanation of why these deficits occur. An adequate theory of the moral development of conduct-disordered children needs to specify the social experiences that lead to delays or deficits in the capacity to make moral judgments. Furthermore, because not all juvenile delinquents are developmentally deficient in their moral reasoning, the groups who are most at risk need to be identified. One promising route would be to attend to the moral relevance (e.g., the conceptual domain) of the behavior or offenses that are committed. Not all delinquent acts are moral offenses. More careful attention needs to be given to the type of act committed, as deficits in moral reasoning would be expected only among the subgroup of delinquents who have committed moral offenses. One practical implication of this, which remains to be empirically tested, is that these delinquents, but not those having committed conventional offenses, would be appropriate candidates for moral-reasoning interventions. In addition, those who focus on the moral reasoning of delinquents need to remember that juvenile delinquency is multiply determined and that immaturities in moral reasoning are only one variable influencing behavioral disordered children's behavior. Thus, research on delinquents' moral reasoning needs to be incorporated in more multidimensional models. Finally, although the psychiatric category examined in this chapter is conduct disorders, the relevant research pertains primarily to those who have come to the attention of the law (e.g., juvenile delinquents). More research is needed on the moral development of children who are classified within the broader category of conduct disorders, including younger children with a range of behavior problems. Such research would be an important contribution to an emerging discipline of developmental psychopathology.

Acknowledgments

I am grateful to Cynthia Rohrbeck and Elliot Turiel for their comments on earlier drafts of this chapter.

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CHAPTER 14

The Development of Prosocial Behavior versus Nonprosocial Behavior in Children

Nancy Eisenberg and Paul Miller

The purpose of this chapter is to review factors related to behavior in children that is egoistic, selfish, and lacking in prosocial elements. There is, however, little research in which the primary intent of the investigator was to study such behavior; most researchers concerned with children's negative moral behavior have focused upon delinquency, aggression, dishonesty, and lack of resistance to temptation, but not upon selfishness and related behaviors (Hoffman, 1970; Parke & Slaby, 1983). Nonetheless, there is considerable information available concerning our topic—information embedded in the large and fast-growing literature concerning the development and maintenance of prosocial behavior (i. e., voluntary behavior intended to benefit another), including altruistic behavior (i. e., voluntary behavior intended to benefit another which is not motivated by the expectation of external reward; Eisenberg, 1982; Staub, 1978). Thus, if one is to understand the lack of prosocial behavior, it is necessary to study it in relation to prosocial development. Consequently, we have used the literature concerning prosocial behavior in children as the basis for this chapter.

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Because most of the relevant research is embedded in the prosocial literature, a few caveats are in order. In the first place, the lack of prosocial behavior does not necessarily imply selfish behavior. Children may not assist others for a variety of reasons, including feelings of incompetence (Midlarsky & Hannah, 1985; Peterson, 1983), lack of social assertiveness (e. g., Larrieu, 1984), or parental emphasis on the inappropriateness of prosocial action in some setting (Peterson, Reaven, & Homer, 1984). In most cases, investigators have not assessed individuals' reasons for failing to assist others. Similarly, people may act in a prosocial manner for a variety of reasons, including egoistic as well as moral reasons (Eisenberg, 1982, 1986). Thus, one cannot simply assume that a behavior that appears to be moral actually reflects positive motives. Nonetheless, behavior that initially is performed for nonmoral reasons may, over the course of socialization, come under the control of moral motives (Staub, 1979). For this reason and because of the lack of information concerning motives for individuals' behavior in most studies, we have operated under the assumption that the lack of comforting, sharing, helping, and other prosocial behaviors can, in general, be considered indicative of an undesirable pattern of behavior.

The body of work concerning prosocial behav-

ior is quite large and diverse in content. Investigators have studied everything from socialization antecedents and correlates of prosocial behavior, to situational factors that elicit or inhibit prosocial responding, to personal and demographic correlates of altruism (Eisenberg, 1982, 1986; Radke-Yarrow, Zahn-Waxler, & Chapman, 1983; Rushton & Sorrentino, 1981; Staub, 1978, 1979). However, due to space constraints, we had to be quite selective in our coverage in this chapter. Therefore, our focus is limited to two topics: (1) personal characteristics of children that have been viewed as most relevant to egoistic versus altruistic responding (i.e., level of empathy, role-taking capabilities, and moral reasoning), and (2) socialization antecedents and correlates of prosocial responding. Even within these areas, our review is brief; however, relevant references are cited for those who desire more information.

Personal Characteristics Associated with Nonprosocial versus Prosocial Responding

Over the years, investigators have examined the relation of a variety of personal characteristics to prosocial responding. Three of these seem to be the most linked conceptually to altruism, and therefore have received greater theoretical and empirical attention than have other personal variables. Consequently, these three were chosen for our dicussion.

Role Taking

Psychologists frequently have suggested that the ability to make inferences about another's cognitive or affective state plays an important role in the development and elicitation of prosocial behavior (e. g., Batson, 1987; Hoffman, 1984; Krebs & Russell, 1981; Staub, 1979). This is because young children (or adults) with limited role-taking capabilities should be less likely than persons with more sophisticated abilities to infer that another is in need or in distress. Consequently, they would be less likely to attend to the other's need or to assist.

Despite the prominent role that perspective taking has been accorded in models of altruism, the empirical relation between the two has been mixed in the literature (e.g., Denham, 1986; Eisenberg, 1986; Krebs & Russell, 1981; Underwood & Moore, 1982). No doubt this is due, in part, to methodological factors (e. g., single or inappropriate indices of role taking) and to conceptual factors (e. g., examining the relation of role taking to prosocial behavior in situations not requiring role taking) (Eisenberg, 1986; Krebs & Russell, 1981). Nonetheless, in a meta-analytic review of the relevant data, Underwood and Moore (1982) did find a significant, positive relation between role-taking capabilities and prosocial responding. Thus, there does seem to be a relation such that low levels of role taking are related to low levels of prosocial behavior. This relation would undoubtedly be stronger if indices of role taking and prosocial behavior were always valid and reliable, if role taking and prosocial behavior were more frequently assessed in the same situation, and if age- and content-appropriate measures had been used in all studies.

Empathy

Both philosophers (e.g., Blum, 1980) and psychologists (e.g., Batson, 1987; Eisenberg, 1986; Hoffman, 1984) frequently have asserted that altruistic behavior is positively related to the individual's level of empathic responding (i.e., responding to another's emotional state or condition with an emotional reaction congruent with that of the other) or sympathetic responding (i.e., responding to another's emotion or condition with other-oriented concern). The assumption has been that individuals who emotionally experience another's need or distress will be more motivated to alleviate the other's distress than would other persons.

As for the data concerning role taking, the empirical research has not always supported the aforementioned theoretical assertion (Eisenberg, 1986; Underwood & Moore, 1982). However, according to a recent meta-analysis (Eisenberg & Miller, 1987), there is a significant positive relation between prosocial behavior and level of empathy, at least as it is assessed with most indices of empathy. The primary exception is for the picture-story indices of empathy that are used with children; with these indices, children are presented with hypothetical story situations and are assumed to experience empathy if they report experiencing the same emotion as the story protagonist. Such indices have been criticized for a variety of reasons (see Eisenberg & Lennon, 1983; Hoffman, 1982), and appear to be less valid than are many other measures of empathy.

The positive relation between empathy and altruism seems to be evident by 1 to 2 years of age (Buchsbaum, 1985; Radke-Yarrow *et al.*, 1983), at

least to some degree, and perhaps is even stronger by adulthood (Eisenberg & Miller, 1987; Underwood & Moore, 1982). Thus, it seems reasonable to conclude that egoistic, nonprosocial responding frequently may be due, at least in part, to the lack of empathic responsiveness, especially in contexts in which an other-orientation is likely to be heightened by attention and response to empathy-evoking cues.

Moral Reasoning

Cognitive-developmental theorists, building on the early work of Piaget (1965), have argued forcefully that the level of one's moral cognitions is related to the quality and the quantity of one's moral behavior (see Blasi, 1980; Eisenberg, 1986, 1987; Kohlberg & Candee, 1984). Such a relation has been found for some domains of moral functioning, including delinquency and prosocial behavior (see Blasi, 1980; Eisenberg, 1986; Underwood & Moore, 1982). With regard to egoistic versus altruistic responding, investigators frequently have found that children who reason at developmentally advanced levels, which tend to reflect more other-oriented concerns and moral principles, are more likely to exhibit prosocial behaviors than children who reason at lower levels. The relation of other-oriented behavior to other-oriented modes of moral reasoning (i.e., those reflecting role taking or sympathy) seems especially clear (see Eisenberg, 1986). Moreover, children who exhibit higher quality (i.e., more altruistic) prosocial behavior provide higher level moral justifications for their actions than children who assist for less altruistic reasons (see Bar-Tal, 1982, Eisenberg, 1986). Thus, it appears that individuals who reason in ways that reflect moral concerns also are likely to act in ways that reflect on other-orientation rather than egoistic concerns. However, it is likely that moral reasoning is related to prosocial behavior only in situations in which there is a moral conflict (i.e., in situations in which moral concerns are relevant; Eisenberg & Shell, 1986). In many circumstances, the performance of nonprosocial versus prosocial behavior may be performed without much conscious processing. In such situations, well-learned patterns of behavior, situational cues, or a variety of personal preferences and needs may be more relevant than the individual's level of moral reasoning.

Summary

Thus, there is empirical support for the notion that egoistic versus other-oriented patterns of behavior are related to the child's capabilities with regard to (or his or her tendency to utilize) role taking, empathy and sympathy, and moral reasoning. Nonetheless, these three aspects of personal functioning are not relevant to behavior in all circumstances. Moreover, it is likely that the child's role-taking skills, empathy/sympathy, and moral reasoning are all influenced by other factors, including prior socialization experiences. We now turn to the issue of socialization.

Socialization of Prosocial Behavior

There are a variety of socialization procedures that have been examined in relation to prosocial behavior. Some of the more important of these are reviewed in the remainder of this chapter. By necessity, this review is very general in content and therefore oversimplifies some patterns of findings. However, it does reflect the overall pattern of findings and can be used to identify areas of particular importance with regard to the socialization of self- versus other-oriented behavior and motivation.

Discipline

Inductions

A common occasion for the teaching of prosocial responses is parents' responses to their children's acts of transgression or inappropriate social responding toward others. It is here that socializing agents have the opportunity to preach, instruct, inform, and reason with the child about their behavior. In the disciplinary context, such reasoning and verbal exhortations generally have been labeled as "inductions" (Hoffman, 1970).

Consistent with earlier reviews (Moore & Eisenberg, 1984; Radke-Yarrow *et al.*, 1983) and research (e.g., Baumrind, 1971; Zahn-Waxler, Radke-Yarrow, & King, 1979), in recent studies researchers have found that inductions relate positively, but not entirely consistently, with prosocial responding (e.g., Abelman, 1985; Dolan, 1983; Mullis, Smith, & Vollmers, 1983). Moreover, there is some evidence that inductive approaches also reduce children's dispositions to act antisocially (Abelman, 1985).

The effectiveness of inductions appears to begin early, by 1.5 to 2.5 years of age (Zahn-Waxler *et al.*, 1979). Moreover, their effect on prosocial behavior appears to be enhanced by several factors, including parents' infrequent use of power-assert-

ive tactics (Abelman, 1985; Hoffman, 1963), high use of emotional expressiveness during discipline encounters (Zahn-Waxler *et al.*, 1979), and prior use of such techniques (e. g., Dlugokinski & Firestone, 1974). In addition, some types of reasoning appear to be more positively related to children's altruistic responding than are other forms (e. g., other-oriented inductive messages that call attention to the consequences of the child's actions on the behavior and feelings of others, and statements of principles and moral prohibitions about the child's behavior toward others, Hoffman, 1970; Zahn-Waxler *et al.*, 1979). Personal characteristics of children, such as general responsiveness (Keller & Bell, 1979) and perceived temperament (Simonds & Simonds, 1981), also may mitigate parental use of inductive forms of reasoning and thereby affect children's opportunity to learn about and acquire appropriate forms of prosocial responses to others.

Researchers have identified numerous aspects of the inductive-rearing context that may promote (or perhaps in their absence hinder) children's acquisition of prosocial behaviors (Dienstbier, Hillman, Lehnhoff, Hillman, & Valkenaar, 1975; Hoffman, 1970, 1983; Staub, 1979; Zahn-Waxler *et al.*, 1979). First, by focusing the child's attention on the consequences that his or her inappropriate behavior has on others' feelings and behavior, the child may learn to take the role of others and to empathize with their feelings and needs. Second, children may learn to make causal attributions about their behavior with regard to others' needs and feelings. Third, inductive messages generally are given within a supportive caretaking context. Therefore, although any given inductive message may be accompanied by a strong expression of parental emotion, the emotion is more likely to be interpreted by the child in terms of the value that the parent assigns to the situation, rather than indicating impending physical punishment or personal threat. Consequently, the child may be able and motivated to attend to information provided by the parent about the situation. Fourth, with inductions, children should be more likely to attribute the cause of their own negative arousal to their transgression (rather than to the threat of punishment or the socializer), and thus develop internally versus externally based motivations for behavior. In addition, when inductions are accompanied by statements regarding socializers' expectations and instructions for reparation, children learn not only that they are responsible for the consequences of their behavior, but also are provided with the means to act responsibly in the present as well as in future interactions. Finally, socializing agents who employ inductive reasoning are more likely to model a calm, nonpunitive approach to negotiating social interaction, one which the child can emulate in other social situations.

Power Assertion

Power-assertive tactics (compliance induced by actual or threatened force, punishment, withdrawal of privileges, and the like) either tend to be associated with lower levels of prosocial behavior (Abelman, 1985; Dlugokinski & Firestone, 1974; Roe, 1980; Turner & Harris, 1984; Moore & Eisenberg, 1984), or are unrelated to children's prosocial development (e. g., Feshbach, 1975; Zahn-Waxler *et al.*, 1979). Only Hoffman & Saltzstein (1967) have reported a positive relation between parental use of power-assertive techniques and prosocial behavior, and that was for boys and not for girls. Moreover, in experimental contexts, although children tend to act in a prosocial manner to avoid punishment (e. g., Hartmann *et al.*, 1976; Morris, Marshall, & Miller, 1973), this effect dissipates once the threat of punishment is removed (Hartmann *et al.*, 1976). Conversely, punishment for prosocial behavior tends to inhibit subsequent prosocial behavior, even if children have just observed an altruistic model (Rushton & Teachman, 1978).

In part, the somewhat inconsistent findings in the literature concerning power-assertion techniques may be because different aspects of negative rearing practices are included in researchers' measures of power assertion, for example, love withdrawal (e.g., Abelman, 1985) or some combination of controlling, demanding, rejecting, and/or restrictive rearing practices (e. g., Feshbach, 1975; Turner & Harris, 1984). More importantly, whether power-assertive tactics hinder children's prosocial development appears related to the presence of other practices within the overall rearing environment. For example, Baumrind (1971, 1986) reported that parents who provided a nurturant, responsive childrearing development, yet maintained high demands for compliance to expectations and occasionally used power-assertive techniques, tended to rear socially responsible boys. However, when parental demands were administered in a punitive, authoritarian context, boys exhibited less socially responsible behavior (Baumrind, 1971). Similarly, children's prosocial and caring responses toward others appear unaffected by moth-

ers' use of physical punishment if they occur in an overall positive parent–child relationship (Roe, 1980).

Children's prosocial development may be hindered within a predominantly power-assertive rearing context for several reasons (Dienstbier *et al.*, 1975; Hoffman, 1970, 1983; Moore & Eisenberg, 1984). First, because of the threat inherent in power-assertive discipline, children may focus their attention on the impending consequences of their behavior for themselves, rather than on the consequences for others. Second, in this situation, control over the child is exerted by actual or implied force, thus creating an external motivation for compliance. Moreover, children learn to attribute the source of their negative arousal to the punitive behavior of the parent rather than to their own transgressions or internal responses, and to make external self-attributions for their behavior (Dix & Grusec, 1983; Smith, Gelfand, Hartmann, & Partlow, 1979). Third, the high arousal resulting from such discipline should make it difficult for the child to attend to the information given by the parent, including any accompanying inductive components. Fourth, by using power-assertive techniques, the parent may model hostile, punitive, and threatening behavior and thereby communicate that such behavior is an acceptable mode of response to others. Although little is known about the intergenerational transmission of such practices, the issue becomes especially salient if, as suggested by Wolfe, Katell and Drabman (1982), children as early as preschool already are beginning to choose disciplinary consequences for others that match parental practices.

Other Nondisciplinary Socialization Procedures

Modeling

Consistent with a social learning perspective, researchers frequently have examined the role of selfish versus altruistic modeling on children's subsequent behavior. In general, exposure to prosocial models has been associated with enhanced prosocial responding, whereas exposure to stingy models appears to disinhibit selfish behavior (e.g., Lipscomb, McAllister, & Bregman, 1985; cf. Moore & Eisenberg, 1984; Radke-Yarrow *et al.*, 1983). The effects of modeling have been shown to persist over time (e.g., Rice & Grusec, 1975; Rushton, 1975) and to generalize in some situations (e.g., Rushton, 1975; Moore & Eisenberg, 1984). Moreover, televised or videotaped models, as well as real-life models, have been found to influence prosocial behavior (Rushton, 1979).

Despite the overall effectiveness of modeling procedures, it is clear that modeling is not always effective in promoting prosocial behavior (cf. Radke-Yarrow *et al.*, 1983). Children seem more likely to imitate the prosocial actions of some persons rather than others, for example, powerful or competent others (e. g., Eisenberg-Berg & Geisheker, 1979; Grusec, 1971). Nonetheless, it is clear the modeling is an important process by which children learn altruistic as well as selfish behavior.

Moral Exhortations

Persons in the child's environment not only enact prosocial behaviors for children to imitate, but also may symbolically (i. e., verbally) enact or promote an egoistic versus an altruistic course of action. For example, socializers may assert that they themselves plan to act in a given manner, or may discuss the merits of a given course of action. Researchers have found that such preachings can affect children's subsequent prosocial behavior, but that their effectiveness varies as a function of content. In general, children's sharing seems to be enhanced by preachings that either provide symbolic modeling, or are likely to evoke role taking and a sympathetic response (e. g., Burleson & Fennelly, 1981; Eisenberg-Berg & Geisheker, 1979; see Moore & Eisenberg, 1984). In contrast, appeals to norms regarding helping or preachings that include the threat of disapproval or that refer to self-oriented reasons for sharing are less effective (e. g., Burleson & Fennelly, 1981; Perry, Bussey, & Freiberg, 1981). These findings are, in general, consistent with the previously discussed research concerning disciplinary practices.

Direct Instructions and the Assignment of Responsibility

Socializers' use of verbal prompts, instructions, or commands to induce subsequent prosocial behaviors also is positively related to prosocial behavior, whereas instructions to behave selfishly are not (e.g., Dressel & Midlarsky, 1978; Weissbrod, 1980; see Moore & Eisenberg, 1984). The effects of instructions have been found for subsequent private as well as public sharing and have been found to persist over weeks (see Moore & Eisenberg, 1984).

The positive effect of assigning children tasks involving helping others has been noted in studies in Western as well as in non-Western cultures (e.g., Staub, 1979; Whiting & Whiting, 1975). It is likely that the aforementioned procedures promote prosocial responding by (1) inducing children to view themselves as prosocial individuals, (2) providing opportunities for reinforcement for induced helping, (3) providing opportunities to role take and learn about others' feelings and perspectives, and (4) providing the chance to learn new prosocial activities that can be repeated subsequently.

Prosocial Attributions

Another way in which socializers' verbalizations can affect children's prosocial tendencies is by serving to modify children's self-attributions regarding their own previous actions. In general, elementary school children are more likely to respond to subsequent appeals for assistance if they have been told that a former, initial prosocial action was due to internal factors (e.g., their helpful nature) than if their behavior has been attributed to external factors (e.g., an adult's expectations) or no attribution has been provided (Grusec, Kuczynski, Rushton, & Simutis, 1978; Moore & Eisenberg, 1984). However, the use of attributions does not seem to be effective until children are approximately 7 to 8 years of age (Eisenberg, Cialdini, McCreath, & Shell, 1987; Grusec & Redler, 1980). It is likely that this is because children do not understand the concept of stability in personality until they reach age 7 to 8 years; therefore, young children should not be motivated to behave in ways consistent with their prior behavior. However, by age 7 to 8, children understand that behavior is a reflection of enduring personality traits, and socializers' comments regarding children's motives for their behavior may alter children's perceptions of their own personality.

Warmth and Supportiveness

Findings concerning the relation of socializers' warmth to children's prosocial behavior and the lack thereof are mixed (Moore & Eisenberg, 1984; Radke-Yarrow *et al.*, 1983). Overall, however, noncontingent warmth seems to be associated with the child behaving as he or she pleases (which may or may not involve selfish behavior, depending on the circumstances), whereas warmth and support in natural settings (which seldom are noncontingent) may be slightly positively associated with prosocial responding (e.g., Bryant & Crockenberg, 1980; Zahn-Waxler *et al.*, 1979). Most likely it is not warmth in itself that enhances an other-orientation; rather, children may be more positively disposed toward supportive, nurturant socializers than toward colder socializers and, therefore, generally receptive to their influence, including disciplinary and other aspects of child-rearing practices (Hoffman, 1970). Moreover, it is likely that any association between socializers' nurturance and children's level of prosocial responding is moderated by other socialization practices. For example, Baumrind (1971) found that sons of both permissive parents (who were warm but low in control and did not set limits) and authoritarian parents (who were somewhat low in nurturance and controlling) were lower in social responsibility than were sons of authoritative parents (who exhibited warmth as well as some clear standards and enforcement of rules). In primary school, authoritative parenting was still positively related to sons' social responsibility, whereas authoritarian parenting was not (Baumrind, 1986).

In summary, the relation of socializers' relative nurturance and support to children's other-oriented behavior is not entirely clear. However, nurturance may enhance the effectiveness of other child-rearing practices and may contribute to the child's developing a positive orientation toward others generally and toward socializers specifically (Hoffman, 1970; Staub, 1979). In contrast, coldness and rejection are likely to undermine the effectiveness of otherwise constructive childrearing practices.

Summary

There is considerable research concerning the development of prosocial behavior; from this work we can draw some tentative conclusions regarding the lack of development of concern for others and the development of an egoistic orientation. Clearly, even young children exhibit the capacity for egoistic and for other-oriented responding (Radke-Yarrow *et al.*, 1983; Zahn-Waxler *et al.*, 1979), although children's motives for assisting others seem to become less self-oriented with age (Bar-Tal, 1982; Eisenberg, 1986). Children who exhibit high levels of role taking, empathy and sympathy, and moral reasoning appear to be more likely than children who are low in these capabilities to exhibit prosocial behavior, although the motives for such behavior no doubt sometimes are self-serving.

Moreover, certain child-rearing practices (and configurations of child-rearing practices) have been associated with the performance of varying levels of prosocial behavior. It is likely that the various child-rearing practices that are associated with other-oriented, helpful behaviors foster prosocial actions by means of a variety of mechanisms. These probably include the following: (1) enhancing children's levels of empathy, role taking, and moral reasoning (e. g., for inductions and preachings); (2) providing children with information regarding the appropriateness and desirability of various courses of action (e.g., modeling, provision of directives and preachings); (3) inducing a prosocial self-image in the child (e. g., provision of altruistic self-attributions, inducing of initial prosocial action); and (4) teaching children about the consequences of their behavior for self and other (e. g., by means of inductions, preachings, modeling, and the inducement of prosocial behavior). On the other hand, the consistent use of power-assertive practices may inhibit positive development not only by failing to provide many of the aforementioned positive outcomes, but also by inducing a negative orientation toward others and an externally based system of morality. In addition, socializers who use relatively high levels of power-assertive techniques provide an aggressive, uncaring model that may disinhibit children's negative tendencies and teach undesirable behaviors (Hoffman, 1970). More information is needed regarding the configurations of child-rearing practices that are most likely to enhance or undermine prosocial development and the role of cultural, institutional, and nonfamilial influences on prosocial development and the development of egoistic versus other types of motives.

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PART III

Overcontrolled Disorders

CHAPTER 15

Anxiety in Children
Nature and Development

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Although the study and treatment of adult anxiety have traditionally received a great deal of theoretical and empirical attention in psychology and in psychiatry, the topic of childhood anxiety has tended to be overlooked (Wolfson, Fields, & Rose, 1987). Yet children with anxiety disorders comprise a significant portion of patients treated at mental health centers (Cytryn, McKnew, Zahn-Waxler, Radke-Yarrow, Gaensbauer, Harmon, & Lamour, 1984). The belief that anxiety in childhood is common and transient appears to have undermined interest in exploring its nature and development (Wolfson *et al.*, 1987). Even if this were so, the distress produced when anxiety is manifest may well warrant attention (Jenkins, Bax, & Hart, 1980). Moreover, there is evidence that childhood anxiety may be predictive of pathology at later development stages of an individual's life (Bowlby, 1973; Kellam, Branch, Agrawal, & Ensminger, 1975). In this chapter, we explore childhood anxiety disorders from a developmental perspective and provide an integrative overview of diagnosis, assessment, epidemiology, evolution, and theory.

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Diagnosis

Although anxiety is associated with many disorders in children and in adults, the revised *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III-R) (American Psychiatric Association, 1987) describes three childhood disorders for which anxiety is the predominant feature: these are Separation Anxiety Disorder, Avoidant Disorder of Childhood or Adolescence, and Overanxious Disorder.

Separation Anxiety Disorder is defined as excessive anxiety over separation from those to whom the child is attached. This anxiety is evidenced by such features as unrealistic and persistent worry about possible harm befalling attachment figures or about some calamitous event separating the child from loved ones; reluctance or refusal to go to school, to sleep without attachment figures, or to be alone; recurrent nightmares with separation themes; and excessive distress or physical symptoms when separated or in anticipation of separation. Symptoms must be present for at least a 2-week period. The prevalence of Separation Anxiety Disorder is not uncommon, and males and females are equally represented. Nearly all children appear to exhibit some degree of separation anxiety, especially in early childhood. This pervasiveness creates certain

diagnostic and operational difficulties. Because the DSM-III-R classification does not provide clear guidelines for "normal" separation anxiety at any given developmental stage, it is left to the judgment of the individual diagnostician to distinguish excessive from normal levels.

Several laboratory studies have explored the factors that influence infants' reaction to separation (Campbell, 1986). Even though nearly all this research has employed nondisordered children as subjects, it does provide valuable information concerning normative developmental separation anxiety. For example, although children younger than 6 months display distress when left alone, they protest the departure of any attending individual as well as the departure of their mother (Bowlby, 1973; Stayton, Ainsworth, & Main, 1973). By the age of 8 months, separation anxiety becomes more specifically elicited by the departure of the infant's mother or primary caregiver. Around 12 months, as the child becomes mobile, the infant becomes more likely to follow the departing mother than to protest by crying (Stayton *et al.*, 1973). Insecurely attached infants, whose mothers appear to have been either unresponsive or unpredictable in their responses, show greater distress upon separation than infants who have experienced more responsive, or more predictable, maternal behavior (Bowlby, 1973; Campbell, 1986). These insecurely attached infants also exhibit less active responses, such as following or searching for their mother, and display less positive behavior upon the mother's return (Stayton & Ainsworth, 1973). Recent, innovative research has shown that children are more securely attached at one year of age when maternal responses in infancy have been consistent, accurate, and appropriate (Isabella, Belsky, & von Eye, 1989).

Weinraub and Lewis (1977) explicitly manipulated the mother–child interaction immediately prior to departure. One group of mothers was instructed to exit the room with no verbal warning or explanation. A second group was told to inform the child that they were leaving. The third group was instructed to provide verbal instructions about how to spend the time when the child was alone. Infants whose mothers left without warning showed the greatest separation anxiety, whereas those given explicit instructions displayed the least. These data are consistent with the more general relationship of predictability to human stress, showing that anxiety and arousal are often decreased when external cues of danger and safety are predictable and when external distractors are available. (S. M. Miller, 1981).

Children voluntarily separating to explore among interesting toys have been found to demonstrate little or no distress (Rheingold & Eckerman, 1970). In contrast, forced separation can evoke considerable distress, even when the child can still see his or her mother (Goldberg & Lewis, 1969). This suggests that perceptions of control over the separation experience can reduce distress and facilitate adaptation, perhaps because the child feels that he or she can terminate the separation if it becomes too aversive (S. M. Miller, 1980). Interestingly, father–infant interactions appear to be more predictive of infant sociability with strangers than are mother–infant relations (Bridges, Connell, & Belsky, 1988). In addition, infant responses during maternal separation can be predicted by differences in frontal brain activity, and hence appear to be related to individual differences in temperament (Davidson & Fox, 1989; Fox & Davidson, 1987).

Thus, the data suggest that adverse separation experiences will be evident when the child is younger, is insecurely attached to the caregiver, and is exposed to unpredictable and uncontrollable separation conditions. Research should continue to address the parent–child interactional factors and the physiological and temperamental characteristics that best predict attachment, as well as affective responding, during and following separation. It would be useful to explore these factors in clinically disordered children and to extend the age range of subjects under study (see Chapter 16, in this volume).

The diagnostic criteria for *Avoidant Disorder of Childhood or Adolescence* include excessive shrinking from contact with unfamiliar people, despite desire for and generally satisfying relations with family members and familiar figures. This avoidance must be severe enough to disrupt social relations with peers and must persist for a period of at least 6 months. Children under the age of 2.5 years are excluded from this diagnosis. In the only epidemiologic study of DSM-III diagnoses for children (Anderson, Williams, McGee, & Silva,1987), no cases of avoidant disorder were found in the 792 11-year-olds who were evaluated. Not surprisingly, there is a salient lack of research on Avoidant Disorder in the psychological literature. What is needed are studies that systematically investigate the diagnostic validity, therapeutic responsiveness, course in and outcome of this disorder (see Chapter 17, in this volume, for a related discussion of temperament).

Overanxious Disorder is defined as excessive or unrealistic anxiety or worry, for a period of 6 months or longer, as indicated by the frequent oc-

currence of symptoms, such as excessive or unrealistic worry about future events, past behaviors, and competence; somatic complaints; marked self-consciousness; excessive need for reassurance; and feelings of tension or an inability to relax. Overanxious Disorder has recently been examined for diagnostic validity, and found to have both convergent and discriminant validity (Mattison, 1984; Mattison & Bagnato, 1987). Specifically, when compared to boys who were diagnosed dysthymic or with Attention Deficit Disorder with Hyperactivity (ADDH), 8- to 10-year-old boys with Overanxious Disorder showed a Schizoid or Anxious profile on the parent version of the Child Behavior Checklist (Achenbach & Edelbrock, 1983), and pathologically elevated scores on the Worry/Oversensitivity factor of the Revised Children's Manifest Anxiety Scale (Reynolds & Richmond, 1978). Thus, the overanxious children displayed a psychological profile that was consistent with their diagnosis and that distinguished them from children who were diagnosed with Dysthymia and with ADDH.

Summary

Overall, although there is some evidence available on the diagnostic validity of childhood anxiety syndromes (e.g., Overanxious Disorder), there has been little empirical investigation of these disorders. This is also true of the diagnoses that are described in the Anxiety Disorders section of the DSM-III-R, including Phobias, Posttraumatic Stress Disorder (see Chapter 20, in this volume), Agoraphobia, and Panic Disorder, as they relate to children. Although these disorders are not usually first seen in childhood or adolescence, it is possible for children and adolescents to meet their diagnostic criteria.

While such phenomena as fears and separation anxiety are common among children, many children who meet the DSM-III-R diagnostic criteria may never receive a diagnosis or undergo therapy. Several factors seem to determine whether and which children actually receive a diagnosis. First, severity of the target behavior appears to be of more diagnostic significance than its presence or absence. Second, timing of a given behavior must be considered. For example, fear of specific stimuli and separation anxiety may be of diagnostic significance when the child is beyond a particular age, yet may be considered developmentally appropriate before that age. Third, another factor has to do with family interactions, which may compensate for (or exacerbate) a child's fearful and avoidant behavior by the way in which social interactions and family activities are structured. The extent to which this occurs will determine how dysfunctional and disruptive the symptoms are to the child's life (Campbell, 1986).

Assessment of Childhood Anxiety Disorder

The most common goals of psychological assessment are diagnosis, treatment design, treatment evaluation, and research. Although conceptually each individual goal may best be met by a particular assessment route, in reality there is much overlap among them (Werry, 1986b). Several symptoms under the childhood-anxiety-disorder umbrella lend themselves to measurement. These include subjective symptoms (e.g., the experience of fear, dread, and panic); behavioral symptoms (e.g., motoric uneasiness, hypervigilance, screaming and crying, and compulsive and escape-avoidant behaviors, such as shyness or school refusal); cognitive symptoms (e.g., increased inattentiveness, distractibility,decreased performance in school, and impaired memory); and physiological symptoms (e.g., increased heart rate, blood pressure, and electrodermal activity) (Werry, 1986a). In addition, enuresis (Schaffer, 1973), recurrent, diffuse abdominal pain (Kolvin & Nicol, 1979), and tics (Werry, 1986a) have been associated with childhood anxiety disorder.

Methods of Assessment

Interviews

Unquestionably, the most popular assessment method is the clinical interview (Mash & Terdal, 1981; L. C. Miller, Barrett, & Hampe, 1974). No matter what the goals or methods of assessment, some form of interview usually occurs. The style of conducting the interview and the interpretation of the interview material vary, depending on the assessor's theoretical perspective (O'Leary & Turkewitz, 1978). Formats range from completely unstructured and open-ended (usually associated with analytically oriented assessments) to those that are highly structured and standardized (usually used by cognitive-behaviorally oriented assessors). The main advantage of the nonstructured interview is its flexibility in analyzing the range and composition

of a particular problem (Bierman & Schwartz, 1986). Given the lack of formality, it also can facilitate a sense of personal rapport with the child and/or parent (Kendall & Ronan, in press). Although this may be an important consideration in treatment-oriented assessment, the interview suffers from a lack of reliability and validity (Mash & Terdal, 1981; Werry, 1986b).

Although imposing structure on the interview can diminish its flexibility, structuring an interview can increase its reliability (Mash & Terdal, 1981). Further, because it is usually easier to administer, it can be more cost- and time-efficient than a non-structured interview (Wiens, 1983). One of the earliest attempts to standardize the clinical interview was by Rutter and Graham (1968; Graham & Rutter, 1968), for use in their classic Isle of Wight study (Rutter, Tizard, Yule, Graham, & Whitmore, 1976). These interviews, which were developed for parents and children, yielded only one general category of anxiety disorder. This single category was then lumped together with others, such as dysphoria, to constitute an even larger category called "emotional disturbance." This system displays the highest reliability and validity of measures currently available (Rutter & Schaffer, 1980; Rutter *et al.*, 1976). It would be useful to borrow on this technique to develop measures that would explore the separate effects of specific anxiety disorders (Werry, 1986b).

Currently, the trend has been toward standardized assessment instruments that allow the clinician to make a formal diagnosis, based on the psychiatric classification system (Mash & Terdal, 1981; Wells, 1981; Werry, 1978). Included in this type of interview are the Schedule for Affective Disorders and Schizophrenia for School-aged Children (KIDDIE-SADS) (Puig-Antich & Chambers, 1978), the Diagnostic Interview for Children and Adolescents (Herjanic & Reich, 1982), the Interview Schedule for Children (Kovacs, 1978, 1983), the Child Assessment Schedule (Hodges, McKnew, Cytryn, Stern, & Kline, 1982), and the Diagnostic Interview Schedule for Children (Costello, Edelbrock, Dulcan, & Kalas, 1984). Although these measures can be easily administered, they tend to be lengthy. For example, the child's portion of the Diagnostic Interview Schedule for Children takes an average of 45 minutes to complete, and the parent's portion takes an average of 70 minutes to complete (Costello, Edelbrock, & Costello, 1985). This factor of length is often impractical, and assessors can lose in time what they gain in reliability.

Symptom Checklists and Rating Scales

The second most popular means of assessing childhood anxiety is through the use of symptom checklists and rating scales. These instruments most often consist of judgments about the presence, severity, and/or frequency of symptoms of psychopathology, made by an involved adult or the child (Conners & Werry, 1979; Humphries & Ciminero, 1979; Mash & Terdal, 1981; Wells, 1981; Werry, 1978). The rater is required to average the rate of specified behaviors across time and situations. The major criticism of symptom checklists and rating scales is that the criteria are often vague (e.g., presence of fear), thus resulting in relatively low inter-rater reliability when more than one informant is used (Werry, 1986b). Although this may be due to the fact that individual raters are witnessing discrete runs of behavior (e.g., teacher vs. parent) (Werry, 1986b), most anxiety scales rely on child self-report measures (Barrios, Hartmann, & Shigetomi, 1981; Johnson & Melamed, 1979). The main advantage of these scales is that they are economical in terms of cost, effort, and time and display reduced variance because of the averaging techniques (Barrios *et al.*, 1981; Werry, 1986b).

One of the most widely employed instruments of this type is the Children's Manifest Anxiety Scale (CMAS) (Casteneda, McCandless, & Palermo, 1956), now revised (RCMAS) (Reynolds & Richmond, 1978). This scale was originally developed as a children's equivalent of the Manifest Anxiety Scale, which is used to assess general or chronic states of anxiety in adults (Taylor, 1951). The CMAS was modified to correct for its lengthy administration time, overly sophisticated reading level, and the inappropriateness of several items (Reynolds & Richmond, 1978). This resulted in the "What I Think and Feel" measure (RCMAS), which is a 37-item scale with 29 Anxiety and 9 Lie Scale items. A factor analysis of the RCMAS by Reynolds and Richmond (1979) uncovered three subscales of anxiety: physiological anxiety, worry and oversensitivity, and concentration anxiety (e.g., inattentiveness and proccupation), which is consistent with earlier work on the CMAS (Finch, Kendall, & Montgomery, 1974).

Findings from several studies show that internal consistency is in the mid to upper .80s (Reynolds & Richmond, 1978; Reynolds & Paget, 1983), and test–retest reliability ranges from .68 to over .90 (Reynolds & Paget, 1983). It correlates highly (.85) with the A-Trait scale of the State-Trait

Anxiety Inventory for Children (Spielberger, 1973), another generalized anxiety measure, showing good convergent validity. It tends to be unrelated to the A-State scale, which taps more transient and situationally dependent anxiety (Reynolds, 1980, 1982), and thereby also shows good discriminant validity. Extensive normative data have been established for American (Reynolds & Richmond, 1978; Reynolds & Paget, 1983), Mexican American (Argulewicz & Miller, 1984), Nigerian (Pela & Reynolds, 1982), and high IQ children (Scholwinski & Reynolds, 1985). Norms vary across locality (Richmond, Sukemune, Ohmoto, Kewamoto, & Hamazaki, 1984), suggesting a need for separate norms for different groups (Kendall & Ronan, in press).

Another measure, The State-Trait Inventory for Children (STAIC), was developed to distinguish transitory or situational aspects of anxiety from more enduring or traitlike anxiety (Spielberger, 1973). Internal consistency has been found to be reasonably good, with alpha coefficients of .78 or higher reported for both scales (Spielberger, 1973). Results further show split-half reliabilities of .89 for the A-State scale and .88 for the A-Trait scale (Finch, Montgomery, & Deardorff, 1974). However, test–retest reliability for both scales is dramatically lower: .63 for the A-State scale and .44 for the A-Trait scale over a 3 month period (Finch, Montgomery, & Deardorff, 1974). Although norms are provided (Spielberger, 1973), their generalizability across geographic regions has not been examined (Wells, 1981). Data validating the distinction between the two scales is at best mixed (Werry, 1986b). Even though some evidence indicates that the A-Trait scale is highly correlated with other measures of chronic, generalized anxiety (e.g., CMAS, RCMAS) than the A-State scale (Speilberger, 1973), other research has produced discrepant results (e.g., Finch *et al.*, 1975; Finch & Nelson, 1974; Montgomery & Finch, 1974).

The Fear Survey Schedule for Children (FSSC) provides information on responses to a wide range of objects and situations (Scherer & Nakamura, 1968). The FSSC is comprised of 80 items drawn from the Wolpe-Lang Fear Survey Schedule for adults (Wolpe & Lang, 1964). The scale groups fears into certain *a priori* categories, such as school, home, social, physical, animal, travel, classical phobia, and miscellaneous. The child rates his or her level of fear on each item according to a 5-point scale. Although the scale is widely used, little evidence exists on its psychometric properties (Ollendick, 1983).

A variant of this scale, the Revised Fear Schedule for Children (FSSC-R) (Ollendick, 1983), uses a 3-point rather than a 5-point response format for ease of administration. Psychometric data show good internal consistency, ranging from .92 to .95. Test–retest reliability has been shown to be stronger over a 1-week period (.82) than over a 3-month period (.55). The FSSC-R is positively correlated (.50) with the A-Trait scale of the STAIC (Ryall & Deitiker, 1979) and negatively correlated with self-concept and internal locus of control in girls (Ollendick, Matson, & Helsel, 1985). Girls tend to score significantly higher than boys. The scale discriminates normal from school phobic children on the basis of the total score (Ollendick, 1983), and factor analyses have yielded factors comparable to those found for the original FSSC (Ollendick, 1983; see also the Louisville Fear Survey Schedule, L. C. Miller, Barrett, Hampe, & Noble, 1972).

In addition to the above measures, several situation-specific scales are available for children, covering a variety of contexts. Among them are the Hospital Fears Rating Scale (Melamed & Siegel, 1975), the Snake Attitude Measure (Kornhaber & Schroeder, 1975), the Personal Report of Confidence as a Speaker (Paul, 1966), and the Test Anxiety Scale for Children (TASC) (Sarason, Davidson, Lighthall, Waite, & Ruebush, 1960). The TASC, a specialized version of the Generalized Anxiety Scale for Children (GASC) (Sarason *et al.*, 1960), is the most widely used of this type. It was designed to assess the effect of anxiety on test scores, primarily tests of achievement and intelligence. The TASC displays reasonable reliability and modest validity (Johnson & Melamed, 1979).

Behavioral Methods

Unlike symptom checklists and rating scales, which measure anxiety retrospectively, true behavioral methods of observation emphasize the assessment of anxiety as it actually occurs. Generally, the procedure is as follows: (1) target behaviors are defined, (2) eliciting stimuli and contingencies are defined, (3) an observer is chosen, (4) a setting for the observation is chosen, (5) a time schedule for observations is devised, and (6) target behaviors are then counted as they occur.

Among the most useful of the behavioral measures are the Behavioral Avoidance Tests (BAT),

which have been utilized since the early part of the century (Jersild & Holmes, 1935). In the prototypic BAT, the client/subject enters a room in which the feared object is overtly present and is asked to approach, touch, and to handle the feared stimulus (Lang & Lazovik, 1963). This procedure yields several objective behavioral measures of fear, such as proximity to the feared object, number of approach tasks completed, response latencies, and the time spent in the presence of the phobic stimulus. The BAT is quite simple and can be administered easily by a paraprofessional. Children's fear responses to objects, such as small animals, high buildings, water, medical procedures, darkness, solid foods, and separation, have all been assessed by the BAT method (Barrios *et al.*, 1981). A passive BAT, one in which the feared object is imagined or symbolized by a different object, can be administered in situations in which the approach of a feared stimulus is beyond physical limitations (e.g., clouds). Passive BAT performances have been shown to correlate highly with active BAT performances in children (Murphy & Bootzin, 1973).

Another frequently employed behavioral measure is the observational rating scale, which consists of observing the child in a contrived or natural setting and recording the occurrence or nonoccurrence of predetermined objective signs of anxiety, such as visual contact, physical proximity, stance, and facial expressions (Barrios *et al.*, 1981; Werry, 1986b). An adaptation of Paul's (1966) Timed Behavior Checklist, used with adults suffering from public-speaking anxiety, also has been widely applied to children. It has served as a model for the development of observational tasks to measure separation anxiety (Preschool Observation Scale of Anxiety: Glennon & Weisz, 1978), test anxiety (Teacher's Rating Scale: Sarason *et al.*, 1960), anxiety about an impending surgery (Observer Rating Scale of Anxiety: Melamed & Siegel, 1975), and dental anxiety (Behavior Profile Rating Scale: Melamed, Hawes, Heiby, & Glick, 1975; Melamed, Weinstein, Hawes, & Katin-Borland, 1975; Melamed, Yurcheson, Fleece, Hutcherson, & Hawes, 1978).

Although the majority of observational rating scales are useful for designing treatment protocols (Barrios *et al.*, 1981; Werry, 1986b), they also have a number of problems, including expectational biases, reactivity, lack of standardized formats and manuals, observer drift, cost of training and equipment, and a lack of reliability and validity (Kazdin, 1977; Kent & Foster, 1977; Morris & Kratochwill, 1983; Wildman & Erickson, 1977). For example, on the BAT, the number of steps or tasks given to the child may vary among assessors by as many as 25 (Barrios *et al.*, 1981). This is significant in light of the fact that fewer and easier steps have been found to result in increased approach behavior (Nawas, 1971). Thus, comparisons across studies and target objects can be difficult.

Physiological Measures

Only a few studies have employed measures of physiological responses to fear and anxiety. The most frequently measured indices are heart rate and electrodermal responses (Barrios *et al.*, 1981; Wells & Vitulano, 1984). For example, in an examination of children's responses to injections, Shapiro (1975) found that an increase in self-reported needle avoidance corresponded with increased heart rate on the day the children were to receive the injection. Conversely, the lower the preinjection heart rate, the less avoidance behavior (e.g., crying, screaming) was observed during needle penetration (see also Darley & Katz, 1973; Stricker & Howitt, 1965). However, increased heart rate may not be the best discriminator of anxiety. Tal and Mihlich (1976) found no differences in heart rate when children imagined a feared situation versus an anger-inducing situation. In addition, increases in heart rate do not necessarily correlate with other measures of childhood anxiety (Kutina & Fischer, 1977; Melamed *et al.*, 1978).

Several methods have been used to assess electrodermal activity. For example, Shapiro (1975) affixed tape bands to the child's index finger for 3 minutes, and the darkness of the fingerprint was reported on a 10-point scale. Although interrater reliability was extremely high for this procedure and skin responses did appear to increase with reported fear, the measure proved to be overly sensitive to nonemotional factors, such as ambient temperature. An alternative method is the Palmar Sweat Index (Melamed & Seigel, 1975; Melamed, Hawes, Heiby, & Glick, 1975; Melamed, Weinstein, Hawes, Katin, Borland, 1975), which entails the quantification of sweat gland activity by means of a plastic impression of the hand. This measure was significantly related to self-reported general fears and to self-reported specific fears, as reported on the Children's Fear Survey Schedule (Melamed *et al.*, 1978). In addition, it displays high interrater reliability (.90) (Melamed *et al.*, 1978) and reasonable test–retest reliability (.64 over 1-day retest and .60 over a 15-day interval) (Lore, 1966).

Unfortunately, inclusion of the physiological components of childhood anxiety requires multimeasure assessment, high levels of technical expertise, as well as sophisticated and complex equipment (Johnson & Lubin, 1972; Lacey & Lacey, 1967). This can entail substantial time, energy, and financial resources. In addition, many of the responses assessed are extremely sensitive to factors that can be difficult to control (Barrios *et al.*, 1981). For example, electrodermal responsivity is reactive to environmental variables, such as novel stimuli and ambient temperature (Raskin, 1973). Despite such difficulties, measures of physiological response are important and should be included in the assessment battery whenever possible (Kendall & Ronan, in press).

Summary

A multimethod measurement approach to childhood anxiety is strongly advocated by many researchers (Barrios *et al.*, 1981; Mash & Terdal, 1981; Werry, 1986b). To date, the majority of studies have tended to rely on semistructured interviews that are designed for the parent or, less often, the child. Such interviews are sometimes accompanied by one of the symptom checklists or rating scales. Behavioral measures tend to be used mainly for purposes of treatment design and as outcome measures to test specific behavioral interventions, such as systematic desensitization. Physiological measures are rarely obtained. Although each area of assessment has its strengths and weaknesses, a common problem is inadequate standardization and insufficient normative data. Future research in each of these areas will advance the overall assessment and understanding of childhood anxiety.

Epidemiology of Childhood Anxiety Disorders

In order to delineate developmentally normal behavior from pathological fear and anxiety, it is essential to define its upper and lower boundaries (Graziano & Mooney, 1984). As epidemiological studies can provide these boundaries; this section briefly evaluates and reviews the epidemiologic literature on childhood anxiety.

General Population-Based Evidence

To enhance the generalizability and clinical utility of epidemiologic findings, there is a need for research that explores the prevalence of "common" disorders in a general population, while rigorously adhering to uniform diagnostic criteria, such as the DSM-III (Achenbach & Edelbrock, 1984; Yule, 1981). One such study looked at prevalence rates for various disorders in a sample of 792 children, 11 years of age, from Dunedin, New Zealand (Anderson *et al.*, 1987). The disorders investigated included Attention Deficit Disorder, Conduct Disorder, Oppositional Disorder, Major Depression, Dysthymia, Separation Anxiety, Overanxious Disorder, Avoidant Disorder, Simple Phobia, Social Phobia, Panic Disorder, Obsessive-Compulsive Disorder, and Psychosis. The children were interviewed at age 11 by using the highly structured Diagnostic Interview Schedule for Children, Child Version. When the children were 5, 7, 9 and 11 years of age, a behavioral history for each child was obtained from a parent (usually the mother) and from a teacher.

The results yielded four levels of agreement, representing different degrees of certainty in identifying a case. Level 1 indicated diagnostic criteria met by more than one source, independently. Level 2 indicated diagnostic criteria met by one source and symptoms confirmed by one or both of the other sources. Level 3 indicated diagnostic criteria met by one source without confirming symptoms from either of the other sources. Level 4 indicated diagnostic criteria met only by combining different symptoms from all three sources, with the same symptom reported by several sources counted only once. Thus, criteria for a Level 1 or a Level 2 diagnosis would be considered more stringent and conservative than criteria for a Level 3 or a Level 4 diagnosis.

When all four levels were included in the analysis, results showed that 139 children had one or more DSM-III disorders, yielding an overall prevalence for disorder of 17.6%. This finding is comparable to the prevalence of emotional disorders found by Rutter *et al.* (1975), in an inner-London sample. When disorder was more conservatively defined as to include only Level 1 and Level 2 criteria, the overall prevalence dropped to 7.3%. The authors suggest that this may well be

> the more clinically relevant prevalence figure, representing those children whose behavior is disordered in a sufficient number of circumstances to bring them to adult attention and possibly clinical referral. (Anderson *et al.*, 1987, p. 74)

Indeed, parents of children with the highest percentage of Level 1 and Level 2 diagnoses (full diag-

nostic criteria or sufficient symptomatology met by at least one adult), reported significantly more incidences of seeking help for their child's behavioral or emotional problem than parents with children having a Level 3 or a Level 4 diagnosis.

Utilizing all four levels, Separation Anxiety was the most prevalent of the anxiety disorders and the third most prevalent disorder overall, diagnosed in 3.5% of the sample. Overanxious Disorder had a prevalence rate of 2.9% and was ranked fifth overall. Simple Phobias were prevalent in 2.4% of the population and were ranked sixth overall. Social Phobias were reported considerably less frequently, with a prevalence rate of 0.9%. No cases of Avoidant Disorder, Panic Disorder, or Obsessive-Compulsive Disorder were reported in this population. Interestingly, close to 50% of the cases of Separation Anxiety were diagnosed at the third level of agreement. All the diagnoses for Simple Phobia and Social Phobia were at Level 3. These findings illustrate how low the prevalence rates are when more rigorous criteria for case identification of anxiety disorder are used, that is, Levels 1 and 2 only.

Of the 219 cases identified, 45% occurred as a single disorder, whereas 55% occurred in combination with one or more other disorders. In virtually all the cases of multiple disorders, at least one anxiety disorder was present. Of all the disorders reported, Attention Deficit Disorder occurred most frequently as a single disorder, whereas Depression–Dysthymia represented the category with the most overlap. The ratio of boys to girls with one or more disorders was 1.7:1. This sex ratio for overall disorder is consistent with the findings of Rutter *et al.* (1975; Rutter, Tizard, & Whitmore, 1970), and Graham (1979). Significant gender differences were also reported for each anxiety disorder. Separation Anxiety displayed a male–female ratio of 0.4 : 1, Simple Phobia had a ratio of 0.6 : 1, and Social Phobia had a ratio of 0.2 : 1. Overanxious Disorder was the only anxiety disorder to display a greater prevalence in males, with a male–female ratio of 1.7 : 1.

Clinically Based Evidence

Few data exist on the prevalence and coherences of childhood anxiety disorders in clinical populations. In one study, the demographic characteristics and patterns of co-morbidity associated with Separation Anxiety Disorder (SAD) and Overanxious Disorder (OAD) were examined and compared in a clinical sample of children and adolescents, aged 5 to 18 (Last, Hersen, Kazdin, Finkestein, & Strauss, 1987). Children were given all the DSM-III anxiety and affective diagnoses for which they qualified without adherence to the DSM-III hierarchical system and diagnostic exclusionary rules, based on the Interview Schedule for Children. However, one guideline was imposed: If one disorder could be subsumed under another without causing additional adjustment problems, then the subsumed disorder was not diagnosed.

Based on the interviews, a total of 69 children (76%) met DSM-III criteria for either SAD (N = 22), OAD (N = 26), or both SAD/OAD (N = 21). The fact that approximately one third of the sample met criteria for both SAD and OAD parallels findings obtained with anxious adults, who often carry an additional diagnosis of an anxiety disorder (Barlow, Vermilyea, Blanchard, Vermilyea, DiNardo, & Cerny, 1985; Barlow, Blanchard, Vermilyea, Vermilyea, & DiNardo, 1986). Furthermore, prevalence rates and co-morbidity patterns for SAD and OAD paralleled those found for agoraphobia and generalized anxiety disorder, their respective adult equivalents (Barlow *et al.*, 1986; DiNardo, O'Brien, Barlow, Waddell, & Blanchard, 1983).

Children with SAD and SAD/OAD were significantly younger at intake than children diagnosed solely with OAD (mean ages: 9.1 for SAD, 9.6 for SAD/OAD, and 13.4 for OAD). There were no significant differences in sex distribution across the three groups, although there were many more females than males in the SAD and SAD/OAD groups. There was a high preponderance of white children in all three groups (SAD 86%; SAD/OAD 95%; and OAD 100%). Given that 35% of the children referred to and evaluated by the clinic are black, this appears to be a meaningful finding. The three groups also differed significantly with respect to social strata, as measured by the Hollingshead (1975) index. Families of SAD children received significantly lower social strata ratings than families of OAD children.

Almost all the children in each of the three groups received at least one additional diagnosis. Approximately one third of the cases in each of the three groups met DSM-III criteria for major depression. Between 15% and 24% of each group were concurrently diagnosed with an attention deficit disorder, and between 14% and 27% of each group presented with a concurrent oppositional disorder. None of the children in the SAD group and very few children in the OAD (7.7%) and the SAD/OAD (9.5%) groups were diagnosed with dysthmic disorder. Only two children received an additional diagnosis of conduct disorder, and no children met crite-

ria for bipolar cyclothymic disorders. However, more than half of the OAD group met criteria for at least one additional anxiety diagnosis, whereas only one child in the SAD group met such criteria. Specifically, the OAD group was significantly more likely to have an additional diagnosis of simple phobia than the SAD group, which had no cases of that disorder.

Summary

The prevalence of anxiety disorders in the general population appears to be low when strict diagnostic criteria are employed. In clinical samples, though SAD appears to be more prevalent than OAD at the prepubertal stage, there is a gradual reversal of prevalence dominance to OAD at or following puberty and a high percentage of dual diagnosis (SAD/OAD) during this transition phase. Thus, SAD may be a risk factor for the later development of OAD. The data also suggest an overall preponderance of females diagnosed with childhood anxiety disorder, with the exception of OAD, which appears to range from neutral to male gender-biased (see also Bauer, 1976; Houston, Fox, & Forbes, 1984; Ollendick *et al.*, 1985). Although it is possible that these results reflect differences between the genders, they may also be artifacts of reporting biases on the part of both parents and children (Bauer, 1976; Ollendick *et al.*, 1985). Finally, children diagnosed with an anxiety disorder appear likely to receive an additional diagnosis.

Evolution and Outcome of Fear and Anxiety

"Normal" Fear and Anxiety

Over 50 years ago, Jersild and Holmes (1935) found the average number of fears in preschool children to be 4.6 when reported by the mother. McFarlene, Allen, and Honzik (1954) reported at least one specific fear in 90% of the normal children they studied between the ages of 2 and 14. Based on parental reports, Lapouse and Monk (1959) reported an average of 11 fears in children between 6 and 12 years of age. Ollendick (1983) found a similar number of fears in children between 8 and 11 years of age, based on childrens' self-reports. These studies suggest that it is common for children to evidence multiple fears.

The frequency of early childhood fears and anxieties appears to be functionally related to the child's developing autonomy. In a recent longitudinal study of preschool children, Draper and James (1985) found that the quantity of fears seems to peak between 2.5 and 4 years of age and then tapers off somewhat. This peak corresponds with the beginning of the child's sense of individuation from parents and the world. The authors reported an inverted-U relationship between the number of fears reported by mothers and the age of the child. This evidence suggests that fears are relatively transient and that the quantity of fears declines with age and autonomy (see also Bauer, 1976; Lapouse & Monk, 1959).

Developmental patterns have been found in the content as well as in the frequency of children's fears and anxiety. Infants typically show avoidant reactions to purely environmental stimuli, such as loud noises (Jersild, 1954). As the child matures, the range of fears broadens to include imagined or specific objects, such as nightmares (Jersild & Holmes, 1935). By the early school years, fears become more internalized and directed toward future situations, such as school or physical well-being (Kennedy, 1965; Simon & Ward, 1974). Moral, religious, and sexual fears appear to take on an increasing importance during adolescence (L. C. Miller, 1983).

Recent findings suggest that there may be a core group of children's fears that is more stable across development. Ollendick *et al.* (1985) measured the frequency and intensity of fears in 126 children and adolescents, between 7 and 18 years of age, using the Revised Fear Survey Schedule for Children. To analyze the data, four age groups were created; 7–9, 10–12, 13–15, and 16–18. Reported intensity of fear did not differ across the groups. Additionally, 8 out of the 10 most common fears (including not being able to breath, a burglar breaking into the house, fire burns, getting hit by a car or truck, death or dead people, bombing attacks/being invaded, looking foolish, and getting poor grades) were shared by almost all the age groups.

Agras, Sylvester, and Oliveau (1969) randomly sampled 325 adults and children of the general population of Burlington, Vermont, interviewing directly respondents over the age of 14 and relying on the mothers' reports of behavior for the younger respondents. Based on items from the Fear Survey for Behavior Therapy and the Fear Scale, the interview schedule listed 40 commonly feared situations and contained questions as to the intensity and duration of fear and avoidance behavior, as well as attempts at treatment by the family or by clergy, physicians, and others. All phobic and possibly phobic subjects were identified based on the

number, type, and intensity of the reported fears. Each of these subjects, the child directly if over age 14 or, if younger, the adult respondent, was then interviewed by a psychiatrist to determine the diagnosis, the history and treatment of the disorder, and the degree of disability using the DSM-III. To minimize experimenter bias, a number of nonphobic individuals were added to this subsample.

These results yielded three categories of fears with differing patterns of onset and chronicity. One category consisted of fears of doctors, injections, darkness, and strangers. Generally, fears of this nature had their onset in early childhood, displayed a drastic reduction in prevalence with increasing age, and were often short-lived. The second category consisted of fears of animals, heights, storms, enclosed places, and social situations. The onset of these fears spanned childhood to early adulthood and appeared to have a much more moderate decline in prevalence with increasing age, indicating a more persistent course. The third category of fears included crowds, death, injury and illness, and separation. These fears displayed a later developmental onset, usually in adulthood, with the greatest prevalence in middle age. Overall, these results suggested a long-term, mildly disabling course for phobias.

"Abnormal" Fear and Anxiety

Relatively few longitudinal studies have explored the course and outcome of childhood anxiety disorders over time (Gittelman, 1986). One short-term study of the entire population of one school found that, of 95 children diagnosed as school phobic, half were frequently absent from school 1 year after diagnosis (Ono, 1972). Although this study represents a rather short-term follow-up, the results suggest that school phobic children often continue to display difficulty with attendance.

Richman, Stevenson, and Graham (1982) found that fears at age 3 predicted the development of a "neurotic" disorder across a 5-year period. Fears at age 3, however, were not associated with higher incidence of all psychiatric disorders, only with "neurotic" disorders within the 5-year follow-up. In a study of children in the Isle of Wight, England, 11 year olds with "emotional" disorder, as measured by questionnaires and interviews with parents and teachers, were found to have twice the risk of psychiatric problems in adolescence as compared to other children (Rutter, Tizard, & Whitmore, 1981). As in the data of Richman and his colleagues, Rutter *et al.* (1976) found that "emotional" disorders were not associated with increased risk of all types of pathology, only later "emotional" disorders. Of the emotionally disordered 11 year olds, not one displayed conduct disorder over the following 4 years (Rutter, 1980).

Other research has explored the outcome and course of anxiety in children who were hospitalized for school phobia. For example, a 6-year follow-up of 67 children admitted for inpatient treatment of "neurotic" disorders revealed continued psychiatric disorder in 32% of the individuals (Warren, 1965). This group of patients included children, aged 11 to 19.5 years at admission, who had been diagnosed as obsessive-compulsive and phobic at intake. Sixteen of these children were admitted with school phobia. Seven, or 44%, of the school phobic children continued to show phobic symptomatology 6 years later, as measured by interviews and/or questionnaires. The nature of the later phobic difficulties was not specified. Of 15 patients who were diagnosed obsessive-compulsive, only 2 were free of obsessional symptoms at follow-up, 5 had severe symptoms; 4 had moderate symptoms; and a final 4 had mild symptoms that emerged under stress.

Berg, Butler, and Hall (1976) assessed the functioning of 100 "secondary school age" school phobic children, 3 years after discharge. They found that 50% of the children displayed continued problems with school attendance. Depending upon the criteria for dysfunction, 50% to 70% displayed other symptomatology (e.g., depression, schizoid personality disorders, schizophrenia), and 5% were diagnosed agoraphobic. Childrens' IQ was inversely correlated with quality of adjustment, with those with higher IQs showing poorer outcomes (see also Roberts, 1975). A follow-up of 14 hospitalized school phobic children, ranging in age from 8 to 16 at discharge, and from 18 to 23 at follow-up, found only one patient to be free of neurotic or personality problems (Weiss & Burke, 1970). Despite these continued difficulties, the sample displayed satisfactory overall functioning, as measured by interviews ($n = 9$) or telephone interview and questionnaire ($n = 5$). Boreham (1983) obtained questionnaire data from 80% ($n = 54$) of a sample of hospitalized school phobic children. Two years after discharge from treatment, 22% reported difficulty leaving home alone and for 6% of this sample the difficulty was severe. However, the sample showed minimal disturbance on measures of mood, school or work attendance, and interpersonal relations with friends.

Another group of studies has investigated the outcomes of school phobic children treated in an outpatient setting. A longitudinal comparison of 24 school phobic children, 18 "neurotic" children, and a small group of "normal" controls found no significant difference between the school phobic and the other neurotic children (Waldron, 1976). The phobic and neurotic children displayed a higher incidence of neuroses and personality disorders and poorer occupational and interpersonal adjustment than did the controls, as determined by semistructured follow-up interviews. However, refusal to attend school was the sole criterion distinguishing the school phobic from neurotic children. This may have resulted in a good deal of overlap between the diagnostic groups (school phobic and other neurotic), as they both exhibited avoidance of new situations, inhibition with respect to expressing aggression, and lives more centered around home of origin. Further, though none of the "normal" controls received a psychiatric diagnosis during the follow-up period, the expected population rate of psychiatric disorder was not represented in the comparison group (Gittelman, 1986).

One to seven years after outpatient treatment, irregular school attendance and moderate to severe maladjustment continued to be a problem for 30% of a sample of 41 school phobic children (Rodriguez, Rodriguez, & Eisenberg, 1959). Children younger than 11 years old at the study's outset displayed more improved outcomes than those 11 years or older. Except for three children diagnosed schizophrenic, psychiatric status at follow-up was not well defined. Nine years after outpatient treatment for school phobia, 60% of the 49 children followed were reported to display adjustment problems (Coolidge, Brodie, & Feeney, 1964). The children were aged 12 to 22 years, but were 4 to 11 at the reported onset of symptoms. In 20% of the sample, the problems were severe, varying in nature from psychosis to character disorders.

Baker and Wills (1979) evaluated occupational status at an average of 6 years after outpatient therapy, in 67 school phobic children. Although 20% of the children had never returned to school, 85% were either employed or enrolled in full-time school at the time of the follow-up. When compared to a general sample of similar sex and age, the school phobic individuals were only slightly worse on occupational and educational status. Whether or not the phobic individuals returned to school during the course of treatment did not relate to work outcomes.

Retrospective and Familial Evidence

The relationship between childhood anxiety and adult disorder is largely unclear, because no long-term prospective data are available. However, about 50% of adult patients with agoraphobia and panic disorder have childhood histories of fearfulness, dependency, separation anxiety, school adjustment difficulties, and phobia (Abe, 1972; Gittelman-Klein & Klein, 1973, 1985; Klein, 1964). For example, Berg, Marks, McGuire, and Lipsedge (1974) found higher rates of childhood school phobias in agoraphobics and in other neurotic patients. A comparison of anxiety-disordered and depressed adults with controls revealed higher rates of school avoidance among the depressed and the anxiety disordered (Tyrer & Tyrer, 1974). These data are consistent with retrospective studies of age of onset in adult patients. In a study of 62 adult patients treated for agoraphobia, Sheehan, Sheehan, and Minichiello (1981) found that 31% had an onset in their first decade, and 55% had an onset by age 20.

Some results suggest that there might be a familial relationship between childhood anxiety disorder and adult pathology (Turner, Beidel, & Costello, 1987). Fifty-nine children between the ages of 7 and 12 were assessed with a battery of self-report inventories and the Child Assessment Schedule (Hodges *et al.*, 1982). Sixteen of the subjects were offspring of a parent undergoing treatment of an anxiety disorder (agoraphobia and obsessive-compulsive disorder); 14 were offspring of a parent undergoing treatment for a dysthymic disorder; and 13 were offspring of a normal parent. Children of anxiety-disordered parents were more than twice as likely as children of dysthymic parents and more than 18 times as likely as children of controls to be diagnosed with any DSM-III disorder. Additionally, children of anxiety-disordered parents were twice as likely to be diagnosed with a DSM-III anxiety disorder than the offspring of dysthymic parents and more than 7 times as likely to reach criteria for such a diagnosis than the offspring of normal parents. Thus, these data suggest that the offspring of parents with an anxiety disorder are at greater risk for developing an anxiety disorder themselves.

Summary

Taken together, the evidence suggests that earlier views regarding the transient nature of fears

may need to be reevaluated. Although the broad contents of fears may alter with different stages of development, the actual intensity of fears may not decline significantly. Indeed, several categories of fears may remain constant throughout the developmental span.

Based on follow-up studies of hospitalized school phobics, 22% to 100% of the samples were found to display anxiety 2 to 18 years after discharge from treatment. Among outpatient school phobic children, 30% to 60% continued to experience difficulties and this rate of problems may be worse than for nondisordered individuals. Even though continued psychiatric and scholastic difficulties appear to be common for school phobic individuals, the waters are muddied by the use of nonspecific, nonoperationalized diagnostic descriptions (e.g., higher rate of "neurotic" disorders) and differing criteria for outcome measures. Further, much of the evidence is drawn from studies of inpatients, who are extremely rare and probably represent a distinct (and typically more severe) subpopulation. To date, attempts to identify predictors of later functioning (such as age of onset, successful short-term therapy outcome, and return to school during therapy) have all been inconclusive (Gittelman, 1986).

Finally, the retrospective "follow-back" studies suggest that childhood anxiety disorders do increase the risk of later psychiatric difficulties. Although there is some suggestion of a particular link between separation anxiety and later agoraphobia and panic disorders, the results are in no way definitive and further investigation is required (Gittelman, 1986). Studies of familial transmission of anxiety disorder, in turn, suggest that the presence of adult anxiety disorders increases the risk for anxiety disorder in the children of these adults. It remains for future research to delineate the exact nature of the relationship between specific childhood anxiety disorders and specific adult diagnoses, using large-scale, long-term longitudinal studies of discrete diagnostic groups.

Theories of Etiology

The causes of anxiety and the contribution of developmental factors have been viewed very differently by the three major clinically-based theories of etiology: psychoanalytic, behavioral, and emotional-processing theory. Psychoanalytic perspectives attribute the cause of anxiety to unsuccessful defenses against instinctual and unconscious impulses. Id impulses, primarily sexual and/or aggressive, must be negotiated to meet environmental and social demands by the ego. Anxiety results when the ego defenses are insufficient to prevent direct expression of these instinctual impulses, and the individual encounters the prohibited urges and desires. The case of Little Hans is a classic example. He was a 5-year-old boy who exhibited great fear and anxiety in the presence of horses, presumably resulting from a castration fear related to unresolved Oedipal conflicts. This conceptualization posits quite different implications for anxiety across psychosexual developmental stages, as the various intrapsychic conflicts are addressed and/or resolved.

Behavioral perspectives postulate that anxiety is a classically conditioned response to specific stimulus conditions. Environmental cues that are temporally associated with an unconditioned state of fear become capable of eliciting the same unpleasant arousal. A two-stage model predicts that this respondently conditioned arousal is operantly maintained by escape and avoidance behaviors. That is, escape or avoidance of the aversive situation reduces the unpleasant arousal before habituation occurs, and the escape/avoidance response is therefore negatively reinforced and maintained (Mowrer, 1960). According to this view, acquisition and maintenance of anxiety should operate independently of age. The only developmentally relevant difference would be that older individuals may have a more extensive learning history of anxious responses, resulting in more stable and pronounced fear behavior. Conversely, individuals with a longer history of nonanxious responses would be more resistant to the development of fear behavior.

Recent research with agoraphobic and obsessive-compulsive adults has led to the development of an emotional-processing theory of fear and anxiety (Foa & Kozak, 1986). This perspective, based on empirical research with exposure treatments (Foa & Kozak, 1985), states that a memory structure of fear serves as a program for escape or avoidance of the feared stimulus. This fear structure includes: (1) information about the feared stimulus situation; (2) information about verbal, physiological, and overt behavioral responses; and (3) interpretive information about the meaning of the stimulus and response elements of the structure (Foa & Kozak, 1986). Emotional processing occurs as new information is assimilated into the fear structure, resulting in either increased or decreased fear

responding. In order for emotional processing to reduce fear and anxiety effectively, two conditions must be met: (1) the fear memory must be activated by fear-relevant information, and (2) the new fear-relevant information must involve elements that are incompatible with some elements in the existing fear structure. That is, appropriate emotional processing (such as that which occurs in exposure therapies) transforms a program for anxious responding, avoidance, and escape into a new memory structure that mediates less emotional responding.

To date, the work of Foa and Kozak has focused on assessing and modifying fear structures, rather than on clarifying the development of these structures (for a review of this research, see Foa & Kozak, 1986). No research has been conducted on either the development or modification of fear structures in children and adolescents. However, emotional processing may well be affected by developmental differences in childrens' perception of threat (Kozak, personal communication), capacity for self-observation, attributional processes, as well as other perceptual and cognitive functions.

Conclusions

The major clinically-based theories posit very different implications for anxiety and fear across development. Although traditional behavioral theories do not consider developmental influences on the etiology of anxiety, the psychoanalytic view and the more current emotional-processing perspective do allow for a developmental approach. To date, however, there has been no systematic, empirical investigation of developmental factors in line with these theoretical views and in conjunction with developmentally based theories such as those relating to temperament and attachment.

Taken together, much research remains to be done on the nature, determinants and development of clinical anxiety disorders in childhood. Use of a multimethod measurement approach to the assessment of childhood anxiety would greatly facilitate this process. While the prevalence of anxiety disorders in the general child population is not high when strict diagnostic criteria are applied, early fears appear to endure over time and may predispose to later psychopathology. Further research on the etiology of childhood anxiety is clearly in order from a perspective that integrates standard models of psychopathology with developmental considerations.

ACKNOWLEDGMENTS

We are indebted to Denise Durbin, Farah Quintos, and Bill Shadel for their invaluable assistance. This chapter was partially supported by Temple University Research Incentive Fund and Temple University Grant-in-Aid of Research to the first author.

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CHAPTER 16

Separation Anxiety

Judith A. Crowell and Everett Waters

Separation anxiety refers to the negative affect in anticipation of or subsequent to departure or loss of an attachment figure. Attachment by definition implies a desire to be close to a particular individual; thus, separation from loved ones is expected to be distressing for infants and adults alike. The behavioral manifestations of separation anxiety have a distinctive developmental course of increase and then decline. Research with infants, children, and adults has identified marked individual differences in proneness to worry about anticipated separations, and in the degree and duration of negative affect when separation occurs. Central questions about separation anxiety concern (1) its typical course of development in infancy, (2) the mechanisms underlying developmental changes and individual differences in separation-related distress at different ages, and (3) the relationship between normative expressions of separation anxiety early in life, extraordinary separation experiences, and clinical phenomena in children and adults.

Development of Separation Responses

The developmental course of separation protest in human infants has been studied in detail in a variety of cultures. Reliable patterns of protest to the departure of a particular individual are rarely established prior to 6 months of age (Layton, Ainsworth, & Main, 1973). In most cultures, the proportion of children who cry following maternal departure in familiar settings reaches between 50% and 75% toward the middle of the second year of life, then declines at varying rates. By age 3, separation protests decline in most cultures so that only 20% to 40% of children cry in response to ordinary separations (Kagan, Kearsley, & Zelazo, 1978).

Cultural differences in the age of onset of separation protest are considerably narrower than differences in the rate of its decline. Ainsworth (1967), Konner (1972), and others have suggested that the onset of separation protest is somewhat earlier in cultures in which infants are in close physical contact with mothers for extended periods, are cared for almost exclusively by the mothers, and have relatively little exposure to strangers. There is also a tendency for early onset of separation protest within a culture to be associated with higher rates of separation protest and slower developmental decline. Interestingly, this is more evident in comparisons between cultures than in comparisons among families within a given culture.

Differences in procedures, test situations, number of assessments, and definitions of separation response make it difficult to construct a definitive developmental picture by compiling data from different studies. For example, the proportion of infants crying upon separation is greater in studies that examine a series of separations with each infant than in studies that examine a single test separation per infant. Familiarization with the observers, use of home versus nonhome observational settings, whether infants are restrained (e.g., in an infant seat or held by an adult) or free to follow, and the man-

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ner of the mother's departure can also influence the likelihood of observing separation protests at a given age. For example, most infants are much more likely to follow a departing attachment figure than to sit and cry. They are more likely to stop crying after a few minutes than to cry continuously and they are more likely to greet the adults' return happily than to restart the cry (Slayton *et al.*, 1973). These are important clues to the mechanisms underlying separation protest and to its function in the human behavioral repertoire.

Mechanisms and Functions of Separation Protest

Psychoanalytic Perspectives

In Freud's (1926/1961) view, both infant–adult and adult–adult attachments are based upon bonds of mental energy that are never voluntarily or completely withdrawn. Indeed, he argued that threats to these cathetic bonds are invariably experienced as painful and necessarily initiating self-protective intrapsychic responses (defense mechanisms). A corollary of this view is that use of defense mechanisms to cope with separation experiences early in life can influence the personality structure and the patterns of love relationships later in life.

Freud's formulation suggested a number of elaborations and alternative hypotheses, including the notion that separation anxiety is a reenactment of birth trauma (Rank), a reflection of ambivalence, anger, and guilt felt toward the mother (Klein), and the expression of a more general concern with fulfillment of basic needs (A. Freud). None of these has been as broadly influential as Freud's original formulation, which remains the inspiration for most speculation and research on relationships between early separation experience and later adjustment.

The primary limitations of Freud's and other psychodynamic interpretations of separation protest lie in the fact that they were primarily based on reconstructions of early events from clinical interviews with adults and were organized around outdated physiological models of motivation and gratification. Use of retrospective methods and reliance on clinical cases pose a significant risk of identifying separation-related experiences as antecedents of neurotic symptoms, when such experiences are common among all infants. Recognition of the weaknesses of Freud's motivational model led the psychoanalyst John Bowlby to propose an ethological view of attachment and a productive alternative interpretation of separation responses in infancy and early childhood.

Ethological Attachment Theory

In the early 1960s, advances in physiology and behavioral learning theory swept away the cornerstones of psychoanalytic drive theory. Bowlby (1958) saw the tide also threatened to sweep away a wide range of fundamental insights about emotion, development, and cognition that were part of Freud's legacy, but which were not inextricably tied to his motivational models and metaphors. Among these were Freud's emphases on:

1. The richness and complexity of cognitive and emotional life in infancy
2. Fundamental similarities between infant–adult ties and love relationships in adulthood
3. Limitations in our capacity to establish and maintain intimate relationships with more than one or a few central figures at one time
4. the fact that love relationships are never given up voluntarily or completely
5. The organization, adaptive function, and clinical implications of grief and mourning
6. The distinction between the behavioral and the cognitive/emotional responses to separation and loss

In brief, Bowlby sought to preserve the insights that led Freud to place the capacity to *love well* alongside the capacity to *work well* in his definition of psychological well-being. To this end, Bowlby (1958, 1969) proposed a model of infant–adult attachment that integrated psychoanalytic, cognitive, and ethological concepts. He proposed that the apparently purposeful aspect of the infant's behavior toward its primary caregiver, which Freud explained in terms of psychodynamic motivational concepts, could better be understood in terms of a behavioral control system. The system serves to manage information about the infant's state, the adult's behavior, and the environment, through a series of sensors, set goals, and effector mechanisms.

Lest this seem as magical as Freud's own formulations, Bowlby (1969) cited numerous examples of animal behavior patterns and man-made devices that employ feedback systems to effect apparently purposeful or intentional behavior in complex environments. He also cited evidence that

many animals are endowed with species-specific biases in learning abilities that make this type of control over complex social behavior patterns possible. Furthermore, he pointed to evolution as the mechanism through which human infants could be equipped with such a capacity. According to Bowlby, an attachment behavioral system developed in the course of primate evolution by virtue of the reproductive advantage afforded individuals who must successfully monitor environmental cues to danger and balance exploration of the environment with proximity to caregivers.

Bowlby's proposal places greater emphasis than Freud's on the environmental sensitivity and responsiveness of infant behavior. It offers a useful perspective on a variety of infant reactions that Freud felt were irrational in the context of the infant's objective experience. Bowlby suggested, for example, that reactions, such as fear of the dark, fear of looming objects, and separation-related fears, are quite rational when viewed in an evolutionary perspective. That is, the attachment behavioral system is attuned to such "natural cues to danger" because individuals who were wary or fearful in the dark or when alone were more likely to avoid predators.

The notion of a balance between attachment and exploratory behavior also helps to make sense of the fact that infants are typically more willing to leave a caregiver on their own than they are to allow the caregiver to leave. Both circumstances involve separation, but from the perspective of the attachment control system, they involve quite different information. Exploration away from the caregiver is *predicated* on an appraisal of the environment as safe and of the caregiver as accessible and responsive. The caregiver's departure, on the other hand, has the predictable *consequence* of reducing access to the caregiver and thus making unfamiliar environments seem more threatening. As a consequence, the balance between attachment and exploration shifts in favor of attachment behavior and the likelihood of proximity seeking and separation protest increases. Thus, the attachment control system is not sensitive to separation *per se,* so much as it is to the "meaning" of separation. To a great extent, this meaning is conveyed by the context in which separation occurs and by reference to past experience.

Despite Bowlby's emphasis on evolutionary mechanisms and on models and data from behavioral biology, his theory does not minimize the role of experience. The capacity to form attachments is considered part of our biological endowment, but attachment is acquired through experience and the attachment control system is exquisitely attuned to environmental information and to past experience (Waters, 1981; Waters & Deane, 1985). One of the central concepts in the theory is that of "working models." The models are cognitive/affective representations of a relationship between the self and a loved one that are built up over time and that give the attachment system access to expectations about the loved one's likely behavior in various situations. This not only places the concepts of experience and information at the heart of Bowlby's theory, it also provides a mechanism linking early experience to later attachment behavior without evoking explanations tied to Freud's energy hypothesis.

The Role of Learning Processes

At one time, there was considerable competition between ethological attachment theory and traditional learning theories (e.g., Gewirtz, 1972). Today, it is generally recognized that species-specific biases in learning abilities are the rule rather than the exception in almost every domain of behavior (Waters & Deane, 1982). Bowlby's model defines the parameters within which learning processes operate to shape and maintain human attachment and separation responses in a variety of environments.

Attachment behavior is clearly learned, as are many aspects of separation responses and reunion behavior. However, by virtue of our primate heritage, we are more likely to learn certain patterns of attachment behavior than others. For example, an attempt to follow is more likely to be the dominant response to separation than a period of crying. Learned patterns of separation behavior are directed specifically toward one or a few figures rather than indiscriminately toward many figures. Human infants are more likely to learn to greet happily and seek renewed contact after separations than to have adult-directed behavior extinguished in the course of brief separations. Ventral-ventral contact is more likely to be associated with comforting than is vocal exchange. Human infants are more likely to seek proximity than to avoid the caretaker when distressed, even if the adult has been strikingly abusive in the past. Within such constraints, the mechanisms of reinforcement, punishment, and imitation are in full play.

Learning theorists have suggested that separation anxiety and distress are manifested when the parent leaves the child and are reinforced when the

protesting infant is responded to by the parent. At present, there is little evidence to support the notion that responding to injuries, hunger, or other discomfort by the caregiver increases the rate of crying in other circumstances. Certainly, from the point of view of attachment theory, these two domains of behavior should be functionally distinct. Infants and young children are expected to be quite discriminating about contexts in which parental responsiveness occurs. Thus, differential control of distress responses and mere dependency should be easy to establish in many circumstances.

Temperamental Constructs

By definition, constitutional differences in behavioral style would show traitlike consistency across situations. Consequently, they do not predict or explain either the developmental course or the environmental sensitivity of separation-related behavior. Nonetheless, traitlike individual differences (whether inherited or acquired) may influence individual differences in the degree or duration of separation responses. Fearfulness, negative affect, low threshold for arousal, and distinctive patterns of response to stress (e.g., activation versus inhibition) may prove relevant. Such influences are consistent with both attachment theory and with learning theories. Unfortunately, the task of defining and validating age-appropriate measures for most temperamental constructs is far from complete. Thus, their role in the development, form, and maintenance of separation response is yet to be determined. Recent conceptual analyses by Goldsmith and Campos (1982) and Sroufe (1985) suggest useful directions.

Cognitive Factors

Cognitive development plays an important role in the acquisition and decline of separation protest in infancy and early childhood. In early infancy, the first true response to separation may involve simple associations between parental departure and isolation, and cries that bring the parents near. That is, the distress is in anticipation of separation rather than a consequence of it. This type of behavior occurs in early infancy and is occasionally elicited by the departure of adults other than the primary caregiver.

Truly differential response to separation from a primary caregiver typically emerges in the second half year of life, when the development of object permanence allows the infant to maintain a representation of a specific adult even when he or she is out of sight. Cicchetti and Sroufe (1978) provided strong evidence of the relationship between cognitive development and separation protest by demonstrating that its onset is delayed in infants with Down syndrome, and that the delay is correlated with the degree of cognitive retardation.

A variety of cognitive factors influence the decline of separation protest in the second and third year of life. With increasing cognitive ability, the infant is more able to monitor and predict an adult's behavior. With age, the infant becomes increasingly familiar with a wider range of environments and circumstances. This knowledge base broadens the infant's view of what is "familiar" and makes the infant less likely to become distressed by separations outside the home. Infants and toddlers learn more about their own advancing coping skills. Within limits, they learn how to modulate interactions with strangers and how to explore new objects and environments without overcommitting themselves and falling into trouble. Language development and improved communication greatly expand the infant's coping and ability to manipulate the environment. Awareness of these emerging skills is an important aspect of self knowledge and plays a role in the decline of separation protest.

Normal Individual Differences

By the end of toddlerhood, all normal children have the cognitive abilities necessary to keep concern over ordinary separations within bounds. Nonetheless, there are considerable individual differences in separation responses at this age. Each of the mechanisms outlined above can contribute to this. Specific experiences, expectations about a caregiver's availability and responsiveness, temperament, and a child's evaluation of its own coping abilities are interrelated in the child's experience and development. In a particular instance, any or several of these may determine a child's response to separation. Similarly, different mechanisms may be operating in different children who have similar responses to separation.

Traumatic Separation

Bowlby's analysis of attachment and separation developed out of his work with 2- to 3-year-olds who had experienced prolonged separations, and from wartime observations of children who had

lost their parents permanently. In these, and in observations of children separated while their mothers were in the hospital having a second child (Bowlby, 1961; Robertson & Bowlby, 1952), Bowlby identified a three-phase response to long-term separation in children from about 6 months to 4 years old. Similar patterns of response to traumatic separations were described by other researchers (Burlingham & Freud, 1944; Illingworth & Holt, 1955). The first phase, *protest* is characterized by active crying and protesting the absence of the mother. The child is active in searching for the mother. The second phase, *despair,* occurs when the child appears to lose hope of seeing her and becomes subdued, remaining preoccupied with her absence. The child may alternate between the protest and despair phases for some time as hope is regained and then lost again. In the third phase, *detachment,* the child seems to forget the mother and, if she returns, appears to be uninterested in her (Bowlby, 1973). These behaviors are particularly associated with institutional care or when a substitute attachment figure is not available (Robertson & Robertson, 1971).

The child's response to the mother in reunion depends in part on the phase manifested at the time of her return. If the child has entered the phase of detachment, there is usually a period of ignoring the mother. If the child has been in the phase of detachment for a very long time, as in a separation of 6 months or more, the child may remain detached and be unable to reconnect with the parent. Generally, the period of unresponsiveness lasts for hours or possibly a day. Ignoring the mother is followed by a period of intense ambivalence on the part of the child, with anger, and clinging behavior occurring in close succession or even simultaneously. Any sign of departure by the mother precipitates intense separation anxiety, with clinging and angry protests. For weeks or even months, the mother will find the child to be demanding of her attention and reproachful if she is not attentive (Bowlby, 1973). Tantrums and other angry outbursts are observed to increase both during and following the separation.

The phases of response to separation represent a process of mourning that, according to Bowlby, may or may not become pathological. He postulates that if the intense ambivalent feelings that are felt by the mourning individual are not expressed, then the behavior and feelings of the individual may become distorted by these active, yet defensively repressed, emotions (Bowlby, 1961). The detachment phase appears to be a phase of such repression, according to Bowlby. The intense attachment behavior and separation anxiety that occur if the child is reunited with the parent constitute a release of these emotions. He suggests that repression of these feelings or lack of opportunity to express them is preliminary to the development of certain forms of psychopathology.

There are a variety of situations that may lead to a traumatic separation of a child from his or her parents. Children's responses to these situations or events, such as hospitalization, parental death, and divorce, have been observed, and symptoms and reactions include, but are not confined to, separation anxiety.

Hospitalization

Admission to the hospital is one of the most common reasons for a young child to be separated from its parents. There is a considerable body of knowledge concerning responses to hospitalization. The patterns of behavior described above have been observed in the hospital when the child is unable to have contact with the parent (Garmezy, 1983; Illingworth & Holt, 1955). The stress of the separation seems to lie particularly in the disruption of the attachment bond without opportunity to reconnect to a new person (Rutter,1983).

There are a number of interventions consistent with attachment theory that can effectively diminish the child's distress in the hospital. In the short term, a child's anxiety concerning separation and other manifestations of distress are considerably alleviated by daily visits or the presence of a parent or other attachment figure. If this is not possible, the presence of a nurse who is assigned to care regularly for the child also reduces emotional disturbances during the stay (Rutter, 1983). The opportunity for the child to have a surrogate attachment figure seems crucial if the parents are not available (Garmezy, 1983). In addition, education of the child, and particularly the parents, concerning the hospital stay appears to benefit children greatly. Children appear to respond to parental attitudes and anxieties as much as to anything that actually occurs in the hospital (Rutter, 1983). Hospital policies in pediatrics and other medical specialities have changed considerably in the past 20 years as a result of knowledge of the potentially problematic responses to separation and hospitalization. Many hospitals now allow parents to room in, provide primary nursing care, do not restrict visiting hours for parents, and allow children to visit on adult wards.

Bowlby's hypothesis that separation and unresolved mourning in infancy and the preschool years may be a precursor to the development of psycho-

pathology had led to a great deal of research on the effects of hospitalization on young children. A single hospital admission is not associated with an increased risk of developing long-term behavioral disturbance, although the short-term disturbances of the type noted by Bowlby may be observed for several months. The disturbances are more intense when the child has a poor relationship with his parents prior to admission or if there is conflict and discord within the family (Garmezy, 1983). Two or more hospitalizations in early childhood are associated with development of later problems (Quinton & Rutter, 1976). The reasons for this are unclear, but may relate to a sensitization effect of the first admission on either children's or parents' reactions, or to alterations in the parent–child relationship which follow the admission (Rutter, 1983). Again, the risk is greatest for children from families that are disrupted or for those who are in conflict with their parents. A problematic outcome is not typical, but is most often seen in boys who have difficult family backgrounds prior to separation (Rutter, 1981).

Death of a Parent

The research in the area of loss of a parent is sparse and plagued by methodological problems; thus, the effects of parental death and mourning on children's behavior and separation anxiety, in particular, are unclear. In the period immediately following a parent's death, several responses have been noted. Boys who lost fathers have been reported by their widowed mothers to show major depressive symptoms (Clayton & Darvish, 1979). Other reactions of children noted by the surviving parents included withdrawal, decreased school performance, sleep problems, and tantrums. The symptom of separation anxiety was not specifically reported, and the children were not observed or interviewed themselves. Major symptoms of depression have been reported in psychiatrically hospitalized children who had experienced the death of a parent (Arthur & Kemme, 1964). The children showed personality changes and aggressive reactions. In this population of very disturbed children, fears of recurrence of the event were noticeable, as were phobic reactions and sleeping problems. Some children expressed suicidal ideation and a wish to join the parent in death. The children in this study had lost their parents through a variety of circumstances including accidents and suicide as well as by natural causes.

Bowlby has hypothesized that loss in the early life of a child is a substantial risk factor for the development of later psychopathology. Rutter (1981) suggests that perhaps only certain forms of early loss predispose for later problems, emphasizing the importance of social circumstances surrounding the loss. A link has been found between parental loss in childhood and depression in adult life, particularly for girls who lost their mothers (Brown, Harris, & Bifulco, 1986). These authors postulate that loss creates a vulnerability, but depression occurs only if there are provoking factors as well as the vulnerability. The vulnerability may relate to a cognitive bias toward helplessness because of emotional deprivation. Or it may relate to lack of social supports following the loss and subsequent adverse life events, in particular, premarital pregnancy and a poor marriage. It is possible that infants and preschool children may be less vulnerable to the effects of parental death than older children (Garmezy, 1983). The younger child seems to grieve with less intensity, and may be protected by lack of cognitive ability to conceptualize death as a universal and irreversible process. Furthermore, the younger child may be more able to attach to a new person and therefore to benefit from caregiving by a consistent replacement attachment figure.

Main and Solomon (1987) described a lack of resolution of mourning in certain adults who experienced loss in childhood. The lack of resolution of mourning is manifested by guilt that the individual as responsible for the death, by bizarre, incoherent, or disorganized ideas about the death or dead person, or by denial of the death or its impact. Infants of these adults are observed to show disorganized attachment behavior in the Strange Situation laboratory procedure (Ainsworth, Blehar, Waters, & Wall, 1978), manifesting mixed patterns of avoidant and resistant behavior, stereotypies, and apprehension or fear of the parent. Some infants direct separation protests toward the stranger rather than the parent. Older children of these parents have shown behaviors that are controlling or manipulative of the parent, in that they are either punitive and provoke guilt in the parent, or they show excessive concern for the parents' well-being and comfort.

Inability to come to a resolution of mourning may relate to the circumstances of the death or the relationship that the individual had with the lost attachment figure. It is plausible that death of the parent under atypical circumstances, such as suicide or accident or when the child witnesses the death, has greater effects on the child's development and behavior than a death that was anticipated or within the realm of the expectable. However, it is also possible that circumstances associated with atypical death are risk factors in themselves for de-

velopment of psychopathology. For example, parental suicide may have a direct impact on the child, but, in addition, the relationship with the parent may have been problematic prior to the suicide or the psychiatric disorder of the parent may be heritable.

Loss of a Parent by Death versus Divorce

Interesting comparisons have been made between the loss of a parent by death and loss because of parental divorce or marital conflict. Blame and anger in the children are frequent sequelae of divorce but are not as evident in children whose parents have died. In both circumstances, guilt is present. However, in divorce, perhaps because the loss is only partial, the child may seek to assuage the guilt and be preoccupied with ideas of restoring and repairing the marriage for a much longer time. The child and family who experience a divorce may be less supported by family and friends than those who by virtue of the death are sanctioned to grieve and be comforted (Wallerstein & Kelly, 1980). Conduct disorders or antisocial problems are significantly less likely to develop in children who have experienced the death of a parent because of illness or accident than in those who come from families that are broken by divorce or separation. In particular, it appears that cold, hostile relationships with and between the parents place the child at particular risk for developing antisocial behaviors. Divorce may improve the child's odds if the home becomes calmer and more harmonious following the departure of one parent, or the child may be "protected" by a positive relationship with one of the parents.

Parental Depression

Parental depression can be considered a form of separation or loss of the parent insofar as it entails relative unresponsiveness of the parent to the child. When a depressed parent is so unresponsive that he or she neglects an infant, the infant may develop a profound disorder known as Reactive Attachment Disorder of Infancy (American Psychiatric Association, 1987). The infant becomes withdrawn, apathetic, and responsive. It may fail to thrive. Toddlers with this disorder can manifest indiscriminate and disorganized attachment behaviors and separation anxiety. In the recovery phase, when the child is receiving good care, very intense attachment behavior may be observed. Clinging, sensitivity to separation, and angry tantrums are reminiscent of behaviors that are seen following long-term or traumatic separation. Lack of care in childhood, as defined by neglect and lack of attention, appears to be linked with depression in adult life (Brown *et al.*, 1986).

Even in less extreme circumstances, infants and children of depressed parents show disordered behavior. Infants of parents with bipolar illness show an increased incidence of insecure/ ambivalent attachment behavior from 12 months to 18 months of age, with decreased capacity for self-regulation of emotion, particularly fear and anger (Cytryn, McKnew, Zahn-Waxler, & Gershon, 1986). By age 2, the same children were noted to have a variety of disturbances, including phobias, eating and sleeping problems, excessive dependency, tantrums, poor impulse control, and difficulty regulating affect, to name a few. Most of the children continued to be classified as insecure in their attachment relationships to the mothers (Zahn-Waxler, McKnew, Cummings, Davenport, & Radke-Yarrow, 1984). At age 5, the children were manifesting mood disorders that resembled adult depression. Thus, for children of parents with mood disorders, insecure attachment and difficulty with regulation of affect in the infant and toddler may be precursors of anxiety and/or depressive illness in the older child (Cytryn *et al.*, 1986).

Psychopathology

Separation anxiety can present to a pathological degree in children, as a primary problem or in conjunction with other disorders of childhood.

Separation Anxiety Disorder

The DSM-III-Revised (American Psychiatric Association, 1987) defines separation anxiety as excessive anxiety concerning separation from attachment figures, beyond that expected for the child's development level. Thus, a very young child may receive the diagnosis if the dysfunction is severe, and the behaviors are considered abnormal in the school-age child or adolescent even if the symptoms are not severe (Klein, Gittelman, Quitkin, & Rifkin, 1980). Unlike many disorders of childhood, Separation Anxiety Disorder occurs with equal frequency in girls and in boys.

The problems manifested by the children range in severity, but may interfere with the children's leaving the home alone. Often they cannot participate in normal activities of their age group, such as going to sleep at friend's houses, going to school or to camp. The child may shadow the parent and be unable to stay in a room alone. Somatic

complaints are common in these children, especially if separation is anticipated or occurs. The child may be fearful that harm will come to the attachment figure. This anxiety seems particularly related to concern that the attachment figure will disappear, as opposed to being a concern for the parent's well-being. The child may worry that he or she will be harmed or abducted. Other fears may also be present, including concerns about death. Sleep disorders are quite common, with nightmares involving the idea of separation. The child may wish to sleep with the parents. Children with the disorder may complain that no one loves them, and they can be very demanding of attention and care. Alternatively, they may be very eager to please.

The etiology of the disorder in general and its predisposing disturbances are not clear. The disorder may appear without obvious precipitant in an apparently well-functioning child. But frequently there is a stressor, usually a loss. The families of these children are most often described as caring and close; the disorder is unusual in families in which there is neglect or hostility. The incidence of separation anxiety is high in children with mothers with Panic Disorder.

Other Disorders Associated with Separation Anxiety

Children with Posttraumatic Stress Disorder may have symptoms of separation anxiety, or they may meet full criteria for Separation Anxiety Disorder. Children with this disorder have experienced a traumatic event that is outside the bounds of normal human experience, and the disorder is associated with strong avoidance of events that are reminiscent of the trauma. Those whose symptoms involve separation anxiety are likely to have had a traumatic and unusual separation, such as being kidnapped. Children with Major Depressive Disorder may also have separation anxiety as part of their depressive symptomatology. One of the most common manifestations of depression and separation anxiety is the syndrome of School Phobia or School Refusal.

Treatment of Separation-Related Problems

Infants, Toddlers, and Preschoolers

There is little clinical research literature or empirically validated advice to parents on behavioral methods for managing separation-related problems in infants and in young children. This may be due primarily to the fact that parents and physicians view distress over separation and even strong protests as more or less an age-appropriate reaction until a child reaches age 4 or 5. Early separation problems are not consistently associated with subsequent problems and thus have not been included in standard diagnostic systems.

Attachment theory can inform both clinicians and parents who are faced with the task of understanding, manipulating, or managing separation-related behavior. It suggests that punishing separation-related behavior is particularly likely to be unsuccessful because the distress it causes heightens the child's sensitivity to separation and the desire to seek proximity. Nonaversive methods, such as training alternative behaviors that can occupy the child during brief separations, reinforcing independence, and desensitization beginning with very brief departures, are likely to be most effective. Such methods have been employed successfully in helping young children to fall asleep alone, a behavior that is related to separation (Cuthbertson & Schevill, 1985).

Ethological theory further suggests that separation training is most likely to succeed if it begins in familiar settings, if there are familiar adults or other children present, and when the child is well and not tired. Most importantly, perhaps it suggests that sometimes separation simply is not in order. Parental responsiveness can play a role in teaching the child to communicate without resorting to cries, and it is also instrumental in teaching the child that caregivers are accessible even when not immediately present. Thus, there is an expectation that parental responsiveness to infant or toddler signals increases rather than decreases independence, and that behavioral intervention can have effects far beyond the target behavior (Posner, 1988).

Psychopathology

The child who experiences separation anxiety to a pathological degree, and who has extreme fear about separation from the parents requires careful evaluation because of the multiple factors that may be present. Although all the factors associated with separation distress in the toddler may be present, family factors are likely to be prominent in many cases. Treatment must therefore involve the family not only to teach parents the skills to help the child (as is true for the toddler or preschooler) but also to understand what factors within the family system may support or maintain the child's symptoms. For

example, a child's extreme closeness and dependence may mask or draw attention away from a difficult marriage and thus may be unconsciously supported by the parents. Furthermore, it is not uncommon to find that the parents of these children are depressed or extremely anxious themselves about separation and other experiences. Family therapy and parental treatment may be necessary to enable the parents to institute management techniques with the child. Clarification and addressing of other problems with the system may free the child from the symptom without direct behavioral intervention.

In some cases, drug therapy may serve as an adjunct to family and behavioral management of the disorder, particularly if depression is a dominant feature. A number of adult patients with agoraphobia report a history of separation anxiety in childhood. The adults with the disorder may have symptoms of clinging and dependent behavior as well as anxiety concerning panic attacks and being out in public. Agoraphobia and panic attacks are effectively treated with imipramine in most cases. Because of the association of the childhood disorder with the adult disorder and some behavioral similarities, imipramine treatment of children with separation anxiety has been tried. Results have tended to show children responding positively to the drug, but investigations have been limited (Klein, Gittelman, Quitkin & Rifkin, 1980).

In summary, the multiplicity of mechanisms associated with separation anxiety probably accounts for the fact that early separation responses are not consistently related to significant clinical problems. It also highlights the advantages of approaching diagnosis and designing interventions as complex problem-solving tasks that require systematic evaluation of diverse etiological models (e.g., Levine & Sandeen, 1985).

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CHAPTER 17

The Temperamental Qualities of Inhibition and Lack of Inhibition

Jerome Kagan, J. Steven Reznick, and Nancy Snidman

Introduction

The increased interest in temperamental qualities during the last 20 years has corrected the theoretical imbalance created by 50 years of extreme environmental interpretations of the sources of behavioral variation in children by reminding investigators of the interdependence of agents and their surroundings. Biological and behavioral phenomena are emergent events resulting from the interactions of inherent qualities within the agent and facilitating or constraining conditions in the contexts in which the agent grows. Scholars have always had difficulty finding a clear, satisfying way to describe the sense meanings of the terms *emergent* and *interaction,* resorting often to illustrative examples from embryology. If a biologist transfers primordial neural crest cells that are destined to become part of the cornea to a different site, those cells may grow into a ganglion of the sympathetic chain if the transfer occurs before a critical time in the differentiation of the cells. No covariance analysis will permit biologists to evaluate the relative importance of the cells' internal characteristics and those of the surrounding tissue with respect to the cells' fate. The outcome is an emergent unity requiring interaction of the cells and their context.

Behavioral and affective dispositions in infants are also the emergent consequence of tonic physiological states, produced by genetic and/or prenatal events, and postnatal environmental experiences (Goldsmith & Campos, 1985; Thomas & Chess, 1977). Some of these dispositions will be transient, but those that persist for months, a few years, or even longer will invite a profile of reactions from others and bias the child to react to some experience in special ways. These stable biases in perception and behavior are called *temperamental qualities.* As might be expected, when a domain is young its categories often originate in the conscious concerns of the community and refer to relatively obvious and therefore easy to quantify characteristics. Many American and European mothers have many things to do with no relatives or daughters to help them; hence, the ease of caring for a baby is one of its salient qualities. An excessively irritable, labile, or fearful infant is harder to nurture than a calm, stable, happy baby, and irritability, lability, and fear are easily observed characteristics. Hence, they have been regarded as primary temperamental variables, analogous to the primacy of air, water, fire, and earth in classic Greek metaphysics.

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The empirical record suggests that one of these qualities, initial withdrawal or behavioral inhibition to the unfamiliar, seems to persist for a longer time than the others, is part of a coherent network of characteristics, and has reasonable analogues in many mammalian species (Blizard, 1981; Bronson, 1981; Brush *et al.*, 1985; Coie & Dodge, 1983; Goldsmith & Gottesman, 1981; Kagan & Moss, 1962; Kagan, Reznick, & Snidman, 1989; Lewis, Feiring, McGuffog, & Jaskir, 1984). The tendency to withdraw from or approach an unfamiliar event emerges at about 8 months in humans, 2 months in monkeys, and 1 month in cats and dogs. The idea that members of a species vary in the likelihood of approach or withdrawal is central in the writings of Aristotle, Plato, and Galen and, in more recent times, Sheldon's tripartite typology of personality. Jung's theory of psychological types made introversion and extroversion major differentiating characteristics in humans (Jung, 1924), and he credits this idea to a rarely cited book by Furneaux Jordan (1896). Jung quotes Jordan:

> There are two generic fundamental [biases] in character . . . two conspicuous types of character . . . one in which the tendency to action is extreme and the tendency to reflection slight, and another in which the proneness to reflection greatly predominates and the impulse for action is feebler. (cited in Jung, 1924, p. 184)

Schneirla's (1959) suggestion that approach and withdrawal are primary dimensions in animals is affirmed by studies of various mammalian groups, including rats, cats, dogs, and monkeys. Scott and Fuller (1965/1974) found that variation in timidity was one of the two most differentiating characteristics in five breeds of dogs, and both Suomi (1984) as well as Stevenson-Hinde, Stillwell-Barnes, and Zunz (1980) have reported similar variations in approach–withdrawal behavior among laboratory reared Macaque monkeys.

In children 1 to 2 years of age, these dispositions appear most often to unfamiliar persons, contexts, and objects, and the withdrawal is often accompanied by crying and/or seeking a target of attachment. In one study of eight 1-year-olds exposed to eight different strangers on four different occasions, two infants were consistently distressed and two showed minimal distress to all the strangers (Smith & Sloboda, 1986). By the child's third birthday, this quality is more difficult to describe, because the feature of withdrawal is considerably more subtle. When an unfamiliar person enters a room, the temperamentally inhibited child, who would have retreated to the mother at 1 year of age, will cease playing and talking and show a prolonged latency to approach the adult. A year later, when in an unfamiliar room with an unfamiliar child, the inhibited child will fail to initiate play for the first 10 to 20 minutes, remaining quiet and close to the mother. And if the child has retained this quality until 7 years of age, initially he or she will be quiet with an unfamiliar adult, will play apart from peers in a group context like the school, will display a serious facial expression, and will become stressed by mild challenge or occasional failure (Kagan *et al.*, 1989).

These conclusions are based primarily on a longitudinal study of two cohorts of Caucasian children who have been followed from age 2 or 3 through 7½ years of age (Garcia-Coll, Kagan, & Reznick, 1984; Gersten, 1986; Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984; Reznick, Kagan, Snidman, Gersten, Baak, & Rosenberg, 1986; Snidman, 1984). The Caucasian children were selected to be behaviorally extreme with respect to the probability of their withdrawal or approach to unfamiliar people and events. The first cohort was selected when they were 21 months old, the second when they were 31 months old. The classification of the child as inhibited (likely to withdraw) or uninhibited (likely to approach) required the child to show consistent withdrawal or approach to a variety of unfamiliar incentives. It was necessary to screen over 400 children in order to find approximately 60 inhibited and 60 uninhibited youngsters with equal numbers of boys and girls in each cohort. Each cohort was evaluated on two successive occasions, and the first cohort is being seen currently at 7½ years of age.

In general, the original dispositions have been preserved through 7½ years of age, even though each quality is displayed in a different form at the older ages. At 21 months, the inhibited children in a laboratory playroom stay close to their mother, withdraw from a female stranger and an unfamiliar toy, and show occasional fretting. At 5½ years, the child is reluctant to interact with an unfamiliar child in a laboratory or school, remains quiet in a testing situation, and is often isolated from his classmates in his kindergarten classroom. By contrast, an uninhibited 21-month-old child approaches the unfamiliar stranger and toy, plays apart from the mother, and, at 5½ years, initiates contact with peers, is vocal and affective with an examiner, and is frequently engaged in social interaction with classmates.

The laboratory index of behavioral inhibition at 21 months was positively correlated with an ag-

gregate index of behavioral inhibition at 5½ years that included behavior with an unfamiliar peer, with an unfamiliar examiner in a laboratory, and with classmates in a school setting ($r = .52, p < .001$). However, there was more obvious preservation of uninhibited than inhibited behavior. It is likely that because most American parents regard sociable, bold, outgoing behavior as more adaptive than a timid, fearful, withdrawn style, they encourage the inhibited child to alter his or her initial tendencies. About 40% of the original group of inhibited children became less inhibited by 5½ years of age, whereas less than 10% of the uninhibited children became more timid. Further, more boys than girls have changed from an inhibited to an uninhibited profile, whereas the small number of uninhibited children who became more timid were girls from working-class families.

The 5½ year-old children who had been classified earlier as inhibited had more fears than uninhibited children. The fears were usually of large animals, fire, a lake or ocean, machines, unfamiliar children, thunderstorms, and monsters on television or in the movies. If, in addition, the inhibited child had a consistently high and stable heart rate (in the laboratory), he or she had an average of 3 out of 10 possible fears; over one half of these children were afraid of monsters and one third had unusual fears or night terrors. The occurrence of a fear in an uninhibited child was usually associated with a prior trauma; this was not true for the inhibited children. For example, one uninhibited boy with an unusual fear suffered two traumata between his original classification and the subsequent assessment at 5½ years—his father had left home, and the boy had been sexually abused by his older cousin.

Biological Contributions

There are several possible explanations of the variation in this complex quality or qualities. During the 40 years when behaviorism and psychoanalytic theory shared popularity, many American and European psychologists viewed the variation in fearful behavior as learned. The American behaviorists offered a logically consistent and intuitively appealing argument based on the conditioning of a state of fear and the biologically prepared response of avoidance to formerly neutral events. The popular textbook example is of the infant who acquired a fear of a white rat. In this model, a child who was consistently shy with unfamiliar adults must have had painful experiences with adults in the past. Hence, many psychologists teaching during that era explained intense stranger anxiety in the infant as a consequence of unpleasant experiences with strangers. In a similar vein, an extreme degree of separation anxiety was the result of experiences of hunger or pain when the mother was away from the home; hence, the mother's absence became the conditioned stimulus for an anxiety reaction. Although this explanation seemed reasonable 30 years ago, only a small number of contemporary psychologists continue to favor a version of this argument.

Two related explanations that are gaining initial consensus do not deny the influence of learning from experience, but implicate the role of inherent individual differences in central nervous system functioning. One explanation holds that children differ in ease of excitability—or threshold of discharge— in those parts of the central nervous system that contribute to the parallel states of psychological uncertainty and autonomic arousal. Most investigators assume that limbic structures play a central part in both phenomena (Gray, 1982). A specific variant of this hypothesis is that some infants are born with low thresholds of reactivity in amygdala and hypothalamus to unfamiliarity or challenge for which the child has no immediate coping response. A related, complementary explanation emphasizes the effectiveness of processes that inhibit the discharge of limbic lobe structures. As Rothbart and Derryberry (1981) note, these two mechanisms are different, even though investigators working with humans cannot measure each separately at the present time. Pavlov chose to emphasize the inhibition function, suggesting that organisms differed in the "strength of the central nervous system," in which strength meant the ability to inhibit the build-up of excitation. Pavlov implied that fearful dogs and shy children had a "weak nervous system." The Polish psychologist Jan Strelau (1985) is responsible for a modern version of Pavlov's ideas.

The environment presents children with at least three classes of events that invite some form of response. The first class refers to unfamiliar events that provoke attempts at assimilation (e.g., an unfamiliar sound or sight). The second class includes events, usually the actions of people, to which the child must issue some action (e.g., an unfamiliar person approaches and offers a toy). In the third class, the environment presents problem situations to which the child must generate cognitive solutions (e.g., test questions in our battery). Most of the time children and adults assimilate unfamiliar events easily, issue socially effective actions to oth-

ers, and generate correct cognitive solutions with minimal delay, and, therefore, minimal limbic lobe arousal. But when there is a delay in the generation of a coping response, the child is simultaneously alerted psychologically and aroused physiologically. We believe that this state should be treated as a special psychological-cum-physiological condition mediated, in part, by limbic structures. We choose to call this state *uncertainty;* others may prefer a different word.

The state of uncertainty/arousal often has peripheral physiological consequences because the amygdala and hypothalamus mutually influence each other, and the latter influences the pituitary-adrenal axis, the reticular activating system, and the sympathetic chain (Axelrod & Reisine, 1984). We have gathered evidence indicating that inhibited children show high levels of reactivity in the peripheral targets of these systems. Specifically, at 5½ years of age, the inhibited children in the first cohort, compared with the uninhibited ones, had higher levels of muscle tension, as reflected in low variability of the pitch periods of vocal utterances (Coster, 1986), higher and more stable heart rates, larger pupillary dilations, and higher levels of salivary cortisol and urinary norepinephrine. The correlations between the mean of eight physiological variables derived from the above measures and the indexes of behavioral inhibition at 21 months, 4 years, and 5½ years were, respectively, .70, .66, and .58 (all correlations were significant at $p < .001$).

We interpret these data to mean that inhibited children have lower thresholds of reactivity in the limbic structures that mediate these peripheral physiological variables. This speculative suggestion is in accord with results of recent neurobehavioral studies of the amygdala and the hypothalamus. The amygdala receives evaluated, meaningful information from secondary sensory areas and hippocampus, and, through projections to the hypothalamus and autonomic nervous system, generates visceral reactions that are accompanied by behavioral signs of fear and feelings of increased emotionality (Aggleton & Passingham, 1981; Gloor, 1978; Sarter & Markowitsch, 1985; Turner, Mishkin, & Knapp, 1980). Indeed, one team of investigators has suggested that conscious feeling tone in humans is generated in the amygdala (Hebben, Corkin, Eichenbaum, & Shedlack, 1985).

The idea that inhibited and uninhibited children differ in the excitability of amygdala and hypothalamus finds support in studies of domestic cats (*Felis catus*), differing in degree of defensive, nonaggressive behavior with rats. The descriptions of the defensive, in contrast to the nondefensive, cats resemble closely our descriptions of inhibited and uninhibited children because defensive cats are less likely to attack rats and are more timid in novel environments as well as with humans. These behaviors are stable over periods as long as 3 years (Adamec & Stark-Adamec, 1986). Stimulation of the basal amygdala evoked stronger multiple-unit activity in the limbic lobe of defensive than nondefensive animals. Further, when the amygdala was stimulated with single pulses of increasing intensity, the peak height of the evoked potential in the ventromedial hypothalamus was significantly greater in defensive than in nondefensive cats. Defensive cats also showed a larger and more prolonged increase in multiple-unit activity in the amygdala while orienting to rats. Adamec and Stark-Adamec (1986) suggested that differences between defensive and nondefensive cats may be due, in part, to a lower threshold of response in the amygdala:

> Some form of synaptic potentiation underlies [this] behavioral disposition [or bias] . . . the behavioral outcome may be determined by the normally occurring excitatory status of limbic substrates [p. 142]. . . . some form of potentiation of synaptic transmission. (p. 132)

This suggestion, together with data indicating that the amygdala appears to mediate the state of conditioned fear in the rat (Hitchcock & Davis, 1986), renders more credible the idea that differences in responsivity of limbic lobe structures make an important contribution to the contrasting behavioral profile of inhibited and uninhibited children.

The reasons for the lower thresholds of reactivity in limbic structures are unclear, but one possible contributing factor is high levels of central norepinephrine. Central norepinephrine appears to amplify the brain's reaction to novelty by suppressing background neural activity and, therefore, increasing the psychological salience of an incentive stimulus (Aston-Jones & Bloom, 1981; Charney & Redmond, 1983; Charney, Heninger, & Breier, 1984; Reiser, 1984). For example, rats that are unable to avoid shock show increased norepinephrine activity, especially in the amygdala and the hypothalamus (Tsuda & Tanaka, 1985). Furthermore, assays of a large number of autonomic and hormonal variables in phobic women exposed to the sources of their fear revealed that blood levels of norepinephrine had the largest number of correlations with the other physiological indexes (Nesse, Curtis, Thyer, McCann, Huber-Smith, & Knopf, 1985).

(See also Bandura, Barr-Taylor, Lloyd-Williams, Mefford, & Barchas, 1985.)

The locus coeruleus is the major source of central norepinephrine and its axons synapse on many parts of the brain, including the amygdala and the hypothalamus. The fact that neurons of the locus coeruleus of rats fire to the novelty of unexpected tones and lights led Aston-Jones and Bloom (1981) to suggest that the increased state of vigilance to novelty is accompanied by increases in the concentration of central norepinephrine, as if the locus coeruleus–norepinephrine system rendered originally neutral, innocuous stimuli anxiety provoking (Reiser, 1984). Thus, one possible hypothesis, albeit speculative, is that inhibited children have tonically higher levels of norepinephrine than do uninhibited children because of the enhanced reactivity of the locus coeruleus. As a result, a mildly unfamiliar or challenging event is more likely to generate neural discharge in the amygdala and hypothalamus of inhibited children, and, during the early years, an accompanying disposition to become quiet, to cease playing, and to withdraw from the event.

Because inhibited children are shy with unfamiliar peers and adults, it is likely that a small number of them will become introverted adults. This is a reasonable suggestion, because adults who are classified as introverts on Eysenck's Scale (Eysenck, 1982) resemble our inhibited children in some respects (Eaves & Eysenck, 1975; Gange, Geen, & Harkins, 1979). In contrast to extroverts, introverts show larger increases in heart rate under mild stress (Hinton & Craske, 1977), and a less variable heart rate in a vigilance procedure (Thackray, Jones, & Touchstone, 1974). It is of interest, therefore, that adults selected to be introverts performed better than extroverts on tests in which they had to detect an infrequent, and often subtle, signal embedded in a background. For example, following a light signal, introverts detected low-frequency auditory signals (500 Hz) better than extroverts, although there was no difference in the detection of 8000 Hz sounds (Stelmack & Campbell, 1974). Further, introverts showed a larger amplitude of the N1–P2 component of the auditory evoked response to 500 Hz, but not to 8000 Hz tones (Stelmack, Achorn, & Michaud, 1977). Additionally, introverts performed better than extroverts on tasks in which the person had to detect the infrequent occurrence of a slight increase in brightness of a circle on a black background (Hockey, 1986). It is believed that norepinephrine suppresses background activity in the central nervous system and therefore enhances signal-to-noise ratios. Thus, if introverts (and inhibited children) had higher levels of central norepinephrine, they should have lower sensory and detection thresholds.

One consequence of the lower thresholds to sensory events is a greater probability of establishing an avoidance reaction to a state of discomfort produced by a mildly unpleasant stimulus. Consider two children, one inhibited and one uninhibited, who have had a mildly painful bowel movement because of a hard bolus. If the salience of the pain was exaggerated for the inhibited child, an association between defecation and an unpleasant state would be probable. According to their mothers, more inhibited than uninhibited children developed brief periods of fear of the toilet as a result of a painful bowel movement.

There may be a modest relation between temperamentally based inhibition in childhood and heightened risk for agoraphobia and panic disorder in adulthood. Panic patients, usually women, report that they were extremely anxious children who showed fear of leaving home and going to school (Gittleman & Klein, 1984). More adult panic patients have high heart rates and higher levels of norepinephrine and cortisol than patients from different diagnostic categories (Freedman, Ianni, Ettedgui, Pohl, & Rainey, 1984; Nesse, Cameron, Curtis, McCaan, & Huber-Smith, 1984). Some investigators believe that panic attacks are due to central noradrenergic activity ascribable to enhanced reactivity in the locus coeruleus. Furthermore, agoraphobia and panic may be heritable, because about 25% of the relatives of panic patients, in contrast to 2% of controls, have some form of anxiety disorder (Harris, Noyes, Crowe, & Chaudhry, 1983), and twin studies imply a genetic contribution to panic attacks but not to generalized anxiety (Torgersen, 1983). We have found in our laboratory that about 60% of children 4 to 6 years of age who have a parent diagnosed as having either panic disorder or panic combined with depression are extremely inhibited. There was not one inhibited child in a control group of children of the same age and social class whose parents had sought psychiatric help for smoking or who had a sibling diagnosed as having attention deficit disorder.[1]

The heritability of the disposition to inhibition and lack of inhibition is also affirmed by studies reviewed recently by Plomin (1986). For example,

[1] This research is being conducted in collaboration with Joseph Biederman, Jerrold Rosenbaum, and Michelle Gersten of the Massachusetts General Hospital, Boston, Massachusetts.

during the first 3 years of life, a tendency to approach or to withdraw from people shows high heritability. Matheny, Riese, and Wilson (1985) and Plomin and Rowe (1979) found that fearfulness to adults during the first 2 years of life was more similar in monozygotic than in dizygotic twin pairs; Buss and Plomin (1975) report coefficients of .62 for monozygotic twins and .13 for dizygotic twins with respect to sociability at 5 years of age (for details, see Plomin, 1986).

Summary

First, although we believe that some children are born with a tendency that favors either behavioral inhibition or lack of inhibition to the unfamiliar as a result of low or high thresholds of excitability in limbic structures, we believe that the physiological and the psychological qualities are malleable to alteration with proper experience. Children in both groups have shown changes in behavior and physiology, despite the statistically significant preservation of initial differences between them. Second, by 4 years of age, most inhibited children have become aware of their behavioral and affective reactions to threat and novelty and want to control their fear and timidity. One 5½ year-old inhibited boy in our study told his mother, "I know I am afraid, but I am trying to not be." Many inhibited children have learned to react to the experience of uncertainty with less timidity. Such children may acquire the ability to interrupt the prepared response of withdrawal and inhibition. As these coping responses become stronger, they might even mute the uncertainty and autonomic arousal that occur in unfamiliar settings. Just as knowledge about the reversibility of a Necker cube makes it easier to see it in both perspectives, so, too, might knowledge about one's physiological and behavioral reactions to unfamiliarity make it easier to gain control over them.

But the fact of change raises the complex issue of classification of these children. Suppose that two children who were classified as inhibited and uninhibited at 2 years of age were identical in their profile of behavior and physiology when they were 5½ years old. We have a few children who fit these criteria. Should we classify the two older children as belonging to the same psychological category because their profiles are identical, or should we assume that some aspects of their earlier profile are still present, but were not measured, and, therefore, treat the two children as psychologically different? Most evolutionary biologists favor the latter strategy, because both origins as well as current characteristics are used in taxonomy. However, this decision rests on the questionable assumption that some original qualities are always permanent or always produce distinct derivatives. At the moment, it is not possible to eliminate one of these perspectives.

Although scientists accept the fact of genetically based behavioral and physiological differences among strains of animals, they have been slightly more reluctant to acknowledge these processes in humans, with the two notable exceptions of the qualities that are called *intelligence* and *psychosis*. One reason for this reluctance comes from our egalitarian ethic that is committed to equal opportunity for status and economic security among all citizens. This political view resists awarding undue weight to differences present at birth that might make it difficult for some children to attain the prizes valued by the society. However, the data we have summarized suggest that fearfulness and timidity are malleable characteristics. Furthermore, there are adaptive aspects to inhibited behavior in an industrialized society. Extremely inhibited children avoid peer groups and, therefore, are likely to invest more effort in school work if they have parents who promote academic mastery. If they are successful in school, they may choose a career that involves intellectual work—such careers enjoy status and economic reward in modern societies. Finally, we note that each life is under the influence of many complex factors, including the person's will to change his or her psychological characteristics. Although some children have a biologically based disposition to be inhibited or uninhibited, it probably contributes no more than 10% to the variation in adult characteristics that our society currently values and that psychologists are able to measure.

Acknowledgments

This research was supported in part by the John D. and Catherine T. MacArthur Foundation and Grant MH 40619 from the National Institute of Mental Health, United States Public Health Service.

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CHAPTER 18

The Importance of Peer Relations

Martha Putallaz and Susanne E. Dunn

As many researchers have noted recently, the study of children's peer relations has not only had a long history but has been the focus of increasing attention as well (e.g., Asher, 1983; Hartup, 1983; Hymel & Rubin, 1985). For example, the proportion of published manuscripts concerning peer relations and social skills in one developmental journal reportedly doubled within the past decade to represent about 22% of the journal's content (Rubin, 1983). As Hartup (1983) put it, "Although the winds of change were detectable . . . , no one could have anticipated the surge of interest in peer relations that marked the 1970s (p. 106)." In addition to the marked increase in research in this area, the motivation for studying children's social status among their peers appears to have changed somewhat as well. Prior to the mid-1970s, researchers were preoccupied with such issues as improving the reliability of social status measurement techniques (e.g., Marshall & McCandless, 1957), examining the correlates of the trait of popularity (e.g., Marshall & McCandless, 1957; McGuire, 1973; Moore & Updegraff, 1964), or investigating the relation between peer reinforcement and social status (e.g., Hartup, Glazer, & Charlesworth, 1967). In contrast to today, little interest was exhibited in studying the long-term importance of peer relations in children's development (Hartup, 1983).

Several investigators have delineated possible reasons for the present change in research motivation and the concomitant surge of interest in children's peer relations. Rubin (1983) signaled out Hartup's (1970) classic chapter on peer interaction and social organization as a major force in the recognition of the peer group as providing a unique developmental context influencing the social and the cognitive growth of children. A second related factor for the increased attention to children's peer relations is associated with the decline in the emphasis of Freudian theory on the parent–child relationship that has allowed a recognition of peers, among others, as important socialization agents (Asher, 1983; Hartup, 1983; Hymel & Rubin, 1985). Third, societal changes have increased the potential opportunities available for interaction with peers and thus their relative influence in children's lives. Children are more likely to have earlier and more extended contact with peers, through such institutions as day-care and after-school programs, and to remain in school for more years than their predecessors (Asher, 1978; Hymel & Rubin, 1985). But perhaps the most critical factor sparking so much interest in this area has been the empirical evidence suggesting that there are negative consequences associated with having poor peer relations during childhood. Children who are not accepted socially by their peers appear to be at risk for a variety of negative consequences in later life, spanning such diverse realms as behavioral, psychological, and academic adjustment (Parker & Asher, 1987; Putallaz & Gottman, 1983).

Our purpose in this chapter is to review the research serving to support the predictive nature of

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children's peer relations as well as to attempt to delineate the possible reasons underlying this relation. Toward this end, we will (1) describe the sociometric questionnaire procedure briefly so as to familiarize the reader with the nature of the assessment data, (2) give a brief overview of the literature illustrating the predictive nature of peer relations, and (3) explore the potential explanations for such a relation.

Sociometric Measures

The majority of studies used to support the claim that inadequate peer relations in childhood are predictive of later maladjustment employ sociometric questionnaires as their assessment technique. Sociometric questionnaires assess the level of attraction or acceptance existing between the individuals of a particular group, such as between children in a classroom. The questionnaires assume primarily one of two forms: either a nomination format, requiring children to name, for example, those classmates they like most (i.e., positive nomination) or like least (i.e., negative nomination), or a rating format, requiring children to rate their classmates along some social dimension (e.g., how much they like to play with each child) (for further discussion, see Asher & Hymel, 1981; Gronlund, 1959; Hymel & Rubin, 1985).

Many studies included in the risk literature have obtained a measure of peer acceptance through such alternative sources as teacher ratings (e.g., Conger & Miller, 1966; Janes, Hesselbrock, Myers, & Penniman, 1979) or from ratings based on comments available in school or clinic records (e.g., Amble, 1967; M. Roff, 1961). As Parker and Asher (1987) have indicated, however, these measures of peer acceptance tend to have been developed *ad hoc,* not systematically, thus making the psychometric properties of such measures unclear. They may suffer from additional problems as well. For example, teacher rankings or ratings appear imperfectly related to peer sociometric measures and may be biased (Gronlund, 1959; Hymel & Rubin, 1985; La Greca, 1981). Further, comments made in clinic or school records are typically made idiosyncratically across settings and time, making standardized coding difficult. In addition, such archival records frequently do not contain obvious indices of the variables of interest; thus, measures less related to sociometric status may be used in their stead (Parker & Asher, 1987).

Finally, some of the risk literature studies have employed assessment questionnaires, such as the "Guess Who" (Hartshorne, May, & Maller, 1929) or the "Class Play" (Bower & Lambert, 1961), which require children to nominate classmates for a variety of positive and negative characteristics or roles. Asher and Hymel (1981) have argued that this type of measure is more accurately considered a peer assessment of behavior or reputation rather than as a sociometric measure of acceptance.

It is important to note that peer acceptance, as measured by sociometric questionnaires, is best conceptualized as an "indicator" variable in the sense that it leads to the search for some other set of variables that explain its variation. It is not a variable that can be objectively observed, like the amount of social interaction or degree of assertiveness. Thus, one problem that we face is that although sociometric measures have high indices of reliability and validity (Asher & Hymel, 1981; Gronlund, 1959), they do not provide any information about what the origins of children's social status might be or what factors might be maintaining it. However, as mentioned earlier, it does appear that indices of peer acceptance have utility in terms of the clinical identification of children at risk for the development of a variety of later problems. At this point, then, an overview of the literature illustrating the predictive nature of children's peer relations will be presented. The reader is directed to a more comprehensive review by Parker and Asher (1987) for greater detail.

Predictive Evidence of Peer Acceptance

Methodological Issues

In examining the wide scope of adolescent or adult difficulties predicted by low social status in the peer group, three general categories of adjustment difficulties emerge, namely, behavioral, psychological, and academic adjustment problems. Each of these outcome areas as they relate to peer acceptance in childhood will be explored in turn. However, before examining each of these areas, some methodological or design issues inherent in this type of research must be considered. Basically, two types of longitudinal designs are employed, namely, follow-back and follow-up (or follow-forward) studies. Follow-back designs identify a group of adolescents or adults presently experiencing adjustment problems, obtain a comparison control group of subjects who reportedly do not have

such problems, and then obtain childhood information on all subjects for the purpose of making comparisons on a variety of childhood variables. Follow-up designs concerned with childhood social status as a predictor variable examine children varying on peer acceptance, and then gather information on their later adolescent or adult adjustment.

As others (e.g., Garmezy, 1974; Parker & Asher, 1987) have pointed out, follow-back research has certain obvious advantages over follow-up research in that it is much quicker to execute because both a childhood and an adolescent or adult data base of information already exist. However, a disadvantage inherent in the follow-back design, and sometimes present in the follow-up design as well, is that the information has been collected for reasons (e.g., school or clinic record keeping) other than to examine the relation between childhood peer status and later adjustment; thus, imperfect measures of status are employed. A related problem with follow-back and a sample of follow-up designs is that biases may exist in the selection of the subject groups. Researchers using both types of designs are frequently forced to use a select group of children (e.g., clinic-referred children) because extensive childhood information is readily available only for such groups, thereby curtailing the potential generalizability of the findings.

Additionally, the results of follow-back studies are only suggestive of a connection between a childhood variable and later adjustment and cannot be used as evidence of a probabilistic predictive risk relation (Garmezy, 1974; Kohlberg, LaCrosse, & Ricks, 1972; Parker & Asher, 1987). Similarly, it is inappropriate to assume that the two types of studies yield similar results. Finding, for example, that the majority of adult schizophrenics experienced difficulty relating to peers during childhood does not necessarily mean that most children with low peer acceptance are likely to develop schizophrenia (Garmezy, 1974; Kohlberg *et al.*, 1972; Parker & Asher, 1987). Follow-back studies, then, provide information more appropriate for understanding the etiology of adult adjustment difficulties, than for evaluating the utility of certain predictor variables for identifying expected rates of later adjustment problems. For this reason, when researchers are trying to understand the predictive utility of a variable, such as peer acceptance, follow-up studies are generally preferred. However, the degree to which the results of the follow-back studies are problematic depends upon the relative importance of different sorts of errors. With this brief discussion of the methodological considerations of both follow-back and follow-up designs in mind, the association between childhood peer relations and later behavioral, psychological, and academic adjustment will now be explored.

Behavioral Adjustment

The available research examining childhood peer acceptance and later behavioral adjustment is suggestive that a relation between the two may exist. Follow-back studies have linked such behavioral outcomes as juvenile delinquency (Conger & Miller, 1966), postjuvenile criminality (M. Roff, 1977), and bad conduct discharges from the military (M. Roff, 1961) to poor childhood peer relations. These outcomes were found using different indices of peer acceptance, namely, teacher ratings of each child's "personal-social behavior" and coded teacher comments regarding the child's general strengths and difficulties (Conger & Miller, 1966), as well as blind evaluations as to "good or poor peer group adjustment" based on information obtained from clinical case histories (M. Roff, 1961). Two distinct samples were also involved: specifically, boys seen at child guidance clinics who later entered the military (M. Roff, 1961) and boys in a public school system for whom school records existed for kindergarten through tenth grade (Conger & Miller, 1966). A follow-back study by M. Roff (1977) offered a unique addition to these results in that the percentage of men exhibiting behavioral adjustment difficulties in adulthood who had been disliked by their childhood peers was explored. Using a peer acceptance score composed of peer nominations (both positive and negative) and teacher ratings of social status, Roff reported that about 80% of postjuvenile offenders had average or below average childhood peer status, with over half of this group being of below average status. These results are suggestive that a large percentage of postjuvenile offenders were disliked by their childhood peers.

Further support for the relation between low peer acceptance and later behavioral adjustment difficulties comes from several follow-up studies. Results of these studies indicated that children who had been identified as having difficulty getting along with their peers were more likely than control groups of their more accepted peers to become juvenile delinquents, as measured by police or juvenile court records (J. D. Roff & Wirt, 1984a; M. Roff, 1975; M. Roff, Sells, & Golden, 1972), or to evidence such behavioral problems in adulthood as

being fired for behavioral reasons, experiencing trouble with the law, using drugs, or receiving dishonorable discharges from military service (Janes *et al.*, 1979). The measures of peer acceptance used also varied across these studies and included positive and negative peer nominations (J. D. Roff & Wirt, 1984a; M. Roff, 1975; M. Roff *et al.*, 1972), a teacher behavior checklist item, "fails to get along with other children" (Janes *et al.*, 1979), and teacher interviews on each child's social adjustment (J. D. Roff & Wirt, 1984a). The samples of children employed included boys who visited a child guidance clinic (J. D. Roff & Wirt, 1984a) as well as an essentially unselected population, namely, third- through sixth-grade boys and girls in the public school systems of two midwestern cities (M. Roff, 1975; M. Roff *et al.*, 1972). As an indication of the magnitude of the relation between peer acceptance and later behavioral adjustment, Parker and Asher (1987) noted that data from M. Roff (1975) and M. Roff *et al.* (1972) indicated that low status children were 1½ to 2 times more likely to become delinquent before the age of 14 than their higher status peers, with the results being more dramatic for boys than for girls.

Although these follow-up studies offer evidence that peer acceptance may be predictive of later behavioral maladjustment, two additional follow-up studies reported only a suggestive (West & Farrington, 1973) or nonsignificant (Kupersmidt, 1983) relation. Parker and Asher (1987) suggested that West and Farrington (1973) may have found weak results because of the hypothetical nature of the wording they employed in their sociometric nomination question and because they did not employ any measure of disliking in their study (i.e., negative sociometric nominations). In terms of the Kupersmidt (1983) study, Parker and Asher (1987) pointed out that a comparatively small sample was involved and that the risk relation was not examined separately by sex. Further, they observed that Kupersmidt (1983) used a definition of delinquency that included less serious offenses (e.g., appearance in the juvenile police or court files) than previous researchers, potentially diluting any effects.

In summary, both follow-back and follow-up research offer support for low peer acceptance as a predictor of behavioral maladjustment in adolescence and adulthood. These findings include studies in which male and female subjects were used as well as peer and teacher ratings of social status. Additionally, clinic and school populations have been examined.

Psychological Adjustment

Numerous follow-back studies have identified a significant association between poor childhood peer relations and such later psychological difficulties as psychosis (Friedlander, 1945; M. Roff, 1963), neurosis (M. Roff, 1957, 1960), and schizophrenia (Fleming & Ricks, 1970; Frazee, 1953; Ricks & Berry, 1970; J. D. Roff, Knight, & Wertheim, 1976). In the above studies, clinic samples were employed and either teacher or therapist comments found in child guidance clinic records were analyzed to assess peer acceptance. In addition to the psychological adjustment outcomes listed above, more general measures of global mental health in the later years have been examined for their relation to low peer acceptance in childhood. In the frequently cited study by Cowen, Pederson, Babigian, Izzo, and Trost (1973), individuals whose names appeared on a comprehensive psychiatric register were found to have had more negative peer nominations (as measured by the Class Play) in third grade than a comparison group of individuals whose names were not found on the register. This finding was particularly striking as negative peer status emerged from a multitude of other childhood variables (e.g., academic performance, self-esteem, teacher ratings) as a strong predictor of later psychological adjustment difficulties.

In contrast, the follow-up studies that have examined peer acceptance and later psychopathology have provided mixed support for the usefulness of childhood peer relations as a predictor of later mental health difficulties. In two instances in which nonrandomly selected populations of children (i.e., clinic children) were studied, conflicting results were found. In one case, children who had difficulty getting along with their peers (based on childhood clinic records supplemented by information in police, court, and school records) did not evidence any greater incidence of psychopathology in adulthood than did a group of more popular peers (Robins, 1966). In the other case, significantly poorer mental health ratings were found for children identified by teachers as having trouble getting along with their peers when compared to children who did not show such difficulties (Janes & Hesselbrock, 1978; Janes *et al.*, 1979). One difference between the two studies was that Robins examined more specific types of psychopathology, namely, psychosis, neurosis, and alcoholism, than did Janes and colleagues who used a more global measure of mental health difficulties, specifically, interviewers' ratings of general psychological ad-

justment and subsequent psychiatric hospitalization. Another difference is that different measures of peer acceptance were used in the two studies. An additional study by J. D. Roff and Wirt (1984b) also examined the relation between receiving mental health treatment and peer acceptance, but in a more random sample of children. J. D. Roff and Wirt (1984b) reported a modest association between later general maladjustment and childhood sociometric status, as measured by positive and negative peer nominations. Their findings may have been modest, as Parker and Asher (1987) noted, because unpopular children were not treated as a separate status group statistically, but rather sociometric scores were correlated with the later measure of psychological adjustment.

In summary, studies examining the association between childhood peer status and later mental health difficulties are mixed, with follow-back research offering more consistent support than follow-up research. It is not surprising that follow-up studies reveal lower relations than do follow-back studies because a high base-rate variable (i.e., low peer acceptance) is predicting to a low base-rate variable (i.e., psychological disorder) in that type of study.

Academic Adjustment

Numerous follow-back studies examining academic adjustment, in the form of voluntary withdrawal from high school, have found significantly more problematic peer relations in the childhood histories of dropouts than nondropouts (Amble, 1967; Bowman & Matthews, 1963; Kuhlen & Collister, 1952; Ullman, 1957). Although all these studies employed school children as subjects, the manner in which peer acceptance was assessed varied, with peer ratings (Ullman, 1957), peer nominations (Bowman & Matthews, 1963; Kuhlen & Collister, 1952), and teacher ratings (Amble, 1967; Bowman & Matthews, 1963; Ullman, 1957) all being used.

Although dropping out of school has been the most heavily researched academic adjustment variable, other outcomes have been examined by follow-back researchers. Ullman (1957) found that attainment of honor role status in eleventh grade related to both peer and teacher ratings of social status in ninth grade. Lambert (1972) examined the relation between children's social status, as measured by the Class Play, and two composite academic adjustment variables, namely, school scholarship (composed of weighted grade point average and math and reading achievement scores) and a "lack of success" index (derived from disciplinary reports, remedial instruction, probationary academic standing, and dropping out of school). Lambert found poor academic adjustment in the ninth and twelfth grades, as measured by both composite indices, to be associated with low peer acceptance seven years earlier. Thus, although the majority of research on the relation between peer status and academic adjustment has concerned dropping out of school, other indicators of educational adjustment appear related as well.

Two follow-up studies support the predictive utility of peer status in identifying children who will exhibit early school withdrawal. Based on positive peer nominations made in sixth grade, Gronlund and Holmlund (1958) found low-status children of both sexes to be more likely than high-status children to withdraw from school prematurely (i.e., 54% vs. 19% for boys, and 35% vs. 4% for girls). Barclay (1966) found children with poor peer relations according to peer and teacher assessments to be two to three times more likely to drop out of school than other children. Finally, Janes *et al.* (1979) found that 25% of a clinic sample of boys identified by teachers as experiencing peer relation problems dropped out of school in comparison to 12.6% of the children not so identified.

Kupersmidt (1983) provided unique information regarding the utility of subclassifications of low sociometric status based on both positive and negative nominations in the prediction of later academic adjustment difficulties. At the time of a 7-year follow-up, children who had been actively disliked or rejected by their peers in fifth grade appeared more likely than children who had been ignored or neglected socially by their peers to become school dropouts (30% vs. 10%), to be retained in grade at least once (45% vs. 25%), but appeared to be indistinguishable in terms of truant behavior defined as unexcused absences (55% vs. 50%).

Thus, follow-up research supports the findings from follow-back research that low social status may be predictive of later academic maladjustment. There is some evidence that it may be useful to distinguish actively disliked children from children who are ignored by their peers in the prediction of later academic adjustment problems.

Concomitant Adjustment

Although thus far we have only described the predictive relation between the adequacy of children's early peer relations and their later adjust-

ment, there is also evidence suggesting that sociometric status is related concurrently to early adjustment in children as well. The majority of work in this vein has concerned the relation between sociometric status and behavioral adjustment. It appears, for example, that in comparison to others, low social status children (as defined using a variety of peer sociometric criteria) have been observed to be more aggressive (Coie & Kupersmidt, 1983; Dodge, 1983; Dodge, Coie, & Brakke, 1982; Ladd, 1983), less likely to display prosocial behavior (e.g., Ladd, 1983; Rubin & Daniels-Beirness, 1983), involved in more negative interactions with peers and teachers (Hartup *et al.*, 1967; Ladd, 1983; Rubin & Daniels-Beirness, 1983; Vosk, Forehand, Parker, & Rickard, 1982), more disagreeable (Putallaz, 1983; Putallaz & Gottman, 1981), more disruptive (Coie & Kupersmidt, 1983; Putallaz & Gottman, 1981), more apt to be off-task (Coie & Kupersmidt, 1983; Gottman, 1977), and more often described by teachers as having conduct problems (Putallaz, White, & Shipman, 1985; Rubin & Daniels-Beirness, 1983; Vosk *et al.*, 1982). Thus, there appears to be a clear negative association between peer acceptance and behavioral adjustment problems.

In terms of psychological adjustment, low social status children have been reported to be more lonely (Asher, Hymel, & Renshaw, 1984; Asher & Wheeler, 1985; Hymel, 1983), more depressed (Putallaz *et al.*, 1985; Vosk *et al.*, 1982), and to have less self-esteem (Baron, 1951; Coopersmith, 1959; Guardo, 1969; Horowitz, 1962; Putallaz *et al.*, 1985) than their more socially accepted peers. Some researchers have also found these children to be more anxious than other children (e.g., Horowitz, 1962; McCandless, Castaneda, & Palermo, 1956; Trent, 1957), although this relation appears more inconsistent (Cowen *et al.*, 1973). Therefore, it seems that the concomitant relation between social status and behavioral adjustment holds for psychological adjustment as well.

Finally, with respect to academic adjustment, lower social status children have been found to perform more poorly on achievement tests (Green, Forehand, Beck, & Vosk, 1980; Putallaz, 1983; Putallaz *et al.*, 1985) and to receive lower marks in school (Putallaz *et al.*, 1985; M. Roff *et al.*, 1972) than their higher status peers. Further, certain specific academic abilities, such as arithmetic ability and reading skill, have also been related to social status (Davis, 1957; McMichael, 1980).

Thus, there does appear to be evidence that sociometric status is related to concurrent adjustment in children, as well as predictive of later problems. Again, a broad range of concomitant negative outcomes was associated with low peer acceptance.

Summary

Even from the brief overview of the risk literature just presented as well as from more comprehensive reports (e.g., Parker & Asher, 1987), it is clear that there is considerable evidence that children not accepted socially by their peers may be a population at risk for the development of a wide variety of negative consequences. Peer acceptance during childhood appears to be associated with future as well as concomitant functioning across such diverse realms as behavioral, psychological, and academic adjustment. Clearly, the studies comprising the risk literature cannot be considered conclusive as they are correlational in nature. It is impossible, for example, to determine the direction of causation in the apparent relation or even to rule out the possibility that some third factor is determining both peer acceptance and the risk variable of interest. Hartup (1983) and Asher (1978) have proposed family problems and relations to be one such variable. Indeed, a recent direction taken in this research field has been to attempt to delineate the manner in which interactional patterns and socialization styles within the family affect children's peer relations (MacDonald & Parke, 1984; Putallaz, 1987; Putallaz & Heflin, in press; Rubin, 1983). However, the sheer wealth of studies and the variety of risk criteria employed are impressive and quite provocative evidence that socially unaccepted children may constitute a population at risk.

Parker and Asher (1987) have added several qualifications to this broad conclusion. First, they pointed out that the evidence appears stronger for the prediction of educational and behavioral adjustment than for psychological adjustment. In fact, the higher incidence rates of behavioral and academic problems should make prediction of these outcomes easier than the prediction of psychological problems. However, they stressed that the difference may also indicate that early peer relations are less predictive of the types of broad mental health outcomes examined thus far. Second, Parker and Asher underscored the fact that, in terms of prediction, indices of peer relationship problems tend to make many false positive errors (although few false negative ones), thus identifying many children not actually at risk. As they pointed out, however, fears of overselection are diminished if any planned intervention includes all children, thereby avoiding the

iatrogenic effects of labeling. Of course, if the degree of overprediction is high, such interventions can be quite expensive. Finally, they identified peer acceptance (particularly indices of rejection) and aggression to better predictors of later adjustment difficulties than shy or withdrawn behavior. Along a similar vein, many researchers (Asher, 1985; Coie, 1985) have recently advocated differentiating rejected children (children actively disliked by their peers) from neglected children (children who are ignored by their peers), and considering the former group as the population at risk rather than the latter group (although see Hymel & Rubin, 1985, for an alternative view).

Explanations for the Risk Relation

If peer relations during childhood are related to later adjustment, it is unclear what the nature of the causal link might be. What is it about not being liked by peers that leads to later difficulties? In other words, we do not know what variables intervene between poor peer relations and negative consequences. Earlier we referred to sociometric status as an indicator variable, not a behaviorally observable variable in itself. Such information is clearly necessary for the development of a theory of social acceptance among children as well as the development of effective interventions.

Some of the authors whose work have led to the premise that low accepted children may be at risk for the development of future problems have speculated on this issue, as have some researchers working in the field of peer relations in general. However, as others (Parker & Asher, 1987; Putallaz & Gottman, 1983) have pointed out, the type of rigorous, process-oriented research necessary to advance our understanding of the potential causal link between peer acceptance and later adjustment has not yet been conducted. Thus, it remains unclear why early peer acceptance would be predictive of such a diverse set of outcomes, let alone under what circumstances it would predict to a particular outcome. Therefore, the explanations that will be presented at this point are merely speculations still in need of rigorous testing, and not yet at a stage meriting an evaluation of their comparative strengths and weaknesses.

One view held is that difficulty in establishing adequate childhood peer relations may be indicative that precursors of later maladjustment are present in the child (Kohlberg *et al.*, 1972). Thus, peer rejection is construed as a correlated lead indicator of other difficulties a child will develop (experience) in the future. In fact, it has even been suggested that peer acceptance might at some point in time be used in the prediction of specific outcomes. As Kohlberg *et al.* (1972) wrote,

> Undifferentiated peer problems seem relatively promising as a nonspecific predictor of later adjustment difficulties. If the particular forms and temporal sequences of peer difficulties are examined, particular kinds of outcome may be predictable. (p. 1262)

A second explanation (Cowen *et al.*, 1973; Vosk *et al.*, 1982) for the relation between early peer relations and later adjustment similarly views the child as coming to the peer group as vulnerable, but adds that the peer group exacerbates this vulnerability, thereby increasing the likelihood that the child will develop future problems. Thus, childhood peer relations act not as an indicator but as a multiplier of early vulnerability. According to Cowen *et al.* (1973),

> The data suggest both that young children identify troubled peers early and that the identified ones are, in some way, aware of these perceptions. . . . If peers so type and behave toward already vulnerable youngsters, a process is in motion whereby the identified ones may become increasingly isolated and "tabbed" by cohorts—a process that can actively exacerbate early difficulties and increase the probability of later, more serious psychiatric problems. (p. 445)

A third possible explanation for the risk relation posits inadequate peer relations as a direct cause of the development of future difficulty. Inadequate socialization resulting from poor peer relations has been suggested as one possible causal avenue for the risk relation. Hartup (1970, 1978, 1983) has argued that there are certain behaviors and norms that can only be appropriately learned in the peer context. For example, peers appear to affect children's development by facilitating the management of aggressive and sexual behavior (Hartup, 1978, 1983), the learning of complex forms of play (Gottman, 1983), the emergence of such interpersonal skills as mutual self-disclosure (Buhrmester & Furman, 1986; Gottman, 1983), and the development of "social intelligence" (Hartup, 1978, 1983). In this same vein, several researchers have observed the tendency of low-status children to interact primarily with younger children rather than their own agemates (Ladd, 1983; Rubin & Daniels-Beirness, 1983) and to engage in more solitary, inappropriate play than others (Coie & Kupersmidt, 1983; Dodge, 1983). This type of peer experience

may lead to future difficulties by limiting the opportunities available to low-status children to learn the knowledge, norms, and skills necessary to interact effectively in larger social contexts and to observe and practice age-appropriate behaviors (Ladd, 1983).

Interestingly, the literature on adult friendship offers another potential avenue by which poor childhood peer relations may cause later difficulties that has been relatively unexplored with children. There is a growing body of social epidemiological literature on adults that suggests that the social support derived from a close, reciprocally confiding relationship may moderate or provide protection from the negative effects of life stress. The two models that have been proposed to explain this association and the conditions under which each holds have been delineated recently in a comprehensive review by Cohen and Wills (1985). The first model, referred to as the general model or additive model, holds that having little or no social support is in and of itself stressful, and thus social support is beneficial regardless of the incidence of stressful events. The second model, referred to as the buffering model, posits that a low level of social support is not stressful by itself and is only related to maladjustment when people are experiencing stressful circumstances. In this case, social support is construed as serving a protective or buffering function in preventing adjustment problems from occurring as a result of the stress. It is possible that a similar causal explanation may be operating with respect to children and that poor peer relations connote inadequate social support and thus a vulnerability to subsequent risks.

It is also possible, however, that it is the experience of being disliked or rejected by one's peers that may lead to future difficulties. In their impressive study of behaviors associated with the emergence of social status, Coie and Kupersmidt (1983) observed that the tendency of rejected children to engage in solitary, inappropriate play followed, not preceded, their rejection within the group. Another instance of how the experience of being rejected may affect children adversely has been suggested by Parker and Asher (1987). Clearly, some children perform poorly in school for a variety of reasons unrelated to their peers (e.g., lower intelligence, less emphasis on academic success at home). However, Parker and Asher pointed out that being rejected may lead children to do poorly academically because they may miss out on informal opportunities to learn school material (e.g., doing homework or studying for tests together). Further, the aversiveness of rejection alone may be sufficient reason to drop out of school because differences in drop-out rates have been found without differences in achievement (Janes *et al.*, 1979; Kuhlen & Collister, 1952).

Thus, in summary, three general perspectives or explanations have been proposed concerning the relation between childhood peer relations and adjustment. According to the first perspective, peer acceptance is not causally related to adjustment but serves as an indicator of later maladjustment because of its relation to some third variable. The second perspective assumes an interactional model and views early peer acceptance as interacting with some third variable to determine level of adjustment. The final perspective hypothesizes that peer acceptance is causally related to adjustment either directly or through some intervening variable. Thus, three models are represented by these perspectives: specifically, an indicator model, an interaction model, and a causal model.

To address the relative validity of these models for behavioral, psychological, and academic adjustment is of clear importance, because each model has very different implications for intervention. For example, causal models suggest direct intervention to increase the level of peer acceptance, whereas interaction models suggest either reducing the level of initial risk or increasing the level of peer acceptance. An assessment of the validity of these models represents a critical direction for research in this field. For the moment, what we do know is that such research is necessary, as peer acceptance and adjustment are related. Exactly how is the next question to be addressed.

ACKNOWLEDGMENTS

The authors would like to acknowledge the support of a William T. Grant Faculty Scholar Award to the first author and to thank Blair Sheppard for his comments made on an earlier version of this chapter.

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CHAPTER 19

Test Anxiety in the School Setting

Allan Wigfield and Jacquelynne S. Eccles

The impact of test anxiety on school performance and learning has interested educators and motivational psychologists for at least the last 50 years. Numerous studies have demonstrated the negative impact of anxiety on school performance (e.g., K. T. Hill & S. B. Sarason, 1966; Manley & Rosemier, 1972; S. B. Sarason, Davidson, Lighthall, Waite, & Ruebush, 1960). Furthermore, K. T. Hill (1980, 1984) suggested that as many as ten million students in elementary and secondary schools perform more poorly on tests than they should because anxiety and deficiencies in test-taking strategies interfere with their performance. In addition, the strength of this negative relationship becomes stronger over the school years. In this chapter, we begin by defining test anxiety and discussing theoretical approaches that have been used to explain it. The major portion of our discussion focuses on the development of anxiety in school settings, and on intervention programs that have tried to alleviate the school performance problems associated with test anxiety.

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Conceptualizing Test Anxiety

Initially, test anxiety was defined in motivational and personality terms. For example, Atkinson (1964) equated test anxiety with the motive to avoid failure. Anxious individuals were said to be more concerned or motivated to avoid failure than to approach success. Atkinson predicted that individuals high in the motive to avoid failure would choose either very easy tasks in which success was very likely, or very hard tasks in which failure would not reflect on the individual's ability. S. B. Sarason *et al.* (1960) conceptualized test anxiety as a relatively stable personality trait that develops when parents hold overly high expectations and are overly critical of their children's achievement efforts. Integrating the approaches of Atkinson and S. B. Sarason *et al.*, K. T. Hill (1972) defined test anxiety in terms of the motivational dispositions to obtain praise, avoid criticism, approach success, and avoid failure. Like S. B. Sarason *et al.*, K. T. Hill believed that overly demanding and critical parents fostered the development of test anxiety in their children. Hill argued that anxious children generally are more aroused than low anxious children, and strive to avoid criticism and failure relatively more than to obtain praise and approach success, because of their greater sensitivity to adults' negative reactions.

Spielberger (1966) made the important distinction between trait and state anxiety, with trait

anxiety defined as a relatively stable disposition toward experiencing anxiety in evaluative settings, and state anxiety defined as a more transitory anxious reaction to particular evaluative situations. This distinction is critical because anxiety varies across situations (see Richardson & Suinn, 1972). Most developmental studies of test anxiety have ignored this distinction, even though it is very likely some children become anxious in most evaluative situations, whereas others may be anxious only in certain kinds of evaluative situations.

In addition to this state–trait distinction, other theorists have begun to identify different components of anxiety. Liebert and Morris (1967) distinguished between worry, the cognitive component of anxiety, and emotionality, the more physiological/affective part of anxiety (i.e., nervousness and tension). According to Liebert and Morris, worry is related to the cognitive and attentional cues associated with evaluation and with failure. In contrast, emotionality is a classically conditioned autonomic/affective reaction to cues associated with evaluation. Morris and Liebert (1973) have shown that these two components are empirically distinct (though correlated), and also that worry relates more strongly (and negatively) to test performance than does emotionality (see Deffenbacher, 1980, and Morris, Davis, & Hutchings, 1981, for reviews of the work on worry and emotionality).

Similar though more differentiated factors have emerged in studies with children. For example, both Dunn (1964, 1965) and Feld and Lewis (1969) studied anxiety in elementary school aged children, using S. B. Sarason *et al.*'s (1960) Test Anxiety Scale for Children (TASC).[1] They each found four distinct (but correlated) factors or components of anxiety, and these factors were conceptually quite similar across the studies. Based on the content of item loading most heavily on each factor, Feld and Lewis labeled these factors Test Anxiety, Poor Self-Evaluation, Somatic Signs of Anxiety, and Remote School Concerns (listed in order of variance accounted for). In subsequent analyses, Feld and Lewis found that the scale based on the Poor Self-Evaluation factor related more closely to school performance than did scales based on the other factors.

In another factor-analytic study of children's anxiety, Nicholls (1976) revised the TASC by rewording some items in order to better distinguish between items assessing poor self-evaluation and items assessing test anxiety, and dropping the Remote School Concerns scale. Factor analysis of sixth-grade children's responses to this new scale produced Poor Self-Evaluation, Test Anxiety, and Somatic Signs of Anxiety factors like those of Feld and Lewis as well as an additional factor for boys and girls (labeled Pleasure in Testing Situations) and one for girls only (labeled Effectiveness of Effort). As found by Feld and Lewis, the Poor Self-Evaluation factor related more strongly to children's achievement test scores than did the other factors.

The factors in these four studies are similar to the two components identified by Liebert and Morris (1967). Worry is similar to test anxiety and to poor self-evaluation in that they all refer to concern over performance, whether one's ability is adequate for the task, and are cognitive assessments. Somatic signs of anxiety and emotionality are similar; both refer to the physiological/affective aspects of anxiety. Furthermore, across all the studies, the cognitive component related most strongly to performance.

Theorists increasingly have been interested in understanding the cognitive component of test anxiety. Wine (1971, 1980) put forth a cognitive-attentional model of anxiety, arguing that the performance differences between high and low anxious individuals reflect differences in their attentional strategies: Low anxious individuals maintain their focus on the task at hand while they are being evaluated, whereas high anxious individuals divide their attention between the task and their ruminations about how they are doing. These ruminations, she suggested, cause high anxious individuals to concentrate less on task performance; consequently, they do less well. Similar hypotheses have been advanced by others (e.g., Geen, 1980; Mueller, 1980; I. G. Sarason, 1980, 1986; Tobias, 1980, 1986).

Evidence has supported this model. High anxious adults have more task-irrelevant thoughts than low anxious adults while they are doing evaluative tasks (Mandler & Watson, 1966; I. G. Sarason & Stoops, 1978), and their task-irrelevant thoughts often center on negative personal characteristics (Doris & S. B. Sarason, 1955; I. G. Sarason &

[1] The TASC and its companion, the Lie Scale for Children (LSC), a defensiveness measure, have been the scales most frequently used in developmental studies of anxiety. Because of the theoretical advances in conceptualizing anxiety, new scales for children need to be developed in this area. Because of space limitations, we will not discuss measurement issues here (see Wine, 1980).

Glanzer, 1962, 1963; I. G. Sarason & Koenig, 1965). Such task-irrelevant, self-deprecatory thinking is especially likely when tasks are described as ability assessments (I. G. Sarason, 1973, 1975). High anxious children also have been shown to attend less well to critical information needed to do problem-solving tasks (Dusek, Kermis, & Mergler, 1975; Dusek, Mergler, & Kermis, 1976), and to engage in more off-task behavior during problem solving (Nottlemann & K. T. Hill, 1977).

These various conceptualizations of test anxiety provide a richer understanding of the construct. It is now assumed that anxiety is a multidimensional construct that has both cognitive and affective/physiological components. The cognitive component consists of task-irrelevant, self-deprecatory thoughts and worries that reduce task-focused attention and so interfere with performance. The affective/physiological component consists of heightened arousal, feelings of nervousness and tension, and other somatic signs. The cognitive aspects of anxiety relate more strongly to test performance than do the affective/physiological aspects.

Although more recent conceptualizations provide us with a richer understanding of the anxiety construct, several important issues remain open. First, the state–trait distinction has not received the attention it deserves, and many researchers implicitly assume anxiety is traitlike. We know little about whether the characteristics of state and trait anxiety are similar or different. Further, different components of anxiety may be more statelike or more traitlike. Deffenbacher (1980), discussing worry and emotionality, cautioned that even though studies of state anxiety show worry and emotionality to be separable constructs, researchers should not conclude they are separate components of trait anxiety. More generally, anxiety may best be conceptualized as a state that is aroused under certain conditions, with individual differences in the arousal tendency. We will discuss conditions that elicit anxiety later.

Second, there has been much debate about whether anxiety causes poor performance, or whether poor performance leads to anxiety. We present evidence in later sections that supports each of these views; for instance, some work shows that changing the testing conditions allows high anxious children to perform better, indicating that anxiety can cause performance problems. Other work shows that repeated failure leads to anxiety; hence, poor performance can cause anxiety. We will return to this issue.

Third, individual difference variables need to be considered more fully. We will discuss gender differences in anxiety later. Anxiety also should vary in students of differing abilities. Because anxiety is associated with low-ability perceptions and negatively related to performance, less able students should be more anxious in evaluative situations. However, many bright, capable students also experience anxiety, due to feeling too much pressure (either self-imposed or other-imposed) to perform well. These students even may believe they are doing poorly if they are not outperforming most other capable students. We believe the anxiety dynamics for these two kinds of students are quite different: One group is anxious because they cannot do the work in school; in contrast, the other group is capable of doing the work, but is anxious for other reasons, and so performs less well. We will return to this issue in the section on anxiety interventions.

Fourth, there has been concern about the distinctiveness of anxiety as a construct. Nicholls (1976) showed that individual items on the TASC confound anxiety and low self-perceptions of ability and so argued that, when that scale is used, it is difficult to determine which construct is being measured. Fennema and Sherman (1976) argued that anxiety may best be conceptualized as a lack of confidence (or poor self-evaluation) in one's ability, because in their work measures of the two constructs correlate very strongly. Perusal of other scales used to measure anxiety shows that many items on those scales tap individuals' beliefs that they lack ability. Is anxiety distinct from negative ability perceptions? Certainly, it appears that Liebert and Morris's Emotionality component of anxiety is distinct from ability perceptions, as is Feld and Lewis's Somatic Signs of Anxiety factor. Both of these refer much more to physiological reactions to evaluation and stress. As we have seen, however, this component of anxiety relates less strongly to test performance than the worry component. And the worry component does seem very intertwined with negative self-evaluations of ability, at least as it has been measured. We would argue that although perceptions of ability may lead to the worry component of anxiety, it is useful to maintain the distinction between the two constructs, particularly because low self-evaluations do not always lead to anxiety; there are children who lack confidence in their ability, yet they are not anxious. Nonetheless, even if ability perceptions precede anxiety in causal sequence, the relation be-

tween the two probably becomes cyclical by the middle elementary school years.

The Development of Test Anxiety

Several theorists (e.g., Dusek, 1980; K. T. Hill, 1972; S. B. Sarason *et al.*, 1960) have proposed that anxiety first emerges during the preschool years, when parents hold overly high expectations for their children and criticize them when they do not satisfy those expectations. This claim, though seemingly well-accepted, has never been assessed adequately. Furthermore, it seems to us that, although parents may create the propensity for children to be anxious, the different kinds of evaluations children experience as they progress through school should exacerbate this propensity in those with it and induce anxiety in other children who face repeated failures. Therefore, both family and school experiences may have important causal influences on the ontogeny of anxiety.

Researchers have assessed more thoroughly the development of test anxiety during the elementary and secondary school years. K. T. Hill and Sarason (1966) conducted the most extensive longitudinal study to date, examining (with the TASC and the LSC) the development of test anxiety across the elementary school years in a sample of 700 mostly white, lower-middle-class children. Children's total anxiety scores increased over time. The negative correlations between anxiety total scores and achievement test scores increased as well, from a negligible level in Grades 1 and 2, to between $-.20$ and $-.25$ at Grades 3 and 4, and in some groups to $-.44$ in fifth and sixth grades.

Other studies suggest that the magnitude of the negative correlations between anxiety and achievement test performance continue to increase over the middle school years (Fyans, 1979; Willig, Harnisch, Hill, & Maehr, 1983). Anxiety scores also become more reliable as children get older (Manley & Rosemier, 1972).

Gender differences have emerged fairly consistently in developmental studies. For example, K. T. Hill and Sarason (1966) found that by the third grade girls' mean TASC scores were higher than boys' scores, but boys' LSC scores were relatively higher than those of girls. Others also have found that girls' anxiety scores are higher than boys' scores (Manley & Rosemier, 1972; Meece, 1981).

K. T. Hill (1972) argued that these gender differences, particularly the discrepancies between TASC and LSC scores, are due to boys' greater defensiveness about admitting anxiety, and concluded that the experience of anxiety is the same for boys and girls. We think this issue deserves closer scrutiny. Boys and girls may become anxious for different reasons, or be anxious about different things. For instance, there is evidence that girls are more sensitive about social approval from adults than are boys (Dweck & Bush, 1976; Maehr & Nicholls, 1980), and so perhaps their anxiety reflects that kind of concern. Boys seem more sensitive to peer evaluation (Dweck & Bush, 1976). Boys' and girls' different interests may play a role too; as they go through school, their interests change, and so it seems likely their concerns about evaluation will also change. Girls may come under more pressure to conform to feminine stereotypes during junior high (J. P. Hill & Lynch, 1983), and at this time may also become more concerned with social activities and popularity than do boys. As a result, their apprehensions may be different. In particular, with regard to math, girls may stereotype math skills as masculine and consequently assume that difficulty reflects lack of talent; alternatively, girls may become concerned about how math interferes with other activities, or may feel some pressure against excelling in math, perhaps leading them to be anxious both about success and failure in math. Boys may be pushed to excell in math, and so become anxious if they do poorly. Such differences may be further complicated by differences in student capabilities across various subject areas. Consequently, even if boys and girls have equivalent propensities for trait and/or state anxiety, it seems quite probable that this propensity will manifest itself differently in boys and in girls.

Because the studies tracing the development of anxiety were descriptive and correlational, we do not know whether higher anxiety scores and stronger negative correlations between anxiety and school performance really do mean that anxiety is increasing, or are a result of children becoming more reliable at answering questions about anxiety. We need studies going beyond these initial correlational studies to better understand the development of anxiety and the processes that relate to anxiety.

Several key issues need to be investigated, beginning with how parental reactions to preschool children's performance in achievement situations shape children's later reactions to being evaluated in school. We also need to assess how the experience of anxiety changes over time. We have seen that different components of anxiety can be identified in school-aged children. How these different components develop has not been charted adequately. We believe children's anxiety first may be

characterized more by affective responses to failure experiences, and later by the more cognitive worry responses. Young children are not overly self-reflective, and so it is doubtful they would engage in the self-preoccupied thought patterns associated with worry. Instead, young children may simply be more easily distracted by failure and may spend more time off-task (see Nottlemann & K. T. Hill, 1977). As their ability perceptions develop and particularly if they come to view ability as a stable entity over which they have little control (Dweck & Bempechat, 1983), children should become increasingly worried about performing poorly. Studies of the thought processes of low and high anxious children as they solve complex tasks (like those of Diener & Dweck, 1978, on the thought processes of learned helpless and mastery oriented children) would be very useful. We know nothing about how these thought processes change over age. More is known about how children process evaluative feedback, and so we turn next to how such processing relates to anxiety.

Children's Processing of Evaluative Feedback

Assuming that anxiety does increase during the elementary school years, it seems likely that children's developing prowess at processing the evaluative information they receive in school contributes to the increase. During the early elementary school years, children receive a great deal of information about their school-related abilities. Initially, children are very positive about how well they will do in school (Stipek, 1984), even if they actually are doing poorly (Entwisle & Hayduk, 1978, 1982). However, during the middle elementary school years, children become increasingly capable of processing and interpreting evaluative feedback from teachers, and comparing their performance to that of other children (see Ruble, 1983), and so develop clearer ideas of their relative standing (in terms of ability) in the classroom (Nicholls, 1979a). They also understand better how their success and failure experiences relate to their future performance possibilities (Parsons & Ruble, 1977; Rholes, Blackwell, Jordan, & Walters, 1980), and their perceptions or ability become more differentiated (see Nicholls, 1984). Further, as children get older, many change their view that ability is malleable to the belief that ability is a stable entity that they cannot change (see Dweck & Bempechat, 1983).

But how might patterns of success and failure experiences, children's beliefs about their ability, and their comparisons of their performance with that of other children relate to anxiety? In K. T. Hill's (1972) view, children who succeed on most school tasks, and so receive positive evaluations from adults, will approach new evaluative situations with confidence, and will be more motivated to approach success than to avoid failure. In contrast, children who experience more failure and/or more critical reactions from adults will become anxious about evaluation, and so be more motivated to avoid failure than to approach success, especially when the evaluation component of the testing is made salient.

Several studies document the negative impact repeated failure experiences have on children's perceptions of their ability (see Dweck & Goetz, 1978). Repeated failure experiences also should elicit anxiety, particularly when children believe that poor performance signifies something negative about them, such as lack of ability. Covington (1983, 1986) argued that students who experience continuing failure in school while still trying inevitably must begin to attribute those failures to lack of ability (see Covington, 1983; Covington & Beery, 1976; Weiner, 1979). Attributing failure to lack of ability produces negative affect, such as shame and humiliation (Covington & Omelich, 1979), and, eventually, anxiety. Covington (1986) showed, using path analysis, that perceptions of low ability do indeed lead to anxiety reactions in evaluative settings. However, doing poorly should not elicit anxiety reactions in the following situations: when tests or school tasks are not defined as measures of ability; if the child does not conceptualize ability as a stable entity of the person; and if the child is not prone to attributing failure to lack of ability. These hypotheses need to be tested.

Social comparison processes may also influence the development of anxiety, through their impact on children's ability perceptions. By comparing their performance to that of others in their classroom, children can define their relative standing in terms of ability. Children who believe they are competent relative to peers should feel more positive (and so less anxious) than those who believe they are less competent than their peers. Thus, there should be changes in anxiety as children's social comparison skills improve. The developmental patterns described earlier are certainly compatible with this prediction. We should find heightened anxiety in settings that make social comparison salient (see Eccles, Midgley, & Adler, 1984), and when changes in the nature of one's peer group lowers one's relative standing. Schwarzer and his

colleagues (R. Schwarzer & Lange, 1983; R. Schwarzer & C. Schwarzer, 1982) have assessed how changes in German students' comparison (or reference) groups influence their test anxiety. In Germany, all students are in heterogeneous classrooms until fifth grade, and then are tracked into high-, middle-, and low-ability schools. R. Schwarzer and C. Schwarzer (1982) assessed test anxiety in a group of students immediately after they made this transition, and in a different group of eighth-grade students who had been tracked for 3 years. R. Schwarzer and Lange (1983) assessed 3-year longitudinal changes in these groups' anxiety scores. Both studies assessed the hypothesis that students doing least well before tracking (and so tracked into the low track) should be most anxious at fifth grade, because their comparison group included many students doing much better than them. In eighth grade, with a more restricted comparison group, this group's anxiety should be lower. Conversely, the fifth-grade students doing best should be least anxious, because their comparison group included poor students, but more anxious at eighth grade because their comparison group then includes only the best students. Results of both the cross-sectional and longitudinal studies clearly supported these predictions.

The work just reviewed suggests that failure experiences, declining ability perceptions, changing views of the meaning of poor performance, developing social comparison skills, and changes in students' reference groups can be tied to the development of anxiety. As yet, however, no developmental study has assessed systematically how these processes relate to the development of anxiety. Based on the correlational work of K. T. Hill and I. G. Sarason and the work on the development of ability perceptions briefly reviewed, it seems that during the early elementary school years, children's failure experiences and ability perceptions would not relate to anxiety, because children remain optimistic after failure, their ability perceptions are relatively undifferentiated, and they basically see ability as an unstable characteristic. By the middle elementary school years when ability perceptions are more differentiated, more closely related to school performance, and are more entitylike, and when children are engaging in social comparison processes more, relations among the constructs should be moderate. Many children with low perceived ability should begin to be anxious in evaluative situations. These relations should increase during the later elementary school years.

School Evaluation and Anxiety

In school, evaluations by teachers, principals, and classmates occur regularly and frequently (Phillips, Pitcher, Worsham & Miller, 1980). The exact form of evaluation varies, and some evaluative practices create more anxiety than others. Furthermore, evaluations of academic performance become more salient in late elementary and secondary school, and classroom characteristics that increase social comparison become more common (see Eccles *et al.*, 1984).

Teachers may influence children's anxiety in a variety of ways. From the theoretical perspectives of S. B. Sarason *et al.* (1960) and K. T. Hill (1972), teachers who set overly high standards and/or criticize students too harshly should be more likely to foster anxiety in their students than other teachers. Classroom observation studies assessing this prediction have yielded mixed results (Zimmerman, 1970), with teacher criticism only weakly predicting anxiety, perhaps because teacher criticism is rarely focused on intellectual content (Blumenfeld, Hamilton, Bossert, Wessels, & Meece, 1983; Parsons, Adler, Futterman, Goff, Kaczala, Meece, & Midgley, 1983).

K. T. Hill (1976) suggested that high anxious children may interpret feedback from teachers differently than do low anxious children, because of their greater sensitivity to adult reaction. Furthermore, Phillips *et al.* (1980) and Parke (1976) suggested that teachers may react differently to high and to low anxious children, providing them with different kinds of feedback. For instance, if teachers are aware that some students are sensitive to criticism, they may try to criticize them less. This is an interesting suggestion, and points to the importance of conceptualizing teacher–student interaction as a bidirectional process. However, Helmke and Fend (1982) found that teacher ratings of their students' anxiety did not correlate with students' own anxiety ratings, and so teachers actually may not be good judges of their students' anxiety levels. Clearly, more process–product studies are needed to assess the relations between teacher criticism and student anxiety. The fact that in laboratory studies high anxious children do less well when their performance is observed (Cox, 1966, 1968) supports the idea that teacher monitoring behaviors may affect anxiety arousal. But demonstrating these effects in classroom settings may be difficult.

The most obvious kinds of evaluations students receive in school are grades and tests. Many

theorists have discussed how external evaluations or rewards (such as grades) have detrimental effects on intrinsic and continuing motivation (Deci & Ryan, 1980; Maehr, 1976; Maehr & Stallings, 1972). K. T. Hill (1977) and K. T. Hill and Wigfield (1984) have discussed how letter grades can promote a focus on ability perceptions, competition, social comparison, and negative self-evaluations, and so produce anxiety. Moreover, the use of a single letter grade for each subject means that teachers' evaluations of student effort, conduct, and achievement all end up reflected in the one grade. Additionally, parents may push children to attain high grades, and be critical if those standards are not met. Unfortunately, studies assessing the impact of various grading practices on anxiety are not available. K. T. Hill and Wigfield (1984) have recommended that letter grades be replaced (particularly in elementary school) with grading systems containing separate evaluations for student achievement, effort, conduct, and social development. They are currently testing the effect of such a change on levels of student anxiety. If the predicted relationship emerges, then changes in grading practices with grade level (see next section) would be one possible explanation for the grade-related increases in anxiety.

Standardized testing is another form of school evaluation that can both affect and be affected by anxiety. Studies conducted with adults and with children show that high anxious individuals perform less well than low anxious individuals when tasks are introduced as tests of ability (Barnard, Zimbardo, & S. B. Sarason, 1968; Lekarczyk & K. T. Hill, 1969; McCoy, 1965; I. G. Sarason, 1972), and many achievement tests are introduced in this fashion (K. T. Hill & Wigfield, 1984). K. T. Hill and Wigfield (1984) discussed the several other unique demands that school achievement tests place on children, all of which may elicit anxiety. These include overly difficult problems, because the same tests often are given at several different grade levels; complex and unfamiliar question and answer formats; and time limits. Time pressure may be the key factor: When laboratory tasks or actual school achievement tests are performed under time pressure, high anxious children do more poorly than low anxious children, but when time pressure is reduced, high anxious children's performance improves substantially (K. T. Hill & Eaton, 1977; K. T. Hill, Wigfield, & Plass, 1980; Plass & Hill, 1986).

These studies also have shown that testing conditions in school can be changed in ways that facilitate anxious children's performance. We will discuss the kinds of testing conditions that facilitate the performance of high anxious children in the section on anxiety interventions.

Changes in Classroom Evaluation across Grades

In general, school evaluation becomes more intensive as children proceed through school. Tests occur more frequently and have greater consequences for students' futures. Many states now have minimal competency tests that students must take before they can graduate from high school, and, of course, many students are quite apprehensive about failing such tests. With concern recently raised by *The Nation at Risk,* some school districts are introducing more frequent competency testing in the primary and in the secondary schools. In addition, tests like the American College Test (ACT) and the Scholastic Aptitude Test (SAT) given in senior high school play a major role in determining to which colleges students are accepted.

Grading practices may also become more rigorous, with most schools adopting letter grades at least by the late elementary school years. There is evidence that secondary school teachers adopt stricter and more differentiated grading policies, leading to a decline in grades during junior high school (Blyth, Simmons, & Bush, 1978; Kavrell & Petersen, 1984; Schulenberg, Asp, & Petersen, 1984). And like test scores, grades take on more meaning, as high school grade-point averages (GPA) are used for college selection and employment purposes. These changes should contribute to heightened evaluation anxiety and to the stronger negative correlations between anxiety and school performance.

Other changes in the classroom environment may also increase the evaluative pressure students feel. Eccles *et al.* (1984) discussed the kinds of classroom environment changes that occur between elementary and junior high, including moving from a smaller to a larger school, having different teachers (and classmates) for each subject, experiencing between-classroom ability grouping, being graded more strictly, and an increasing emphasis on competition and social comparison. These changes make the school environment more impersonal, threatening, diffuse, and less pleasant for some students at a time when students themselves are going

through major physiological changes (Randhawa & Michayluk, 1975; Welch, 1979).

Eccles *et al.* (1984) related these school environment changes to the negative shift in achievement beliefs and attitudes that occurs for some students after the transition to junior high school. Particularly relevant to achievement anxiety are the stricter grading policies, and greater emphasis placed on social comparison, ability perceptions, and competition. We have mentioned work showing that external evaluative practices can decrease intrinsic and continuing motivation. Competition and social comparison also may foster an ego or self-focused educational orientation in students, in which trying to outperform other students becomes more important than mastering school material (Nicholls, 1979b). In such situations, many children will perceive themselves as failures, because they are not outperforming others. This kind of learning environment should be especially detrimental to high anxious students for several reasons. First, such an environment should increase the tendency of anxious children to focus on themselves and ruminate about their performance, rather than focusing on the achievement tasks at hand. Second, because anxious children already are apprehensive about failure, an emphasis on outperforming others should make the consequences of failure even more devastating. Third, an emphasis on competition and social comparison should make anxious children's self-evaluations even less positive, because many already are performing more poorly than their high anxious counterparts. The greater focus on ability and evaluation in many junior high and secondary schools should have especially deleterious effects on the motivation and performance of test anxious children.

To avert these problems, Nicholls (1979b) suggested that optimal motivation for most students will occur when their attention is focused on task mastery, rather than outperforming others, and they are given tasks of appropriate difficulty for them. High anxious students, in particular, should benefit from this kind of educational orientation, because they have difficulty appropriately focusing their attention on the task at hand. Another option may be cooperative learning, as various studies indicate that cooperative learning structures facilitate student motivation (see reviews by Ames, 1984; Johnson & Johnson, 1974; Slavin, 1977, 1983). Such goal structures may be particularly beneficial to anxious students who do not respond well to competition and pressure (Covington & Omelich, 1982). Unfortunately, as Eccles *et al.* (1984) discussed, most schools are not moving in these directions, and instead continue practices that may increase many students' anxiety. Some intervention programs have been shown to help anxious students perform better in evaluative situations, and we will turn to them next.

Alleviating Anxiety through Intervention

Many anxiety intervention programs have been developed, mostly for use with college students and adults. These programs have been reviewed by Deffenbacher (1980), Denny (1980), Tryon (1980), and Wine (1980). The different kinds of treatment programs range from those teaching relaxation skills to those attempting to change the thought patterns of text anxious individuals. Most treatment studies have shown posttest reductions in anxiety, but few have shown corresponding increases in test performance (see Denny, 1980; Tryon, 1980). Because it is difficult to change behavior and because performance in evaluative situations is influenced by many factors in addition to anxiety, perhaps it is not surprising that programs designed to reduce anxiety do not always have an impact on behavior.

However, the quality of the program obviously makes a big contribution as well. Until recently, most studies used systematic desensitization and relaxation techniques in attempting to alleviate test anxiety, and so focused on the emotionality component of anxiety. As both Tryon (1980) and Wine (1980) have discussed, this emphasis should be changed, because worry has been shown to relate more strongly to test performance. Further, Denny (1980) concluded that treatments dealing with worry by teaching anxious students to focus their attention on the task and not to ruminate about the possibility of their failing have been more successful than simple relaxation treatments in obtaining performance gains as well as reducing anxiety. However, Tryon (1980) has argued that programs combining relaxation techniques and cognitive restructuring may be the most effective kind of program, because it has been difficult to assess which components of the training programs exert the most positive effects (but see Finger & Gallassi, 1977, for an interesting attempt to assess the separate and joint effects of training to reduce worry and emotionality). We agree that the move away from simple desensitization and relaxation techniques is a good one.

Results of some lab studies provide suggestions for what features anxiety intervention pro-

grams should include. In lab studies, changing task instructions to be less evaluative and providing more success feedback have improved the performance of anxious individuals (I. G. Sarason, 1972). However, low anxious individuals' performance sometimes declines under these conditions, a point we will return to. And success experiences alone may not be enough to ensure continued success once failure is encountered again (Dweck, 1975). Other laboratory studies also have shown that task-focusing instructions help anxious children perform better (Dusek *et al.*, 1975, 1976; Mueller, 1978).

Modeling of successful task-completion strategies is another effective way to aid the performance of high anxious individuals in evaluative situations. I. G. Sarason (1973) found that high anxious individuals performed better after they observed models successfully doing an anagrams task while verbalizing the successful strategy they were using. In a second study (I. G. Sarason, 1975), female subjects observed one of four models: a model who admitted anxiety but also described the task strategies she used to cope with it, a model not admitting anxiety, one simply admitting anxiety, and a neutral model. Subsequently, high anxious subjects who had observed the "anxious but coping" model outperformed the low anxious subjects in that condition. These studies suggest that anxious individuals may need strategy training as well as relaxation and "worry reduction" training.

The most systematic anxiety intervention work done with children in school settings is that of K. T. Hill and his colleagues (see K. T. Hill, 1984; K. T. Hill & Wigfield, 1984). This research has focused on three major testing parameters: test time limits and time pressure, success–failure experiences, and testing instructions and mechanics. In a study of the influence of time pressure on children's arithmetic performance, K. T. Hill and Eaton (1977) showed that high anxious children performed much less accurately and more slowly than low anxious children when time limits were imposed. When time pressure was removed, high anxious children performed as well and as quickly as low anxious children on simple arithmetic problems. Plass and K. T. Hill (1986), using age-appropriate math problems administered in group-testing situations to third and fourth graders, obtained similar results: high anxious boys performed as well as low anxious boys when test time limits were removed.

Williams (1976) examined how different kinds of task instructions influenced fifth- and sixth-grade children's performance on age-appropriate math problems. When the task was introduced as a test of ability, high anxious children did worse than low anxious children. In contrast, when anxiety was reduced by giving "reassurance" instructions, or by telling children that the experimenter was only interested in group and not individual performance, high anxious children did much better and actually outperformed middle and low anxious children in the "group performance" condition.

Combining these treatments, K. T. Hill *et al.* (1980) assessed how changes in test time limits and test instructions influenced junior high school students' performance in an actual achievement test situation. Students took the districts' achievement test in math and English under one of four conditions: standard testing conditions, a relaxed time limits condition, a condition in which students were told not to worry about missing difficult items, and a combined condition in which students were given more time and also told to not worry about missing difficult items. Under the standard testing conditions, the low anxious students did much better than the high anxious students. In contrast, with no time pressure, either by itself or in the condition combining relaxed time limits and difficulty information, middle and high anxious students performed much better on the math subtest. In fact, high anxious eighth graders actually outperformed their low anxious counterparts in the condition with relaxed time limits and with information about how to deal with difficult problems.

The results of this latter study are particularly important because they were obtained in a school achievement testing program, and so provide strong evidence that anxious children's performance can be improved when testing conditions are changed. Indeed, it appears that testing dynamics interfere with anxious children's performance in these testing situations, so that it is not lack of knowledge but the testing conditions that lead to their poorer performance. Thus, achievement tests given in standard ways with time limits underestimate the achievement of many high anxious students.

Recently, K. T. Hill and his colleagues have turned to more in-depth classroom training programs to facilitate the performance of high anxious children. These programs (see Ambuel, Hartman, Nandakumar, & K. T. Hill, 1983; K. T. Hill, 1986; K. T. Hill & Wigfield, 1984, for descriptions) include two components: training test-taking strategies, and training positive motivation and coping skills for evaluative situation. The training is done by classroom teachers as part of their regular in-

struction, and the sessions last several weeks. Initial results of these programs are quite encouraging, and ongoing analyses are assessing how the programs influence the performance of high anxious children at different age levels, and also how the different components of the program influence test performance.

Based on these results, K. T. Hill (1984) and K. T. Hill and Wigfield (1984) have recommended that schools should consider giving tests in two ways: using standard testing conditions for low anxious children and those who have a history of doing well in evaluative situations, because those children often thrive in situations in which there is some evaluative pressure but actually perform somewhat less well when evaluative pressure is removed (see K. T. Hill, 1972; I. G. Sarason, 1972, 1973), and using optimizing conditions for high anxious children. Schools could assess students' anxiety and test-taking skills, to identify which students would benefit from taking tests under optimizing or standard conditions, and benefit from classroom test-taking strategy programs. In this way, achievement tests would better reflect the actual learning of students and not the influence of testing dynamics. If time and budgets allowed, children could take the tests under both conditions, with the higher of the two sets of scores providing the best estimate of achievement.

This intervention work, and the other work on changing testing conditions and task instructions, seems to indicate that anxiety has causal precedence in the anxiety-performance relationship. However, work reviewed earlier shows that repeated failure experiences (and thus poor performance) leads to anxiety. In general, we agree with K. T. Hill (1972) that the cause–effect relationship between anxiety and performance becomes cyclical. We think a better understanding of this relationship could be obtained by looking at individual differences. For low achieving children, performance difficulties likely lead to anxiety. To perform better, these children will need more than changes in testing conditions, because they will not have mastered the material to be tested. Additional skill training will be needed as well. For capable students who are anxious, perhaps anxiety causes their performance problems. With less perceived pressure, more relaxed testing procedures, and a less evaluative classroom environment, these students may be able to demonstrate their mastery of the material. Those developing intervention programs to deal with anxiety need to keep these differences in mind.

Future intervention programs also should consider the age of the child more closely, to deal with the unique problems associated with anxiety at different ages. For instance, if our earlier speculations are correct, making sure young children stay task-engaged may help improve their performance, as would providing test-taking strategies. For older children, programs incorporating worry reduction training may be more successful, because as children get older, it is likely that the worry component of anxiety is what interferes with their test performance.

Conclusion and Directions for Future Research

The theoretical and empirical work on test anxiety done over the last 15 to 20 years has greatly increased our understanding of anxiety as a construct. Additionally, successful intervention strategies have begun to be developed that can reduce the debilitating effects of anxiety on achievement performance. Although these accomplishments are laudable, we still have much to learn, particularly about the development of anxiety.

First and foremost, we must examine the ontogeny of anxiety (or the propensity to be anxious) during the late preschool years and into elementary school. Do early parent–child interactions in achievement situations set the stage for anxious reactions to school evaluation? What is the developmental course of anxiety over the elementary school years; that is, how do the different components of anxiety develop? And what sorts of school evaluative practices either foster or ameliorate the effects of anxiety? Research into these issues would help us understand anxiety better, but also lead to the development of better intervention programs to deal with anxiety in the school setting.

In order to do this research, we must develop new and better ways to measure anxiety. New scales need to be developed that assess the state–trait distinction, and worry–emotionality components of anxiety in children. Measures of the ruminations and other self-deprecatory thoughts anxious individuals engage in would also be useful to clarify the cognitive component of anxiety and so develop interventions to deal with excess worry. In addition to new scales, other kinds of measures of anxiety need to be developed. These could include teacher, parent, and peer–rating scales, and different kinds of observational systems for use during class instruction, seatwork, tests, and so on. Partic-

ularly in the developmental area, methodology has not kept up with theory.

As we have pointed out, anxiety does not develop in isolation from other constructs, and so we need to study further its relationships with other constructs. We have discussed the debate over the relationship between anxiety and ability perceptions. We also need to look at the relationships between anxiety and other important achievement beliefs, such as task value. Very little research has examined the link of anxiety to those aspects of motivation associated with task value. Covington and Beery (1976) have suggested that in order to maintain their sense of self-worth, students may reduce their effort if they anticipate failing. A similar process could occur with test anxiety and task value: One way to cope with anxiety over anticipated failure is to reduce the value one attaches to the subject area, thus reducing the potential psychological cost of failure and providing an additional excuse for the low-effort strategy outlined by Covington and Beery. The impact of such a strategy on performance has not been assessed. To the extent that lowered task value leads to reduced effort, this strategy for coping with anxiety will lead to poorer performance by decreasing the likelihood of success. On the other hand, to the extent that lowered task value reduces state test anxiety, this coping strategy may decrease the negative effects of test anxiety on test performance.

Alternatively, the very fact that one is highly anxious about a particular subject area may, through classical conditioning of affective states, lead to avoidance and/or intense dislike of that subject area. Descriptions of math anxiety illustrate the power of these affective associations (Tobias, 1978). It seems likely that the desensitization interventions discussed earlier in this chapter may be essential to undo these associations before skill and attentional training can be effective. But, in any case, very little is known about how or at what age these affective and motivational consequences of anxiety develop, and virtually nothing is known about how these consequences are related to the more cognitive and attentional components of anxiety.

Although the cognitive aspects of anxiety have been emphasized of late, it is useful in closing to put anxiety into the larger context of emotional arousal. Emotional arousal is usually assumed to be necessary for action. However, excessively high levels of arousal may disrupt optimal performance just as unusually low levels of arousal lead to insufficient motivation. This curvilinear relationship between arousal and performance is well described by the Yerkes–Dodson curve. To the extent that test anxiety is a continuum ranging from very low arousal to very high arousal, then the negative effects of anxiety we have been discussing really reflect only part of the picture. For some children, the problem may be one of under-arousal instead of over-arousal. This problem may be reflected in the poorer performance of low test anxious children in the more relaxed testing conditions reported earlier; such conditions may not be arousing enough to these children. Ideally, teachers and testers should strive to bring all children to their peak level of arousal when important testing is taking place. Unfortunately, different motivating strategies appear to be needed for children with differing levels of test anxiety, and teachers and testers rarely have the luxury to tailor the testing situation to these individual needs.

Finally, although some successful intervention programs for use in the school setting have been developed, there is important new work on intervention programs that needs to be done. Given our emphasis on developmental issues, we think different intervention programs should be developed for children at different ages. Younger anxious children may benefit most from encouragement, test-taking skills training, and task-focus training, and older children may benefit most from the kinds of cognitive retraining that have been successful with adults. We know little about which age groups would benefit most from different kinds of programs such as these. Further, with regard to the intervention programs that have been successful, we need to have a better understanding of which aspects of the programs have the most beneficial effects, in order to be able to improve those programs further.

Acknowledgments

The authors would like to thank Paul Pintrich for helpful comments on an earlier draft of the chapter. The writing of this chapter was supported in part by grant BNS-8510504 from the National Science Foundation to both authors.

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CHAPTER 20

Posttraumatic Stress in Children following Natural and Human-Made Trauma

Laura M. Davidson and Andrew Baum

Posttraumatic stress disorder (PTSD) may represent one of the most profound consequences of exposure to a stressor. Although the disorder has been included as a diagnostic category in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) (American Psychiatric Association, 1980), its symptoms resemble severe chronic stress and expression of these symptoms may not necessarily reflect psychopathology (Davidson & Baum, 1986). Despite the fact that the disorder is well documented in adults, less is known about the potential for a similar syndrome to develop in childhood. The severe traumas that children may experience, ranging from the kind of hostage situation at Cokeville, Wyoming, in 1986, to more common experiences of abuse or the witnessing of havoc or homicide, can produce stress symptoms in such children.

Much of our knowledge of PTSD has been derived from studies of war veterans. Even William Shakespeare, among others, portrayed soldiers and warriors as victims of the horror of combat. However, the disorder is not unique to soldiers. In fact, the industrial revolution may have heralded a rise in the incidence or diagnosis of the disorder (Trimble, 1981). For example, with increasing reliance on railways, accidents were more likely, and experts began to describe disorders with PTSD-like symptoms, which they termed "railway spine." Because there was no observable cause for the disorders, experts argued that microstructural lesions in the spinal cord were causing observed problems (Erichsen, 1882). Although this explanation was not accepted by everyone (Page, 1885), it remained the prevailing view through World War I, during which cases of severe emotional reactions to combat were referred to as "shell shock," in part because it was believed that the concussion of exploding shells and heavy artillery resulted in damage to the central nervous system. During World War II, psychiatrists began to recognize that a number of factors were associated with severe combat reactions, including fatigue, danger, environmental conditions, and premorbid personality factors (Craighill, 1966), and PTSD became recognized as a psychological disorder rather than one of organic origin. Despite the fact that PTSD is not a new problem, much of our current knowledge of it has been derived recently from studies of veterans of the Vietnam war. Characteristics of this particular

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conflict may have made those who fought in it particularly susceptible to developing PTSD (Wilson, 1980).

In this chapter, we will discuss the consequences of stress and trauma among children, using the more extensive literature of research and experience with adult victims to provide a context for the reporting of data and speculation, and with PTSD as an organizing model for this discussion. Precipitating events most frequently observed or discussed as responsible for prolonged stress reactions will be considered, as will common symptoms of distress. The primary focus of this chapter will be on the development of psychophysiological symptoms of stress and PTSD among children exposed to trauma of human and natural origin.

Precipitating Events

PTSD may be caused by a number of different events, but exposure to a stressor outside the realm of normal human experience is the essential defining characteristic. The stressor can be a natural disaster (e.g., severe storms, floods, or tornadoes) or of human origin (wars or nuclear accidents). Human-made events seem to be particularly likely to cause psychiatric problems. On the nature of war as a stressor, Garmezy states (1983):

> Wars provide all the trappings that accompany malnutrition, dislocation, traumatic injury, death, and separation. Events such as these have an intensity that has assigned them the status of near-universal stressor, experiences that tend to disrupt the behavior of most individuals irrespective of their role status, their geographical locale, or perhaps even the historical era in which they occur. (p. 46)

Although some events may approximate this notion of near-universal stressor, not all traumatic events lead to pathological outcomes. The eventual outcome can be viewed as an interaction between characteristics of the person (styles of coping, social support, and preexisting psychopathology) and characteristics of the event and environment. Characteristics of traumatic events that have been associated with negative outcomes in adults include bereavement (Gleser, Green, & Winget, 1981), displacement (Huerta & Horton, 1978), life-threat (Adler, 1943), and exposure to the grotesque (B. L. Green, Wilson, & Lindy, 1985).

But events that are stressful for adults may not be stressful for children and likewise events that threaten or tax children may not have the same impact on adults. Pynoos and Eth (1985) found age-dependent differences in PTSD among a sample of children who had witnessed a murder. In general, these researchers found that younger children had the most difficulty and required the most assistance in assimilating the traumatic event. These findings are similar to those reported by studies that have examined children undergoing the stress of hospitalization or parental divorce; again younger children were the most severely affected (Rutter, 1966, 1983). Maccoby (1983) explained this age phenomenon by noting that with increasing age children develop a greater repertoire of coping responses.

Gender may also play a role in the development of problems in children. Although some studies suggest that adult women report more disturbance following a disaster (e.g., Flynn & Chalmers, 1980), the results may be reversed for children. Boys appear more adversely affected by such events as hospitalizations, divorce, and parental discord (Hetherington, 1980; Rutter, 1970, 1982). This may be because parents have different expectations of boys and may not be as willing to provide additional support during times of need (Elder, 1979), or because the stress may be more salient to them (Block, Block, & Morrison, 1981). However, in disaster research, the results are mixed. Some researchers found no association between sex and symptoms (Block, Silber, & Perry, 1956; Terr, 1981), others found girls to be more distressed (Gleser *et al.*, 1981), and others found more problems among boys (Burke, Borus, Burns, Millstein, & Beasley, 1982; Milgrim & Milgrim, 1976).

Children may not be upset by events that cannot be immediately perceived as harmful. They are not bothered directly by threats that are remote or abstract, but they may be influenced by the anxiety experienced by their parents under such circumstances. In general, children are most adversely affected by circumstances that separate them from their primary caregiver (Garmezey, 1983), probably because in children successful coping is dependent in a large part on the ability to demand compensatory care from caregivers (Leiderman, 1983). Even a brief separation may have a negative effect on children. Research on adults suggests that separation during a crisis may make that situation more frightening and stressful. The initial emotional distress and fear may persist beyond the immediate postimpact period (e.g., Fritz & Marks, 1954). The consequences may be even more dramatic for children.

Symptoms of PTSD

Although there are many symptoms of PTSD in adults, some are considered necessary for diagnosis. For example, the traumatic event must be reexperienced. This may occur through intrusive and recurrent recollections of the event, dreams, or through dissociative states during which events may seem to be occurring again. Emotional "anesthesia" also occurs following the traumatic event, and symptoms that may represent this characteristic include diminished affect or an inability to feel emotions and a loss of interest in previously significant activities or people. A number of symptoms reflecting excessive autonomic arousal may be present and include an exaggerated startle response or hyperalertness, difficulty in sleeping, and memory impairment. Victims may also avoid reminders of the traumatic event because their symptoms may intensify under such circumstances, and they may feel guilty about their survival or circumstances surrounding their survival.

Although researchers have examined the consequences of natural and human-made disasters in children, few have looked specifically at the experience of stress or the development of PTSD. However, some research provides information about the kinds of symptoms experienced by children who have been exposed to traumatic events, and these symptoms may vary depending on the child's age. Young children's behavior may regress to a previous level of functioning: for example, they may lose bowel and bladder control, become irritable and cry frequently, develop stranger anxiety, suck their thumbs, develop fears and eating problems, and exhibit increased motor activity and trembling. Because preschoolers lack fully developed verbal skills, their symptoms are often exhibited in nonverbal channels. For example, they may reenact the event through play; MacLean (1977) reported that a boy who had been attacked by a leopard continued to play out these circumstances.

School-aged children may develop similar problems. Fears and anxieties have been reported to be their predominant symptoms (Butterfield & Wright, 1984). Because school-aged children often indicate that they are aware of their physical symptoms during and after the traumatic event, they may develop psychosomatic complaints including visual and hearing problems and headaches (Pynoos & Eth, 1985). Since they are in school, their problems are often exhibited there; they may fight with their peers or withdraw from friends and they may refuse to go to school or not pay attention and hence suffer declining performance. They also report sleep disturbances, including difficulty falling to sleep as well as midnight wakenings, nightmares, sonambulism, and bed-wetting. School-age children may elaborately reenact the traumatic event.

Exposure to traumatic events in preadolescents and adolescents may result in premature entrance into adulthood or closure of identity-formation processes (Pynoos & Eth, 1985). Unlike preschoolers and younger school-aged children, the preadolescents and adolescents cannot use play as a way of working through their problems, their actions can become self-destructive, and they may exhibit aggression and suicidal ideation. Like younger children, they often develop a number of physical complaints; they may become withdrawn, and their sleep and appetite may suffer. Their performance in school often changes; they may avoid school, fail their classes, or become disruptive.

Terr (1983, 1985) has identified posttraumatic play as a symptom unique to the children with the disorder. The themes of this type of play link it to the traumatic event, and it is compulsive and repetitive in nature. This play often involves others and generally fails to relieve any of the accompanying anxiety. Unlike adults with the disorder, Terr (1983) found no evidence of psychic numbing or flashbacks in her sample of children.

Natural versus Technological Disasters

Fundamental differences exist between natural disasters and those of human origin. Whether this is reflected in different levels of stress or dysfunction is not clear, but accounts suggest that for adults, disasters of human origin tend to be related to more deleterious emotional consequences (e.g., Baum, Fleming, & Davidson, 1983). This may be because of the differences between the two types of events and the meaning that each has for adults. Although both are sudden and powerful, the damage caused by each may differ in significant ways. Damage caused by natural disasters is generally visible, and previous experience with similar natural events can help predict typical outcomes. A flood may destroy homes and property and disrupt power and sanitation, and these efforts are relatively predictable. Technological events cause a different type of damage, which is more insidious and often more harmful. Although these types of events often involve the release of toxic chemicals and radiation,

we currently do not know all the health hazards these substances pose. In addition, it is almost impossible to predict each person's future risk of developing a problem partly because exposure and susceptibility are difficult to measure. Furthermore, when technological disasters occur there may never be a time when the victims can say the worst is over. Because some types of cancers or other health consequences of toxic exposure take twenty years or more to develop, the worst consequences of chemical exposure may not be seen until future generations reach maturity. On the other hand, natural disasters do have a "turning point," after which the worst is over and recovery is expected. Finally, when technological disasters occur, they often erode confidence in our ability to control our inventions, and people not directly affected may feel more vulnerable. Hence, technological disasters represent a loss of control over a situation in which someone is supposed to have had control.

Recent research has revealed that technological disasters, such as the one at the Three Mile Island nuclear power station, are associated with symptoms of PTSD in adults. At Three Mile Island (TMI), chronic stress levels were measured using self-report, behavioral, and biochemical measures in a group of people living within 5 miles of the power plant. Symptoms of PTSD were assessed using the Impact of Events Scale (Horowitz, Wilner, & Alvarez, 1979). Results indicated that TMI area residents experienced more symptoms of chronic stress and of PTSD than did a comparable but nonvictimized control group (Davidson & Baum, 1986). In addition, research comparing a group of people living near a toxic dump site in New Jersey to a group of people who experienced a major flood in Mississippi, and to a control population matched on demographic variables, confirmed expectations that exposure to technological disasters is accompanied by disturbances not associated with those caused by disasters of natural origin (Baum, 1987).

Although the distinction between natural and technological disasters provides a useful framework for predicting the long-term consequences of disasters in adults, its efficacy in children is limited. Because loss of or separation from a parent is the most common cause of disordered functioning in childhood, technological disasters may not be more harmful than natural ones during this period in the life span. In fact, the greater destructive power of a natural disaster may be more threatening or more likely to affect parent–child relationships. Whereas adults may be threatened by abstract concepts like radiation exposure, children are more afraid of immediate and concrete events (Melamed & Siegel, 1988). Although the mere threat of radiation and subsequent development of cancer may not pose as severe problems for children as for adults, actual experience with cancer may cause more problems for children. Nir (1985) concluded that the diagnosis of cancer was accompanied by posttraumatic stress symptoms in children almost without exception. Hence, technological disasters may cause distress in children through different mechanisms among adults.

Rutter, Cox, Tupling, Berger, and Yule (1975) isolated three factors that ameliorate the consequences of stress in children: they are "personality" factors including flexibility and gender (boys being more susceptible than girls), family cohesion, and supports outside the family environment (community, church, and school). Although technological disasters may not directly threaten children, they may cause mental health consequences indirectly by affecting family and community functioning. Although exposure to radiation may not worry children, it does have an impact on adults. Research indicates that parental anxiety has a negative effect on the adjustment of children (Melamed & Siegel, 1988). In addition, studies have shown that children can show symptoms of PTSD even when they have not been exposed to a traumatic event. Research on the children of the Holocaust victims shows that children identify with the experiences of their parents and may act out the rage felt by their parents (Sigal, 1971; Solkoff, 1981). They may develop problems because of parental rejection and lack of understanding. Thus, the stress experienced by the survivors may influence their parenting behaviors. Similarly, children of Vietnam war veterans may exhibit symptoms of PTSD, including guilt, sleep disturbances, anxiety, a preoccupation with their parent's trauma, and may experience fantasies and flashbacks in the same way as their parents (Rosenbeck & Nathan, 1985).

Human-Made Events

Following World War II, researchers examined the effects of this conflict on child survivors. Perhaps the group of most severely traumatized children were those who were held in Nazi concentration camps. Anna Freud cared for a group of these children in her Hampstead nursery and reported:

> [They were] without a doubt "rejected infants" . . . deprived of motherlove, oral satisfaction, stability in their relationships and their surroundings. They were passed from one hand to another during their first year, and were uprooted again three times during their fourth year . . . they were neither deficient, delinquent, nor psychotic. They had found an alternate replacement for their libido and, on the strength of this, had mastered some of their anxieties, and developed social attitudes. That they were able to acquire a new language in the midst of their upheavals, bears witness to a basically unharmed contact with the environment. (Freud & Dann, 1951, p. 168)

In a 30-year follow-up of survivors who had been placed in such a self-help group, Robinson and Hemmendinger (1982) found no evidence of psychoses among the group, although some subjects reported depression, insomnia, and nightmares. However, children who were hospitalized did not fare as well; 50% under 17 years of age showed evidence of psychoses, 78% under 7 were psychotic, and 100% under 3 were severely debilitated. These results illustrate the fact that the youngest children were the most severely affected. Of these younger children, Hogman (1983) said

> since the youngest children left orphans seemed to have been most scared by the war experience and to have had the most difficulty in their search for identity, it may well be that they suffered from lack of memories. (p. 64)

Because the hospitalized children fared much worse than those in a more homelike environment, inclusion in a functioning social group seemed to be important in preventing later symptom formation.

Children exposed to air raids were also studied during World War II. Bodman (1941) and Freud and Burlingham (1943) reported that the children who stayed with their parents fared the best. Even though they were not spared death, injury, and destruction, they often viewed the conflict as an adventure or an accident like other accidents of childhood (Bodman, 1941; Freud & Burlingham, 1943).

The continuing conflict in and around Israel has provided a more recent setting to explore the relationship between trauma and symptom formation in children. Ziv and Israeli (1973) administered the Hebrew Children's Manifest anxiety scale to kibbutzim children under shelling and kibbutzim children not under shelling and found no difference in anxiety between the two groups. On the other hand, Milgrim and Milgrim (1976) administered a general anxiety scale 4 months before the Yom Kippur War and then again while the army was still mobilized. Overall, levels of anxiety almost doubled. The children who reported the greatest wartime levels of anxiety were the ones who had reported the least amount of anxiety during peace. Kristal (1975) also found that children exposed to shelling had a higher incidence of bruxism. The children from the shelled villages also exhibited more anxiety than the nonshelled group following a movie of shelling. These results parallel those reported on Vietnam veterans in which veterans with posttraumatic stress were more reactive to reminders of combat than were controls (Blanchard, Kolb, Pallmeyer, & Gerardi, 1982; Malloy, Fairbank, & Keane, 1983). Another group of researchers (Ziv, Kruglanski, & Shulman, 1974) compared attitudes between children who were exposed to shelling and those who not exposed. Although there were no differences in attitude about the war between the two groups, those exposed to shelling reported greater local patriotism and exhibited more covert aggression.

In a study of the Belfast riots during 1969, Lyons (1971) noted few cases of psychiatric symptoms in children. Problems typically occurred in children whose parents were similarly affected. Arroyo and Eth (1985) studied 30 children who were referred to a psychiatric clinic; they all had been exposed to strife in Central America. In this group, they were able to diagnose 10 cases of PTSD, and the most severely affected and difficult to treat were the youngest children.

The accident that occurred at the Three Mile Island nuclear power plant provided researchers with the opportunity to study the effects of human-made disasters other than war on children. Most researchers reported no ill-effects in these children or effects that were quickly resolved following this event. Dohrenwend, Dohrenwend, Kasl, and Warheit (1979) measured distress in adolescents 2 months after the accident occurred. Although 25% reported some acute psychological distress, this rapidly dissipated. Those who were evacuated and lived closer to the plant experienced the greatest amount of distress. Three and one half years after the accident, Bromet, Hough, and Connell (1984) found no differences between TMI children and children living near an undamaged nuclear reactor in terms of social competence, behavior problems, fearfulness, and self-esteem. Similarly, although Baum and his colleagues continue to find symptoms of PTSD among adults living near TMI, they have found less support for similar problems among the children. Three and one half years after the accident, study participants were asked about problems that their children may have experienced during the

preceding 12-month period. Although the parents continued to report more symptoms of stress than a control population, they did not report more overall symptoms in their children. There were, however, some differences between the two groups in terms of somatization, fear, and behavioral problems (see Table 1). These data would suggest that children may show some evidence of disturbance following a nuclear accident but that they are less severely affected than are adults.

Following in the wake of World War II and again after the accident at TMI, researchers have examined the impact of the threat of nuclear disaster on children. Escalona (1982) suggested that growing up in an environment that tolerates and ignores the risks of total human destruction has an impact on the development of attitudes in children. In particular, it is suggested that this social climate fosters an investment in the here-and-now and less of an interest in developing long-term goals. Studies indicate that young people are becoming more concerned with the nuclear threat. In a 1970 survey of 17- and 18-year-olds, 7% often worried about the threat of nuclear war; by 1982, 31% of 17 to 18-year-olds voiced the same worry (Bachman, 1983). In a study conducted on 10- to 17-year-olds after the accident at the Three Mile Island nuclear power station, researchers found several age-dependent differences (Schwebel & Schwebel, 1981). Younger children had less confidence in our ability to avoid another accident and were more likely to advocate closing the plants. Older children felt we were more in control of nuclear power and were more likely to suggest added safety precautions rather than closing the plant. These researchers also found increasing concern with nuclear accidents. Although 41% of the youths in 1960 predicted nuclear war, 70% of the children polled in 1981 predicted another nuclear accident.

Table 1. Items Endorsed More Frequently by the Three Mile Island Group

Item	Chi-square test
Disobedient at school	$\chi^2(1) = 4.66, p < .03$
Talks too much	$\chi^2(1) = 4.54, p < .04$
Doesn't get along well with other children	$\chi^2(1) = 4.59, p < .03$
Fears certain animals, situations, or places other than school	$\chi^2(1) = 7.70, p < .006$
Cruel to animals	$\chi^2(1) = 3.82, p < .05$
Sleeps less than most children	$\chi^2(1) = 5.48, p < .02$
Physical problems without known medical cause including:	
Headaches	$\chi^2(1) = 4.46, p < .03$
Nausea, feels sick	$\chi^2(1) = 4.66, p < .03$
Problems with eyes	$\chi^2(1) = 4.66, p < .03$

Acts of terrorism appear to have a profound impact on children. For example, the Chowchilla school-bus kidnapping, which occurred on July 15, 1976, influenced all the children involved. For approximately 27 hours, 26 children ranging in age from 5 to 14 were missing and were held by kidnappers under bizarre circumstances. A school bus that the children were on was stopped by three masked men, the children were transferred to boarded-up vans, were driven for 11 hours, and finally were buried in a truck-trailer from which they were finally able to escape. Terr (1979) interviewed 23 of these children within 14 months of the event and also did a follow-up study 4 to 5 years after the kidnapping on 25 of the children (Terr, 1983). In the 4-year follow-up, she found that every child was suffering from some symptoms of PTSD. Brief psychiatric intervention at the time of the incident did not alleviate the symptoms. Children had a pessimism about the future, a fear of omens, and incorrect perceptions; they also experienced shame, fear, anxiety, nightmares, and dreams.

In another strange act of terrorism against children, a sniper fired a semiautomatic rifle at a group of children who were leaving school. One child was killed, 13 were injured, and many more were pinned to the playground during the siege. Nineteen to 39 days following the event, 159 children aged 5 to 13 participated in semistructured interviews (Pynoos, Frederick, Arroyo, Nader, Eth, Lyon-Levine, Silverstein, & Nunez, 1985). Results indicated that 60% of the children experienced some form of PTSD and that severely threatening incidents do lead to acute PTSD among children. Symptom severity was directly related to exposure to life threat. There were no relationships between symptoms and age, sex, or ethnicity. In a 6-month to 1-year follow-up of children exposed to this event, continuing symptoms of distress were noted, varying directly with acquaintance with the dead child (Pynoos, Nader, Frederick, Gonda, & Stuber, 1986). Researchers have examined the consequences of other types of acts against children, including abuse and incest (Goodwin, 1985; A. H. Green, 1985) and have found symptoms of posttraumatic stress in both of these victimized groups.

Natural Disaster

Natural disasters can strike unexpectedly and produce problems among certain groups. In a study of children exposed to a blizzard and subsequent flood with pre- and postdisaster data available, Burke *et al.* (1982) found no differences in overall functioning, although there was an increase in aggressiveness among the children. Those children who were designated by their parents to have "special needs" and boys in general had the most adjustment problems. Other researchers have examined the relationship between parental problems and symptom formation in children following natural disasters. In general, there seems to be a positive correlation between the two. In a study of the 1953 tornado that hit a movie theater in Vicksburg, Minnesota, Bloch, Silber, and Perry (1956) found that proximity to the impact zone, injury of family member, and dependent parents predicted emotional disturbance in children. Nonetheless only 13% of the children were severely affected. Similar results were reported by McFarlane (1984) in children exposed to Australian bushfires. Twenty-three percent of the children who were studied exhibited some psychological disturbances, including sleeplessness, nightmares, enuresis, clinging, and school phobia. Adult strain was thought to be reflected in the problems of the children.

Childhood fears are often exacerbated by natural disasters. In a study of the aftermath of a lightening bolt that hit a soccer field killing one child and injuring two, researchers assessed fear, sleep disturbances, and somatic complaints in the uninjured players (Dollinger, O'Donnell, & Staley, 1984). They found more fears of lightening among the experimental than among the control groups as well as more fear in general. Fears were associated with sleep disturbances, somatic complaints, and overall emotional upset. In the study of Cyclone Tracy, which struck Darwin, Australia, in 1974 killing 50 and injuring more than 500, Milne (1977) assessed child behavior through 267 parental reports. Although changes in behavior were generally small, he found that 26% of the children had storm-associated fears 7 to 10 months after the disaster.

Conclusions

Although children are not immune to the psychological sequelae of human-made and natural disasters, the accompanying disturbances are often minimal and short-lived. As Garmezy (1983) stated,

> there is little gained by those who cry havoc while failing to heed the recurrent findings of our literature on the ability of children to meet and to conquer adversity. (p. 78)

However, children may exhibit problems following disasters or other kinds of victimization. These problems may resemble those of the posttraumatic stress disorder. Parental loss or vulnerability appears to put children at higher risk for developing psychopathologies, and when PTSD does develop, its accompanying features may reflect the age of the victim.

Acknowledgments

Preparation of this chapter was facilitated by research grant R07265 from the Uniformed Services University of the Health Sciences. The opinions or assertions contained herein are the private ones of the authors and are not to be construed as official or reflecting the views of the Department of Defense or the Uniformed Services University of the Health Sciences.

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CHAPTER 21

The Developmental Psychopathology of Child Maltreatment

Dante Cicchetti and Kurt Olsen

Introduction

Child maltreatment is a complex, insidious problem that, although predominant in impoverished families (Pelton, 1978), cuts across all sectors of society. The American Association for Protecting Children (1986) tallied 1,727,000 reports of suspected child maltreatment in 1984. Forty two percent of these reports were substantiated. A national survey reported that 10.7% of parents admitted to having perpetrated a "severe violent act" against their child in the previous year (Straus & Gelles, 1986), and prevalence rates of sexual abuse have been estimated to be as high as 62% for girls and 31% for boys (Dubowitz, 1986). The economic and human costs of maltreatment in American society are astronomical. It is likely that billions of dollars are spent in treatment and social service costs and are lost in lessened productivity for a generation of maltreated children (Dubowitz, 1986). The human costs are a litany of psychological tragedies: Maltreated children suffer from poor peer relations, cognitive deficits, and low self-esteem among other problems; moreover, they tend to be more aggressive than their peers as well as having other social problems (see Aber & Cicchetti, 1984, for a review). The emotional damage that is due to maltreatment may last a lifetime.

Goals of This Chapter

After placing the study of child maltreatment into its proper historical context, we will provide a summary of what is currently known about the definition, etiology, developmental sequelae, and prevention of the various forms of child maltreatment. Finally, we will suggest prescriptions to guide future research and treatment. Even though child maltreatment is not listed as a diagnostic entity in the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1987), we believe that maltreatment should be conceived as a "relational psychopathology"—that is, the result of a dysfunction in the parent–child–environment transactional system (Cicchetti & Rizley, 1981; Sameroff & Chandler, 1975).

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Historical Perspective

History documents that the problem of child maltreatment has existed since the beginning of civilization (Aries, 1962; Radbill, 1968; Ross, 1980). Unfortunately, our understanding of the etiology, intergenerational transmission, and the developmental sequelae of this pervasive social problem largely has been the result of relatively recent systematic inquiry. Until a generation ago, modern society had refused to recognize the scope and gravity of child maltreatment. In fact, prior to the 1960s, many sectors of our society (e.g., medical personnel) failed even to acknowledge its existence (Dubowitz & Newberger, 1989).

In 1946, John Caffey, a pediatric radiologist, suggested that infants' incurred multiple fractures in association with intracranial bleeding might be the result of parental abuse or neglect. Several years later, Silverman (1953) reported on the high incidence of physical injury in infants and chided his colleagues on their reluctance to recognize the possibility that some of these injuries may have resulted from parental abuse.

Apparently, it was not until 1961 when these efforts had a clear impact among the medical community (Cicchetti, Taraldson, & Egeland, 1978; Lynch, 1985). Alarmed by the increasing number of children with nonaccidental injuries being admitted to pediatric clinics, C. Henry Kempe and his colleagues conducted a symposium on child abuse at the annual national meeting of the American Academy of Pediatrics. In an effort to underscore the seriousness of the problem, Kempe and his colleagues, in an influential article published in the *Journal of the American Medical Association*, coined the term "battered child syndrome" (Kempe, Silverman, Steele, Droegemueller, & Silver, 1962). As a direct result of this paper, a dramatic increase in legislative activity ensued, resulting in the establishment of mandated child abuse reporting laws for all 50 states before the year 1970. Moreover, at about this same time, a heightened sensitivity to the needs of children occurred during a contemporary ethos that was increasingly concerned about the rights of the disadvantaged sectors of our society. For example, regional centers for retarded children, Project Head Start, and Medicaid were all initiated during this era (Dubowitz & Newberger, 1989). Furthermore, during the past two decades, the legal status of children has changed considerably (Koocher, 1976; Polier, 1975; Rodham, 1973; Wald, 1975), and children are now seen as having a right to the fulfillment of their developmental needs (Alvy, 1975; Derdeyn, 1977; Goldstein, Freud, & Solnit, 1973).

Despite the positive consequences of the yeoman efforts of Kempe and his colleagues (Cicchetti & Aber, 1980; Lynch, 1985), a number of unfortunate negative ramifications ensued. The adoption of the term *battered child syndrome* connoted a psychologically disturbed parent-as-perpetrator model of child abuse (Parke & Collmer, 1975). This "narrow" etiological view of helpless infants and children being battered maliciously by psychiatrically ill parents (Spinetta & Rigler, 1972; Steele & Pollack, 1968) virtually absolved society of any responsibility for the occurrence of child abuse (Alvy, 1975). In addition, the treatment prescriptions emanating from this medicalization and legalization of child abuse focused on supplying treatment for the abusing parent without stressing the need to provide concomitant intervention for the abused child (Cicchetti *et al.*, 1978).

During the 1970s, the laws mandating the reporting of child abuse were changed. The Child Abuse Prevention and Treatment Act of 1974 expanded the definition of child abuse to encompass emotional injury, neglect, parental deprivation of medical services, and factors deleterious to children's moral development (Dubowitz & Newberger, 1989; Giovannoni, 1989). This broadened definition of child abuse contributed to the belief that not all abusing parents set out with the intention to destroy their helpless offspring.

Increasingly, broader social factors (e.g., societal attitudes, structures, and stressors) were implicated as playing causal roles in child maltreatment. David Gil's theory of child abuse highlights the essence of sociological theorizing. Viewing the etiology of child abuse in terms of a sociocultural perspective, Gil (1975) delineated five causal dimensions: (1) the society's basic social philosophy, its dominant value, concept of humans, and the nature of its institutions; (2) the society's definition of childhood, that is, the rights and expectations of children; (3) the society's acceptance of the use of force in attaining ends; (4) "triggering contexts," which included poverty, overcrowding, inadequate services, large numbers of children, social isolation, and the like; and (5) various forms of psychopathology which Gil considered to be deeply rooted in the social environment of the individual.

Bronfenbrenner (1974a,b) also generated a sociological model of child abuse focusing on factors that potentiate high-risk situations. Bronfenbrenner

related child abuse to the degree to which "social support systems" exist and operate for parents. He postulated that the occurrence of child abuse was a function of how the human ecology may enhance or undermine the parenting process. A "total human ecology" that provides adequate support decreases the probability that abuse will occur, whereas an unsupportive, stress-laden ecology accentuates its likelihood. Garbarino and his colleagues (Garbarino, 1976; Garbarino & Gilliam, 1980) have provided corroborative empirical evidence for Bronfenbrenner's position.

In a landmark article, Pelton (1978) argued that the "myth of classlessness" surrounding child abuse that had been promulgated by government officials and social scientists supported the "narrow" psychopathological viewpoint on the etiology of child abuse. Pelton concluded that poverty *per se* created stresses that could result in the maltreatment of children.

As the etiological models became less focused on parental psychopathology as the cause of child abuse, increased attention was paid to the prevention of child abuse and to the research and treatment of maltreated children (Kempe & Helfer, 1972; Kempe & Kempe, 1978; Martin, 1976). As careful analyses of the foster care system of the United States demonstrated significant problems (Children's Defense Fund, 1979; Fanshel & Shinn, 1972; Gruber, 1978; Mnookin, 1973), efforts were made to keep children at home whenever possible. The work of Jane Knitzer and her colleagues is exemplary among these studies. They brilliantly documented and analyzed the dangerous tendency for temporary foster care to become permanent, much to the detriment of the child, who would often develop more soundly and happily living with an abusive parent rather than with a series of foster parents (Children's Defense Fund, 1979). As a result of such investigations, growing numbers of clinicians advocated that the entire family be treated in maltreatment cases (Cicchetti *et al.*, 1978; Kempe & Kempe, 1978).

In recent years, two new theoretical viewpoints on child maltreatment have been introduced: the ecological model (Belsky, 1980) and the transactional model (Cicchetti & Rizley, 1981). Although these models will be elaborated upon in a later section of this chapter, they are significant for our current purposes because both are explicitly developmental in their orientation to the causes, intergenerational transmission, and sequelae of maltreatment. The influence of developmental theorizing into the field of child maltreatment has resulted in the formation of appropriately complex etiological models and a rich framework for conceptualizing the effects that maltreatment has upon adaptive and maladaptive ontogenetic processes.

The Developmental Psychopathology Perspective

The domain of developmental psychopathology has emerged as an outgrowth of the thinking that emphasized that the developmental approach can be applied to any culture or population, normal or otherwise deviant (Werner, 1948). Nearly all of the great systematizers in psychology, psychiatry, and psychoanalysis have argued that we can learn more about the normal functioning of an organism by studying its psychopathology and, likewise, more about its psychopathology by studying its normal condition (Cicchetti, 1984). The study of children who are at risk for developmental psychopathology can make many important contributions to our existing theories of normal development (Cicchetti, 1984; Rutter & Garmezy, 1983). The empirical investigation of populations in which differing patterns of socioemotional, cognitive, social-cognitive, and linguistic development may be expected as a consequence of the pervasive and enduring influences that characterize the transaction among the child, the parent, and the environment, such as is the case with maltreated children and their families, provides the appropriate basis for affirming and challenging current developmental theory.

As "experiments in nature" (Bronfenbrenner, 1979), the investigation of maltreated children and their families can provide information relevant to many issues and controversies in developmental theory. For example, research on maltreatment can enhance our understanding about the determinants of parenting (Belsky, 1984; Belsky & Vondra, 1989), the intergenerational transmission of parenting (Hunter & Kilstrom, 1979; Kaufman & Zigler, 1989), the socioemotional bases of communicative development (Cicchetti & Beeghly, 1987; Gersten, Coster, Schneider-Rosen, Carlson, & Cicchetti, 1986), and the development of intimate relationships across the lifespan (Crittenden, 1988; Main & Goldwyn, 1984; Morris, 1983; Schneider-Rosen & Cicchetti, 1984). Each of these topics provides but an illustration of how the study of maltreatment phenomena from a developmental per-

spective can greatly elucidate our knowledge of normal ontogenetic processes as well as significantly contribute to our corpus of information on maltreatment *per se*.

Definitional Issues

Child maltreatment is a heterogeneous problem. In mode of maltreatment, symptomatology, etiology, and sequelae, there are subtle and complex differences of type and severity (Cicchetti & Rizley, 1981). Consequently, in the past, definitions of child maltreatment have varied widely across research samples. Until recently, the lack of precision in conceptualizing the independent variable of child maltreatment has resulted in a blurring of distinctions and a dearth of fine discriminations among maltreatment subtypes. Early clinical investigations and research reports either grouped all maltreated children together or studied only physically abused children (Aber & Cicchetti, 1984). As time went on, researchers began to compare physically abused children to neglected children in an attempt to ascertain the differential impact of these types of maltreatment (Aber & Cicchetti, 1984). Both practical limitations and theoretical considerations have contributed to the paucity of clinical and research investigations that have attended to defining the disparate subtypes of maltreatment. Practically, it is extremely difficult to gain access to highly sensitive and classified records of maltreatment. Furthermore, such records often are not kept systematically with research or even nosological considerations in mind. Additionally, maltreating parents themselves often make unreliable reports about the particular details of the maltreatment situation for a host of reasons, such as shame, fear of legal or social repercussions, and self-denial, among others.

Recent research efforts have come a long way in the specification of a nosology of maltreatment (Cicchetti, Carlson, Braunwald, & Aber, 1987; Egeland & Sroufe, 1981). An important first step has been the development of a series of 8 categories and an 87-item checklist for social service case workers by Giovannoni and Becerra (1979). They surveyed a variety of professionals involved with child maltreatment and derived a list of 8 factors which they considered to be a rough consensual definition of child maltreatment. Based on this work, Giovannoni and Becerra (1979) developed a checklist and validated it on a sample of 949 cases of reported maltreatment. Their subtypes include: physical injury, sexual abuse, drug/alcohol abuse (by the child), emotional mistreatment, moral/legal problems (by the adult), failure to provide, child behavior problems, and inadequate physical environment. They reported that the single best predictor of whether or not a social service agency or family court will choose to intervene and remove a child from his or her home is the absolute number of different types of maltreatment perpetrated against a child. In fact, most children who have experienced maltreatment have been subjected to more than one subtype (Aber & Allen, 1987; Cicchetti & Rizley, 1981; Egeland & Sroufe, 1981). For these reasons, among others, researchers and theoreticians have called for the inclusion of subtype definitions in future maltreatment research (Aber & Cicchetti, 1984; Besharov, 1981; Cicchetti & Rizley, 1981).

Aber and Zigler (1981) provided a thorough critique of various conceptualizations and definitions of child maltreatment. They outlined the medical-diagnostic, sociological, legal, and research definitions, including the major determining factors of each. In the *medical-diagnostic definition,* the focus is on the individual abuser. Aber and Zigler (1981) cite the work of C. H. Kempe and colleagues (1962) as an attempt to diagnose the underlying pathology that potentiates a parent's infliction of harm upon a child. In keeping with a medical-diagnostic model, efforts in the 1960s and the 1970s were made to predict reliably the "risk" for maltreatment based upon parent personality measures. Maltreating phenomena were seen as a subcategory of psychopathology. In an important study, Newberger, Reed, Daniel, Hyde, and Kotelchuck (1977) showed a high percentage of erroneous predictions derived from their categorizations of children into abuse, failure to thrive, accident, ingestion, or control cases. Cicchetti and Aber (1980) argued that efforts at predicting individual cases of child abuse should be abandoned whereas methods of preventing child abuse should be encouraged. They stated that it is unlikely that we will ever reduce our false positive rates to a point where reasonably accurate predictions of families "at risk for abuse" can be made.

In the *sociological definition,* the focus is on the act of maltreatment. Giovannoni and Becerra (1979) are cited as the best exemplars of this approach. Aber and Zigler's (1981) major criticism of the sociological approach is that the information gathered via social service agents may not reflect accurately the severity or scope of maltreatment occurrences. This criticism seems to be a meth-

odological/research question that can potentially be remediated by more stringent tests of the Giovannoni and Becerra (1979) checklist and adaptations of it to other agencies and persons (e.g., teachers, therapists, parents, and neighbors).

The third perspective that Aber and Zigler (1981) describe is the *legal definition*. In criteria put forth in the Juvenile Justice Standards Project (1977), the definitional focus shifts to the notion of actual or possible harm to the child (i.e., physical harm or severe anxiety, depression, withdrawal, or untoward aggression toward self or others). In this framework, the issue of intentionality is addressed as well. If the harm to the child can be shown to have been unintentional, then the circumstances are not considered to be maltreatment.

The final scheme elucidated by Aber and Zigler (1981) is the *research definition*. They reviewed two strategies that are common in research. One is the use of reports to state departments of social service as the research definition of maltreatment. This approach suffers from the variability of different reporters' criteria and the likelihood that many cases are unreported, resulting in an atypical sample of reported cases. The second strategy they outline involved a narrow criterion of only physical violence. This definition increases reliability but sacrifices breadth.

As an alternative to the four definitional schemes which they critiqued, Aber and Zigler (1981) proposed three sets of definitions for three different social purposes: (1) legal definitions are needed to inform family courts and lawmakers and caseworkers concerned with coercive state intervention in custody issues; (2) case-management definitions are needed to aid in clinical decision making; and (3) research definitions are needed to allow investigations of causality and specificity of sequelae in various circumstances of maltreatment. This conceptualization of the need for three types of definitions is laudable in that it recognizes the social complexity of maltreatment phenomena. The real-world uses of definitions are varied enough so that the clinical case manager and the family court judge may look at the same circumstances from very different perspectives, yet both of them might find the researcher's definitions to be irrelevant to their work. However, this state of affairs does not need to exist. The researcher can do much to inform and to integrate all three fields. As the quality of data on the sequelae of maltreatment evolves sufficiently to allow for stronger causal statements (e.g., about the role of emotional neglect in the development of emotional damage), then the legal thinker, the clinician, and the researcher can begin to speak the same language, and the three sets of definitions will begin to converge.

Sequelae of Child Maltreatment: Initial Studies

Empirical research on the cognitive, linguistic, socioemotional, and social-cognitive sequelae of child maltreatment is a very recent phenomenon. In addition to the contributions they can make to the formulation of an integrative developmental theory, studies of the sequelae of maltreatment are very important for enhancing the quality of clinical, legal, and policy-making decisions for maltreated children (Aber & Zigler, 1981; Cicchetti & Aber, 1980; Juvenile Justice Standards Project, 1977). Decisions concerning such issues as whether to report a child as maltreated, whether to remove coercively a child from the home, how to develop services to meet the specific psychological needs of maltreated children, and how to evaluate these service efforts would all benefit from a solid and sophisticated database on the developmental sequelae of maltreatment (Aber & Cicchetti, 1984; Cicchetti *et al.*, 1978; Wald, 1975; Wald, Carlsmith, Leiderman, & Smith, 1983; Wald, Carlsmith & Leiderman, 1988).

When there are prominent and pervasive disturbances in the parent–child–environment transaction, the child is at a greater risk for suffering the negative consequences of the "continuum of caretaking casualty" (Sameroff & Chandler, 1975). In a review and critique of the available research on the social, emotional, and cognitive developmental consequences of maltreatment, Aber and Cicchetti (1984) highlighted some of the methodological inadequacies of the initial studies. Before the late 1970s, the vast majority of research conducted on the developmental sequelae of maltreatment was atheoretical, cross-sectional, and often severely flawed conceptually and methodologically (e.g., no control groups; improperly matched comparison samples; inappropriate measures; little or no attention paid to the child's developmental status during data analyses; and "nonblind" experimenters and coders assessing the children's functioning). Because most of this research was atheoretically conceived and adevelopmentally executed and interpreted, these studies primarily presented a great deal of unintegrated data on the clinical problems, social difficulties, and poor school performance of maltreated children (see Aber & Cicchetti, 1984).

Not surprisingly, the conclusions emanating from these various reports often contain apparently contradictory information (e.g., maltreated children are described as hypervigilant or less perceptive, and aggressive or withdrawn). Thus, in the absence of an overarching developmental theory, it was difficult to make definitive statements about the social, emotional, and cognitive consequences of maltreatment. Moreover, the lack of a sound theory to guide the research programs made it difficult to put even the findings from the well-designed studies to scientific or to practical use. Recently, Gersten *et al.* (1986) reached a similar conclusion with respect to the research conducted on the psycholinguistic development of maltreated children.

The Influence of Current Etiological Models upon the New Wave of Studies on the Developmental Sequelae of Child Maltreatment

Increasingly, theorists and researchers conceptualize children's developmental outcomes as having multiple, interrelated causal factors, rather than as being direct outcomes of singular antecedents. An adequate model for conceptualizing maltreatment phenomena must be complex and developmental, allowing for multiple pathways to adaptive and maladaptive outcomes. Further, such a model must transcend the simplistic linear "main effects" models that have been common in the psychological study of maltreatment.

The recognition that parental psychopathology and environmental stress were neither necessary, nor sufficient, factors to cause child maltreatment (e.g., Gelles, 1973; Parke & Collmer, 1975), led some theoreticians to develop more integrative, *interactive* etiological models of child maltreatment (Parke & Collmer, 1975). Although these models were an improvement over linear models in that they integrated two mutually influencing sets of variables, interactive models present a too static depiction of the developmental processes leading to maltreatment. Maltreatment is held to be a function of two variables: parental psychopathology and stress. The interaction between these factors is held to be additive. Psychopathology places a parent at one of several degrees of risk, and all that is required to cause an incident of child abuse is a certain amount of environmental stress. Moreover, according to this model, the result of this interaction between psychopathology and stress at one point in time does not enter into the determination of the parent's behavior at a later point. Clearly, however, this discontinuity is unsatisfactory. What we should like to see in our model of child maltreatment is the parent's state at one point in time (psychopathology, psychological organization, and so on), as well as the state of the parent's environment (family organization and dynamics, interpersonal relationships, psychoecology in the workplace, and so on), also entering into the determination of the parent's state at a later point, along with psychopathology and stress.

Belsky (1980) has developed the best system of ecological integration of the multiple causes of child maltreatment. Psychopathology in parents, child characteristics that may potentiate maltreatment, patterns of dysfunctional family interaction, stressful social forces, and abuse-promoting societal values are integrated into one model incorporating the ecological perspective of Bronfenbrenner (1979) with ontogenetic developmental thinking. Belsky's model has four levels of analysis into which most of the empirical research on child maltreatment can fit. The four levels are: (1) ontogenetic development, (2) the microsystem, (3) the exosystem, and (4) the macrosystem. *Ontogenetic development* refers to the psychological characteristics of the parents (skills, beliefs, and personality characteristics). The *microsystem* is the family setting and its dynamics. The *exosystem* is the most immediate social network in which the family members are incorporated (jobs, schools, churches, extended family, and informal networks). The *macrosystem* includes the current state of the society as it pertains to maltreatment (mores, laws, and cultural norms).

Using the ideas of such ecological thinkers as Belsky (1980) and Garbarino and Gilliam (1980), new empirical projects can be designed to collect a theoretically balanced set of data, which can address multiple levels within the same studies. Such investigations will lead us to understand the feedback patterns that exist among the levels.

Another theoretical perspective that holds promise for elucidating our understanding of child maltreatment is the transactional model. In 1975, Sameroff and Chandler proposed a biological model of development that took into account the interrelations among dynamic systems and the processes characterizing system breakdown. Focusing on the concept of risk factors, Cicchetti and Rizley (1981) have extended the transactional model to examine the etiology and intergenerational trans-

mission of child maltreatment. Risk factors were classified into two broad categories: *potentiating factors,* which increase the probability of maltreatment, and *compensatory factors,* which decrease the risk of maltreatment. Under each category two subgroupings were distinguished: transient, fluctuating, "state" factors, and more permanent, enduring conditions or "trait" factors.

Long-term vulnerability factors included all relatively enduring factors, conditions, or attributes that serve to potentiate maltreatment. These may involve parental, child, or environmental characteristics. Vulnerability factors may be biological in nature; historical (e.g., a parent with a history of being maltreated); psychological (e.g., poor frustration tolerance, psychopathology on the part of parent or child, or high trait levels of aggression and anger); and sociological (e.g., inadequate social networks or chaotic neighborhoods).

Transient challengers included the short-term conditions and stresses. Significant stressors include loss (of status, a job, or a loved one), physical injury or illness, legal difficulties, marital or family problems, discipline problems with children, and the emergence of a child into a new and more difficult developmental period.

Long-term protective factors included relatively enduring or permanent conditions that decrease the risk of maltreatment. Examples of likely protective factors include parent and child "desirable" attributes, such as good temperament, high intelligence, flexibility and adaptability, enduring good health, a parent's history of good parenting, good education, and secure quality intimate relationships between the parent figures.

Transient Buffers included factors that may protect a family from stress. Probable buffers include sudden improvement in financial conditions, parent's finding desired work, child's entry into school or daycare, periods of marital harmony, and a child's transition out of a difficult developmental period.

Cicchetti and Rizley (1981) argued that it is necessary to examine both positive and negative "risk" factors in order to understand the occurrence of maltreatment and the specific forms it will take. There are multiple etiologies for child maltreatment. Probably all combinations of vulnerable parents, vulnerable children, and environmental challenges exist in families. According to the transactional model, maltreatment is expressed only when potentiating factors override compensatory ones. It is then that an act of abuse is committed or a maltreatment condition is allowed to begin.

Recent Research Studies on the Developmental Sequelae of Child Maltreatment

Influenced by the appearance of these more sophisticated etiological models, research on the consequences of child maltreatment has improved significantly. Next, we will describe the current status of knowledge on the developmental sequelae of maltreatment.

Cognitive and Social-Cognitive Development

Very little is known about the specific nature of the cognitive deficits in maltreated children. Many studies have shown decreased performance on intelligence tests in maltreated children (Aber & Allen, 1987; Barahal, Waterman, & Martin, 1981; Hoffman-Plotkin & Twentyman, 1984).

In an important longitudinal study, Egeland and Sroufe (1981) found that children of psychologically unavailable mothers made precipitous declines in their cognitive functioning (i.e., over 40 points) between the ages of 9 and 24 months. This decline was more severe than in any of the other maltreating groups in their study, including the offspring of neglecting mothers. These results suggest a relationship between affective nurturance and cognitive development that goes beyond the deficits of understimulation (neglect) *per se.*

Barahal *et al.* (1981) found that six- to eight-year-old abused children had a more external locus of control and were less able to understand complex social roles than their nonmaltreated peers. Furthermore, they were more inaccurate than controls in their ability to identify others' feelings or to take the perspective of another. However, the strength of these findings decreased when the effect of IQ was partialled out. Contrary to the popular view that low IQs result in decreased ability in other areas of ego functioning, Barahal *et al.* (1981) suggested that low social-cognitive skill levels lead to decreased ability to perform on intelligence tests. They concluded that therapeutic and preventive efforts should target social sensitivity and perspective-taking skills in maltreated children and their parents.

Smetana and Kelly (1989) proposed a broad definition of social cognition, including three domains of developing social knowledge: *persons as psychological systems* (psychological knowledge), *social relations* (moral reasoning), and *social systems* (societal knowledge). A small amount of re-

search has been done in each area. Frodi and Smetana (1984) predicted enhanced social sensitivity in abused and neglected children based on the hypothesis that they are hypervigilant to parental anger as a defensive strategy. No differences were found between any of the groups. In an investigation of moral reasoning, Smetana, Kelly, and Twentyman (1984) found no overall differences in the internalized standards of behavior between abused, neglected, and control children. However, they did find that moral judgments of abused and neglected children were different from those of controls only in areas that are closely related to their own experiences of maltreatment. These findings suggest that maltreated children may be more sensitive to the wrongness of transgressions with personal salience. This speculation is interesting when considered simultaneously with the knowledge of the increased incidence of aggression among maltreated children (Aber & Cicchetti, 1984). If these children are apt to judge aggression as wrong while frequently becoming aggressive themselves, it bodes poorly for a healthy cognitive integration of their self-systems.

Social-Emotional Development

Affective Expression

Abnormalities in the development of affective communication between maltreated infants and their caretakers have been studied by Gaensbauer and his colleagues. Gaensbauer and Sands (1979) identified six patterns of distorted affective communications from infant to caretaker, including affective withdrawal, lack of pleasure, inconsistency/unpredictability, shallowness, ambivalence/ambiguity, and negative affective communications. In a subsequent study, Gaensbauer, Mrazek, and Harmon (1980) delineated four affective patterns that appeared to be relatively consistent and that could represent the predominant dysfunctional communicative patterns of mother–infant dyads. These four groups were labeled as *developmentally and affectively retarded, depressed, ambivalent/affectively labile,* and *angry*. Although the direction of causality of these atypical communication patterns remains ambiguous, it is apparent that deviant styles of affective displays, decreased responsivity, and poor quality interactions tend to characterize maltreating dyads. The work of Frodi and Lamb (1980) indicates that maltreating parents have different psychophysiological responses to the cries of infants, thereby suggesting that these parents may be less effective than nonmaltreating parents in responding to the affective expressions of their infants. However, the mutually reinforcing nature of the inadequacies in the infant's communicative system, and also the differential impact of emotional displays upon the contingent, sensitive, responsiveness of the caregiver, may serve to perpetuate the deviant patterns of interaction in such dyads. Distorted interactions may result in atypical developmental outcomes in the emotional and behavioral repertoire of the maltreated infants. Thus, it is essential that research be directed toward illuminating the transactional nature of maltreatment (Cicchetti & Rizley, 1981), focusing on the developmentally salient emergence, expression, mediation, and control of affective states.

The Development of Attachment

Since the advent of Bowlby's (1969) seminal exposition on attachment theory, there has been general assent among developmental psychologists that the establishment of secure attachment relationships between an infant and his or her caregiver is one of the primary tasks during the first year of life. According to Bowlby's formulation, the attachment relationship has a physical and a psychological function. A number of diverse infant behaviors, such as smiling, vocalizing, and clinging, promote physical proximity to and contact with the attachment figure. In addition, the relationship has the psychological set-goal of "felt security" that will enable the infant to explore the social and the inanimate worlds (Sroufe & Waters, 1977).

Contemporary elaborations of attachment theory have emphasized this psychological function of the attachment relationship, stressing the enduring affective tie between infant and caregiver (e.g., Ainsworth, 1973; Sroufe & Waters, 1977). To this end, rather than focusing on individual behaviors emitted by the infant, the organization of the attachment behaviors is considered and the quality of the relationship is seen to be dependent upon the quality of interaction between the dyad during the first year of life. The widely employed "Strange Situation" procedure was developed by Ainsworth and Wittig (1969) as a means by which to assess the quality of the attachment relationship. The infant's responses to a new room and an unfamiliar female stranger, both in the presence and in the absence of the caregiver, are appraised (see Ainsworth, Blehar, Wa-

ters, & Wall, 1978, for a detailed elaboration of this technique).

Based upon the organization of infant behaviors during the Strange Situation, infants are classified into one of three categories. Infants in Groups A and C are considered to be insecurely attached to the caregiver and will either avoid her (Group A) or manifest angry, resistant behavior alternating with proximity-seeking or passive behavior (Group C) upon reunion. In contrast, the securely attached infant (Group B) will use the caregiver as a secure base from which to explore the environment. If distressed, the infant will first approach the caregiver to seek comfort and will then return to play. If not distressed, the infant will greet the caregiver positively and will actively initiate contact. Approximately 70% of all nonclinical samples of infants are securely attached, whereas 30% (20% A, 10% C) are insecurely attached to their primary caregiver (Ainsworth *et al.*, 1978).

A number of recent empirical studies have examined the relationship between maltreatment and quality of attachment (Crittenden, 1988; Egeland & Sroufe, 1981; Schneider-Rosen & Cicchetti, 1984; and Schneider-Rosen, Braunwald, Carlson, & Cicchetti, 1985). The important similarities among these four studies are their adherence to a theoretically derived focus on this stage-salient developmental task of infancy and their collective employment of the Strange Situation procedure as a means of assessing attachment. These uniformities permit increased confidence in the consistent finding by all four studies that maltreated infants are significantly more likely to be insecurely attached to their caregivers than comparison infants.

Schneider-Rosen and Cicchetti (1984), for example, found that in a sample of eighteen 19-month-old maltreated infants, 12 (67%) were classified as insecure (Group A = 7, Group C = 5), whereas 6 (33%) were classified as secure (Group B). In contrast, 5 (26%) of the 19 matched comparison infants were classified as insecure (Group A = 2; Group C = 3), whereas 14 (74%) were classified as secure. This marked group difference clearly demonstrates the deleterious impact that maltreatment may have upon this developmental task of infancy.

Crittenden's (1988) investigation addressed the somewhat surprising finding of the other investigations that some maltreated infants appear to be securely attached to the caregiver. When Ainsworth's A/B/C system was used to classify the infants' behavior in the Strange Situation, it appeared attached to their caregivers (Group B). However, Crittenden derived a new classification (A/C) characterized by anxious behavior combined with modthat a number of maltreated infants were securely erate-to-high proximity-seeking, avoidance, and resistance. When this additional classification was used, *none* of the neglected or abused infants were classified as secure (but 50% of the problematic infants and 88% of the comparison infants were so classified). These classifications of caregiver–infant attachment were associated with distinct patterns of behavior on the part of both members of the dyad, such that the maltreating mothers were less sensitive and responsive, more hostile and/or inconsistent with their infants, whereas the maltreated infants were characterized as being more difficult or passive than the comparison infants.

Crittenden (1988) suggested that some maltreated infants may receive a secure classification by appearing pseudocooperative, and their caregivers may appear pseudosensitive, but in these cases the dyad is fearful of really challenging the relationship. However, Crittenden argued, the true nature of the relationship is not captured by a forced classification as A, B, or C. Thus, the addition of the A/C group permits a clearer characterization of the infants' behavior.

Additional refinements in the attachment coding system have lead to the identification of a new attachment pattern, known as "disorganized/disoriented" or "Type D" (Main, Kaplan, & Cassidy, 1985). Infants classified as "Type D" are characterized by fear or wariness of their attachment figure, dazed and disoriented facial expressions, and contradictory attachment behaviors belonging to mutually exclusive attachment categories. For example, many "D" babies will behave like a Type A and a Type C infant. Approximately 10% to 15% of nonclinical samples of middle-class and lower-class infants cannot be classified as A, B, or C and fit the D pattern. In a recent study, Carlson, Barnett, Braunwald, and Cicchetti (1989) studied the attachment relationships of a group of 12-month-old maltreated and demographically matched nonmaltreated comparison infants. Carlson *et al.* (1989) found that approximately 80% of the maltreated infants were categorized as Type D. Refinements in the attachment classification system have allowed for a more sensitive understanding of the problem in some maltreating dyads.

Using a modification of the Strange Situation procedure, Lamb, Gaensbauer, Malkin, and Schultz (1985) found that children who were mal-

treated by their primary caregiver were more likely than nonmaltreated children to develop an insecure attachment with their biological mothers, and to a somewhat lesser extent, with their foster mothers. For children maltreated by someone other than the primary caregiver, this finding did not hold up.

Longitudinal studies by Egeland and Sroufe (1981) and Schneider-Rosen *et al.* (1985) have demonstrated the instability of attachment classifications of maltreated infants, particularly those who were classified as secure upon the initial assessment. For example, in the study by Schneider-Rosen *et al.* (1985) of the four maltreated infants who had a secure attachment at 12 months of age, three shifted to insecure attachment relationship by 24 months (2 A's and 1 C). The results suggest that the early secure relationships, where they exist, are potentially transient in nature. These changes in quality of attachment may occur when the ongoing transaction between the infant and the caregiver is disrupted in any way. The durability of the relationship may be unable to withstand the stress of factors either external to the dyad (e.g., poverty, family illness) or within it (e.g., the experience of maltreatment).

This finding corroborates that of Egeland and Sroufe (1981), who also found that the attachment classifications of maltreated infants were less stable than comparison infants over a period of 6 months. The changes in the attachment classifications that occurred were more likely to be from secure to insecure, or insecure to insecure, than from insecure to secure. There were four groups of infants, defined by maternal behavior and attitude toward their infants: psychologically unavailable ($n = 19$), physically abusive ($n = 24$), hostile/verbally abusive ($n = 19$), neglectful ($n = 25$). Of these four groups of infants, a substantial number had also been physically abused. In addition, there was a control group of 85 infants who received adequate care. When making the attachment classifications, these investigators included a Group D.

The many comparisons that were made between the four groups of maltreated infants and the comparison group of infants all demonstrate that the maltreated infants were significantly less likely to be securely attached to their caregivers, and more likely to shift attachment classifications over time. For example, among the infants of the psychologically unavailable mothers who were not physically abused, the number of securely attached infants fell from 57% at 12 months to 0% at 18 months, whereas the number of insecure/avoidant (Group A) attachments rose from 43% to 86% and 14% shifted to Group D. In comparison, there was a slight increase in the number of secure attachments (67% to 71%) and slight reductions in the number of insecure/avoidant (18% to 16%) and insecure/resistant (15% to 13%) attachments among the control infants from 12 to 18 months. These results support those of the Schneider-Rosen *et al.* (1985) study and highlight the stability of the attachment classifications of the nonmaltreated comparison infants. This investigation is also important because the results differentiate among the effects of various forms of maltreatment on infant emotional development.

The Development of the Self

Schneider-Rosen and Cicchetti (1984) observed 19-month-old maltreated children and matched nonmaltreated infants in the Strange Situation and in the standard mirror-and-rouge paradigm (Lewis & Brooks-Gunn, 1979) to investigate the hypothesis that qualitative differences in the quality of the attachment relationship could be related to individual differences in the emergence of visual self-recognition, one potent indicator of the emergence of the self system and of the beginnings of individuation. When data for the entire sample of infants were analyzed, it was found that securely attached infants were more likely to show visual self-recognition earlier than insecurely attached infants. However, an independent analysis of each group of infants uncovered a differential pattern of results. Ninety percent of the nonmaltreated infants who recognized themselves were securely attached. In contrast, among the maltreated infants who recognized themselves, there was not a significant relationship between visual self-recognition and quality of attachment. Moreover, an analysis of the affective reactions of infants to their rouge-marked noses revealed that the nonmaltreated infants were likely to show an increase in positive affect following inspection of their faces, whereas the maltreated infants were likely to exhibit neutral or negative affect to this situation.

In addition to studies of visual self-recognition, investigations of the "self-system" in maltreated children have been conducted by researchers interested in internal state/emotional language. Previous research in middle-class samples has shown that internal state words first emerge during the second year and burgeon during the third. By 28 months, the majority of children have mastered verbal labels for perception (i.e., the five senses), physiological states, volition, and ability;

more than half discussed emotions, moral conformity, and obligation; whereas only a few had begun to talk about cognition (i.e., thought processes). Children also become increasingly able to use internal state labels for both self and other, reflecting a growing awareness of self as distinct from other (Bretherton & Beeghly, 1982).

Beeghly, Carlson, and Cicchetti (1986) and Cicchetti and Beeghly (1987) investigated internal state language in 31-month-old maltreated and nonmaltreated toddlers from welfare-dependent homes interacting with their mothers in the laboratory. Two questions were of particular interest: Do lower socio-economic status (SES) children use internal state language similarly to that reported for middle-class children? What impact does early maltreatment have on low SES children's use of internal state language?

Considerable heterogeneity in internal state language production was seen for both maltreated and nonmaltreated 30-month-olds. For both groups, the diversity of internal state words, the flexibility of attributional focus, and the degree of decontextualization in use were associated with indices of general linguistic maturity, supporting similar observations reported for middle-class children. However, although maltreated and nonmaltreated children did not differ significantly in receptive vocabulary, significant group differences were found for productive and internal state language variables. Maltreated toddlers used proportionally fewer internal state words, showed less differentiation in attributional focus, and were more context-bound in the use of internal state language than their nonmaltreated peers. In contrast, the maltreated and nonmaltreated children did not differ significantly in the categorical content of their internal state language (e.g., words about perception, volition, and the like), with two exceptions. Nonmaltreated children produced proportionally more utterances about physiological states (hunger, thirst, states of consciousness) and more utterances about negative affect (hate, disgust, anger, bad feelings). For the most part, the distribution of words in each category was markedly similar to that seen in middle-class 28-month-olds. Children spoke most about volition and perception, and least about cognition (thought processes).

Analyses of the maternal interview data yielded similar patterns of results. Maltreating mothers reported that their 30-month-olds produced fewer internal state words and attributed internal states to fewer social agents than did nonmaltreating mothers, corroborating the observational findings. In support of the validity of the interview data for use with low SES mothers, reported child internal state language was significantly correlated with observed child internal state language production (average $R = .50$).

The results of the maternal language interview revealed that, with very few exceptions, the maltreated toddlers produced far fewer internal state words than did the middle-class nonmaltreated youngsters of the same age (Bretherton & Beeghly, 1982). In contrast, the percentages of *nonmaltreated* children reported to use different categories of internal state language were markedly similar to that reported for middle-class children. One exception was the moral judgement/obligation category. More middle-class children used this category than either maltreated or nonmaltreated children (65% vs. 34%, 36%). Interestingly, this is the one category in which the maltreated children seem to be on par with the nonmaltreated children. Similar patterns of results were observed for children's ability to use internal state words for both self and other. That is, maltreated toddlers lagged greatly behind their nonmaltreated comparisons in the use of internal state words about the self and other individuals. The lower SES nonmaltreated children were very similar in this capacity to nonmaltreated middle-class youngsters.

Language Development

In the past decade, much theoretical and empirical work has addressed the question of environmental influences on the development of language (Bates, Bretherton, Beeghly-Smith, & McNew, 1982; Bretherton, Bates, Benigni, Camaioni, & Volterra, 1979; Snow & Ferguson, 1977). Developmental psycholinguists have emphasized the importance of the acquisition of communicative and discourse skills (Bates, 1976; Bruner, 1975; Dore, 1974).

Observational studies comparing maltreating and nonmaltreating mother–child dyads have consistently yielded differences in interactive behavior along dimensions critical to the ontogenesis of these "pragmatic" skills (Bates, 1976). Burgess and Conger (1978), in a home study investigating the communication patterns of abusive, neglectful, and nonmaltreating children, found that the mothers in the two maltreating groups conversed less with their children than did the comparison mothers. Furthermore, neglectful and abusive mothers were more negative, more controlling, and less positive than the nonmaltreating mothers in their verbalization.

Aragona and Eyberg (1981) found that, in a laboratory play session, neglecting mothers gave less verbal praise and acknowledgment and more criticism and commands than did mothers of behavior problem, nonmaltreated children. Bousha and Twentyman (1984) showed that the rate of mother–child interaction was much lower for neglecting than for comparison mothers. Even though this result was not especially surprising, it is noteworthy that the incidence of maternal, verbal, instructional interaction was especially suppressed in neglecting mothers. Likewise, Wasserman, Green, and Allen (1983) reported that abusive mothers were significantly less likely to initiate play interactions with their infants, far more likely to ignore them, and less likely to employ verbal means to instruct the infant about his or her environmental surroundings.

In one of the few studies relating the maltreated toddler's communicative development to the mother's verbal style, Westerman and Haustead (1982) compared the conversations between an abused child and the natural mother to those of the abused child and the foster mother. These investigators found that the abusive natural mother was less able than the foster mother to utilize linguistic forms that facilitate communication, thereby placing excessive burdens upon the child for managing the dialogue.

Gersten and her colleagues (1986) studied the communicative development of maltreated 2-year-olds and a matched comparison group of lower-class nonmaltreated youngsters. These investigators observed these toddlers in a semistructured and an unstructured play interaction with their mothers. Contrary to expectations, they found no significant differences between the two groups in their communicative behavior. In light of the current controversy surrounding the role of environmental influences on developing language skills, the failure to document a relation between the experience of maltreatment and communicative functioning is noteworthy. These investigators put forth two interpretations of these data. The first is that early language development is so highly canalized that variations in interactive experience are of little consequence. This interpretation is consistent with the view that language acquisition may proceed on the basis of minimal environmental input, thereby unaffected by variations in social experience. It may be that the evolutionary fit between mothers and children is so good that all mothers do what must be done in order to get communication underway (Bates *et al.*, 1982). However, this interpretation does not preclude the possibility that *later* developments in language may be adversely affected by environmental conditions. Support for this interpretation may be found in the large numbers of school-aged maltreated children who have been described as having communicative difficulties, particularly with their discourse skills (e.g., Blager & Martin, 1976).

Furthermore, it is possible that the influence of maltreatment on communicative behavior may become more apparent as the child attempts to achieve autonomy during the third or fourth years. At that time, certain patterns of communicative behavior may emerge that reflect coping mechanisms used to deal with the maltreating environment. For example, Schneider-Rosen *et al.* (1985) described how some maltreated children avert gaze, minimize face-to-face contact or physical closeness, and rarely initiate social exchanges with an unresponsive and unpredictable caregiver. Similarly, the child may adopt styles of communicative behavior that reduce the likelihood of prolonged interaction or subsequent abuse. For instance, an abused girl in the sample of Gersten *et al.* (1986) directed almost all of her utterances to a doll or toy rather than to an overtly hostile mother. Viewed in this light, the communicative deficits noted in populations of maltreated school children may stem from socioemotional rather than linguistic difficulties.

In contrast, Gersten *et al.* (1986) found that attachment security was highly related to variations in language performance for maltreated and for nonmaltreated children. As a group, securely attached toddlers demonstrated a more elaborate vocabulary and used syntactically more complex language than insecure toddlers. The relative reliance on particular *types* of communication by the two groups was examined using the fundamental categories of utterances. Resulting analyses revealed differences on four of the separate categories, and on one of the composite categories. Securely attached toddlers used a significantly greater proportion of "nominals" (e.g., truck, ball), "descriptors" (e.g., pretty doll, blue block), and utterances describing the activities and feelings of other persons (e.g., You sad mommy? You make a house?). On the other hand, insecurely attached toddlers used a greater proportion of "exchanges" and "filler." These types of utterances are relatively content free and serve primarily to mark turns during conversation without supplying novel information. These data suggest that the verbalizations of securely attached toddlers contained proportionately more content language and less low-quality language material.

These results cannot be explained merely on

the basis of differences in overall cognitive ability. There were no significant group differences found either between maltreated and comparison toddlers or between securely and insecurely attached toddlers. Since it is conceivable that the effects of adverse environmental influences may only appear once the basic communicative patterns have been more firmly established (Bretherton *et al.*, 1979), Coster, Gersten, Beeghly, and Cicchetti (in press) chose to study a group of maltreated children at a higher stage of early language development to ascertain if these toddlers may begin to manifest deviations in their communicative processes. They studied two groups of twenty 31-month-old toddlers and their mothers in the same interactional paradigms utilized in the Gersten *et al.* (1986) study. One group was comprised of legally identified maltreated children, and the other was made up of a well-matched, lower-class, nonmaltreated comparison group. In contrast to the Gersten *et al.* (1986) findings, many compelling statistically significant findings emerged in the linguistic behavior between the two groups.

Maltreated toddlers used syntactically less complex language, and were lower on all measures of expressive vocabulary as well as on total number of different words utilized. Since the maltreated and nonmaltreated toddlers did not differ on the total number of utterances they employed, the expressive language differences obtained cannot be attributed to differences in linguistic output. Coster *et al.* (in press) found no differences between the two groups on receptive language. Thus, the 31-month-old maltreated youngsters had less well-developed expressive language than the nonmaltreated toddlers.

In addition to assessing the expressive language of the maltreated toddlers, Coster and her colleagues found that maltreated children likewise showed deficits in their discourse abilities. For example, maltreated toddlers used significantly more "conversational devices" and fewer descriptive utterances than their nonmaltreated comparisons. Moreover, the maltreated youngsters talked considerably less about their own activity and made fewer requests for information. Furthermore, the maltreated toddlers utilized far less decontextualized speech than their maltreated comparisons. Specifically, the maltreated youngsters made fewer references to persons or events outside the "here and now."

Finally, Coster *et al.* (in press) compared the communicative behavior of the mothers of the maltreated and nonmaltreated youngsters. Correlations between the children's mean length of utterance (MLU) and the mothers' total utterances, proportion of eliciting utterances, proportion of descriptive utterances, and proportion of utterances discussing others were examined; however, no statistically significant differences occurred. The findings from this investigation show that major differences in the communicative behavior of maltreated and nonmaltreated youngsters exist by 31 months of age, underscoring the contribution that social and emotional factors play in the emergence of language acquisition.

Development of Peer Relations

George and Main (1979) observed 10 infants/toddlers between the ages of 12 and 36 months in day-care centers that had been especially designed to be therapeutic for physical abuse cases. They watched the children during several types of activity over a 3-month period. They paid especially close attention to the socially directed behavior of the children, looking for instances of *approaches* to others (adults and same-age peers), *avoidance* of others, and *aggressive* instances. A fourth category was established that concerned what they termed *approach–avoidance* behavior. This last category was used to characterize instances in which the child showed both approach and avoidance behaviors at the same time (e.g., crawls toward the caregiver with head and eyes turned away), or in the same behavioral sequence (e.g., infant creeps toward another child but suddenly veers away before making contact). In order to determine whether or not the patterns of behavior observed in the group of abused children differed from what might be expected of other children who had not been abused, George and Main compared the frequencies observed to those of a well-matched control group of 10 children.

George and Main (1979) found many differences between how physically abused and nonabused children interacted with both peers and caregivers. As a group, physically abused children were less likely to approach the adult caregivers, they were more likely to show avoidance to approaches made by both adult caregivers and their age-mates, they were more likely to direct aggressive responses to adults, and they were more likely to respond to a friendly approach on the part of both peers and adults with approach–avoidance behaviors.

Within these behavioral categories, there were some other differences in the ways abused children's behavior was organized when compared to

the nonabused sample. With respect to approaches made to the caregiver, George and Main (1979) noted that both abused and nonabused children make occasional *spontaneous* approaches to the adult, and that the differences in this aspect of approach behavior do not discriminate between them. However, in response to a *friendly* overture by the adult, the abused children failed to approach as often as did the nonabused children. Further, when the abused group did approach the adult in response to a friendly overture, they were likely to approach indirectly; that is, they would come to the adult from the side or from behind, or, most unusually, by turning around and walking *backwards* to the adult. There were also interesting differences with regard to aggressive behaviors that discriminated the abused from the nonabused group. As noted above, there was a difference in the amount of aggression directed to caregiver. Specifically, abused children were more likely to physically assault or threaten to assault caregivers than were the nonabused children. Further, abused infants physically assaulted other infants nearly twice as often as did the control group infants.

In discussing their results, George and Main noted that the majority of the differences between abused and nonabused infants in their study concern the organization of behavior with respect to caregivers. They also pointed out that avoidance and approach–avoidance behaviors were most often seen in response to friendly overtures made by the caregivers. This is somewhat anomalous since one might expect that a child, even one who might have reason to be apprehensive of adults, would not be hesitant to approach an adult who was being overtly affiliative. George and Main interpreted this intriguing finding by suggesting that avoidance in this circumstance serves the function of enabling the infant to *maintain control* over behavior. In concluding their discussion, George and Main (1979) stated that the behavior of abused children does indeed resemble the behavior of rejected infants from normal samples. In another analysis on the same sample of children, Main and George (1985) examined previously collected behavioral protocols for instances of peer upsets in close proximity to the abused and nonabused children. They made ratings of the focus of children's responses to these upsets and found that the physically abused toddlers were significantly more likely to respond negatively or aggressively to their peers' naturally occurring distress than were comparison children. In contrast to the nonabused children, who were likely to respond to peer distress with simple interest, concerned empathy, or sadness, the physically abused children demonstrated a clear lack of empathic concern for their peers in distress, attacking them both physically and verbally.

Straker and Jacobson (1981) observed 19 physically abused children and 38 nonabused children demographically matched on race, age, sex, developmental stage, and SES. One abused child and two comparison children were seen in a 5-minute free-play group that was videotaped. Straker and Jacobson (1981) found that the abused children engaged in less social interaction than the controls; however, they found no differences between the two groups on hostility.

Hoffman-Plotkin and Twentyman (1984) observed 14 abused, 14 neglected, and 14 matched comparison preschool children. From codings of behavioral observations during free play, they found that neglected children interacted less with peers and were less prosocial to them. Interestingly, the neglected children interacted with their teachers and were as prosocial to them as the controls were. Abused children, though they interacted with other children at the same rate as the nonabused children, were more aggressive in their social interactions with peers.

Howes and Espinosa (1985) conducted four 5-minute observations of maltreated and nonmaltreated children in newly formed and existing peer groups. These investigators compared the peer behavior of maltreated children with that of two demographically comparable groups of nonmaltreated children, one clinic-referred, the other nonclinic referred. Howes and Espinosa found that abused children interacting with peers they knew well were similar to comparison children either in well-established or in new peer groups. In addition, the abused children in established groups and the nonclinic comparisons expressed more positive affect and directed more positive and less negative social behaviors to peers. Finally, the nonclinic referred comparison children were more interpersonally skillful than either abused or clinic-referred nonmaltreated children who were in newly formed peer groups.

Erickson, Egeland, and Pianta (1989) followed up a group of maltreated children from their early infancy (Egeland & Sroufe, 1981) until they entered kindergarten. Neglected children manifested poor peer acceptance, whereas physically abused children were angry at and aggressive with their peers. In addition, children of psychologically

unavailable mothers revealed many behavior problems at school, and sexually abused children were needy and dependent. All groups of maltreated children merited many referrals for special help prior to the end of kindergarten.

Finally, Kaufman and Cicchetti (in press) studied 70 maltreated and 67 demographically matched nonmaltreated school-aged children. These children attended a summer day camp and did not know each other prior to their meeting in the camp. Kaufman and Cicchetti found that maltreated children were rated as having significantly lower self-esteem than their matched comparisons. In addition, behavioral ratings of the maltreated children revealed that they had significantly lower prosocial scores and higher withdrawal scores than the comparison children.

Taken in tandem, the findings of the theoretically guided studies on the sequelae of child maltreatment underscore the devastating impacts that maltreatment phenomena have upon the functioning of these children. Specifically, maltreated children manifest impairments on all of the early stage-salient developmental issues (e.g., the development of a secure attachment, the development of an autonomous self, communicative development, and peer relations). They also show deficits in each domain of development (affective, cognitive, social, social-cognitive, and linguistic), and are at heightened risk for the development of behavior problems and later psychopathology.

Prevention

Preventive programs exist that attempt to target the multiple problems in families at risk for maltreatment. Teen parents, primiparous mothers, parents of handicapped children, and parents living in poverty, among others, have been treated through a variety of intervention modes, such as medical and health education, home visitation by professionals, counseling, and parenting courses (Dubowitz, 1986). Additionally, in many communities, hotlines are available for parents in distress. Typically, these hotlines have accompanying public service advertising campaigns on television and in local newspapers. These advertisements stress the importance and availability of help to parents who feel they are on the verge of abusing their children. The advertisements can be considered to be primary preventive interventions, although no systematic studies have been done to demonstrate their potency in reducing maltreatment in a community. Indeed, few putative preventive efforts have been tested empirically in a well-controlled manner.

One example of a well-designed preventive research methodology is a study conducted by David Olds and his colleagues (Olds & Henderson, 1989). They randomly assigned 400 pregnant women who had at least one of a cluster of risk factors (teenaged, poverty, unmarried, primiparous) to four treatment groups. The first group was a "no intervention" control, which included a screening of the baby at 1 and 2 years of age. The second group included the screenings and the provision of free transportation to the prenatal and well-baby medical appointments. The third group built upon that plan by adding to it 9 visits to the home by a nurse during the pregnancy. The fourth group extended the nurse's visits to the baby's second birthday. The nurses' visits were designed to promote linkage with formal service agencies when appropriate and to enhance the social support of the mothers (and fathers when present). Furthermore, the nurses provided parenting and health education and consultation while emphasizing the need for personal planning. Finally, clarification of values around social and family issues was included. In this study, Group 4 was clearly receiving a relatively broad and intensive family service. Olds and Henderson pointed out the need for multimodal interventions in maltreatment (cf. Cicchetti *et al.*, 1978; Kempe & Kempe, 1978). In this study, the clearest benefits were reaped by the subjects at greatest risk who received the most extensive treatment. Among the poor, unmarried, teenaged mothers, 19% of those in the control group abused or neglected their children within the first two years of life. Those poor, unmarried, teenaged mothers in the long-term nurse-visited group maltreated at the rate of 4%. For teenagers in general, there is no evidence that the program was effective.

Corroborating evidence for scolding rates, provision of appropriate play materials, and avoidance of restriction was found for the poor, unmarried, nurse-visited mothers. Moreover, their children showed a trend toward higher developmental quotients and fewer emergency room visits than their peers in the control group. The findings for the poor, unmarried, teenaged mothers suggest that an intensive and extensive intervention program can prevent child maltreatment and promote good parenting in groups at high risk for parenting dysfunction. Although optimistic, these results also suggest

that the program was less effective for the broader cross-sample of mothers at lower risk levels. Further, this program demonstrated that only the very intensive, expensive, and long-term treatment program had important effects.

Conclusion

Although great advances have been made in the last decade in defining child maltreatment and in understanding the causes, consequences, prevention, and remediation of child maltreatment and family dysfunction, there remain serious gaps in our knowledge. In the future, researchers, theoreticians, and clinicians must address a number of critical issues if they are to form the type of data base that is needed to guide clinical, legal, and policy decision making. Thus, we close this chapter by making a series of 12 recommendations for improving this existing state of affairs.

1. More large-scale longitudinal research on the consequences of maltreatment should be conducted.

2. Research should be carried out in multiple contexts (e.g., the home, in the laboratory, at school, and so on).

3. Researchers should examine the interrelation among developmental domains assessed concurrently (e.g., the relation between emotion and cognition) and predictively (e.g., how the quality of children's early self-cognitions will affect their later adaptation).

4. Attempts should be made to identify competently adapting ("resilient") maltreated children.

Because not all maltreated children manifest developmental difficulties and/or psychopathology, it is important to uncover the processes whereby these disadvantaged children attain competent outcomes.

5. Researchers should study the links between maltreatment and current and future developmental psychopathology.

Retrospective studies implicate child maltreatment in the histories of many forms of child and adult psychopathology (Bemporad, Smith, Hanson, & Cicchetti, 1982; Rogeness, Amrung, Macedo, Harris, & Fisher, 1986; Rutter & Giller, 1983). Although several recent studies have examined the contemporaneous relation between maltreatment and either behavior problems or psychopathology (Oates, 1984; Salzinger, Kaplan, Pelcovitz, Samit, & Krieger, 1984), additional studies are needed to map out the pathways from maltreatment to either a positive or psychopathological outcome.

6. Studies focusing on maltreating parents from a developmental perspective are needed to complement the proportionately larger knowledge base on the maltreated children.

Within the transactional model, the relatively rich corpus of data on children and the burgeoning interest in broad environmental/ecological issues call for a proportional increase in the developmental study of parents.

7. Because virtually nothing is published about maltreating fathers or "father-figures," it is especially crucial to undertake such studies.

8. Further psychometric development and validation of existing classificatory systems are needed in order for researchers, clinicians, and policymakers to reach consensus on a nosology of child maltreatment (Cicchetti & Barnett, in press).

9. Research is needed to demonstrate the effectiveness of various treatment and prevention programs. Very little empirical data support the remedial value of treatment programs, even though many have intuitive appeal.

10. Treatment programs should be multifaceted.

The conceptualization of child maltreatment as having a multifactored etiology suggests that any treatment facility that expects to help maltreating parents and their families must have at its disposal a wide variety of intervention methods to deal with the diverse treatment needs of the clients (see Cicchetti *et al.*, 1978, for a proposal along these lines).

11. Treatment efforts should focus on the entire family, including nonmaltreated siblings.

12. Developmental knowledge of maltreated children and their parents culled from research programs should be used to guide clinical decision making.

A developmental scheme is also necessary for tracing the roots, etiology, and nature of maladaptation so that treatment interventions may be appropriately timed and guided. For example, there are disturbances in internal-state language usage in maltreated children (Cicchetti & Beeghly, 1987). Therapies enhancing recognition of, and verbalization about, personal emotions should help such children develop better impulse control and self-regulation (Cicchetti, Toth, Bush, & Gillespie, 1988). However, altering the child's mode of communication without ensuring the environment's ability to tolerate this change would be a disservice. Thus, intensive work with the parent must supplement the child-focused intervention.

ACKNOWLEDGMENTS

We extend our appreciation to Jennifer White for her assistance in the preparation of this manuscript and to Victoria Gill for typing it. The preparation of this chapter was supported, in part, by grants from the National Center on Child Abuse and Neglect (90-C-1929), the National Institute of Mental Health (1-RO1-MH37960), and the William T. Grant Foundation to Dante Cicchetti.

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CHAPTER 22

Childhood Fears and Phobias

Charles Wenar

Developmental psychopathology means different things to different people. Here it will be defined as "normal development gone awry" (Wenar, 1982). The basic assumption is that all development is one. As a matter of personal preference, some investigators attempt to trace the path of normal development in order to discover what changes take place at different ages and what laws govern such changes. Other investigators, again as a matter of personal preference, are interested in behaviors that represent significant deviations from the norm and in exploring the ways in which variables responsible for normal development have failed to function as they should. Thus, all abnormalities become developmental disturbances. In this view of childhood disturbances normal development is the basic frame of reference for defining and understanding abnormal development. The search for understanding is conducted at the interface between normal and abnormal development, each challenging and illuminating the other.

From a historical perspective, one might have expected a rich literature on childhood phobias as deviant development. Early on, Freud's psychoanalysis of little Hans and Watson's conditioning of fear in little Albert served as prototypical investigations in the first two decades of this century. But actually, the literature is both meager and myopic. With a few exceptions, investigators have studied fears rather than phobias and have been concerned with charting changes in fear-producing stimuli over time and relating such changes to the obvious variables of intelligence, sex, and socioeconomic status. Ironically, the classical study of Jersild and Holmes (1935), conducted over 50 years ago, stands as the most sophisticated research, conceptually and empirically, while providing the richest leads as to the developmental course of normal fears. Two recent pleas to broaden the scope of investigation beyond stimulus charting, one by Graziano, DeGiovanni, and Garcia (1979), the other by Morris and Kratochwill (1983), have had no visible impact upon researchers.

Because of the meager data on phobias, the current attempt to place them within a developmental context will have to rely heavily on the research of Jersild and Holmes (1935), augmented by more recent studies of fears and phobias. Much of the presentation will be speculative, the hope being that psychologists will be alerted to the many issues that have been bypassed by normative studies of eliciting stimuli.

Definition

The definitional issue is a complex one that requires distinguishing among fear, anxiety, avoidance behavior, and phobias, as well as distinguishing between fear and anxiety as behaviors and as constructs, between conscious and unconscious anxiety, and between anxiety as a state and as a trait. Because Miller (1983) has admirably dis-

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cussed such complexities and since the concern here is with development, the definitional knot will be cut arbitrarily by adopting a generally accepted definition of fear and phobias.

There is general agreement that fear involves three response systems: overt behavior, covert feelings and thoughts, and physiological activity. The fact that these three systems may not be highly correlated is a chronic problem for researchers who are hoping for a single absolute measure or, failing that, for multiple measures that are sufficiently related to establish construct validity.

On the stimulus side, there is general agreement that fear is a normal reaction to an environmental threat; Graziano *et al.* (1979) write of "genuine" threat; Morris and Kratochwill (1983) prefer "real"; whereas Miller (1983) refers to "external" threat. Although no one would quarrel with the idea that fear is normal, adaptive, and even necessary for survival, the adjectives "genuine," "real," and "external" are troublesome. Almost unwittingly, the problem is introduced when Morris and Kratochwill (1983) write of a "real or perceived threat" (p. 28), thus raising the question whether threat is in the environment or in the individual or whether it can be in both. Among the list of children's fears, for example, physical injury exists alongside of ghosts and wild animals, with the former posing a genuine threat, and the latter not. In order to understand the role that perception and reality play in eliciting fear, two developmental considerations need to be introduced: the first consideration concerns the inherent fears that are observed during the first year and a half of life; the second concerns socialization during the toddler/preschooler period.

In early infancy, a sudden loud noise is frequently listed among the stimuli that elicit a fear response, although, in and of itself, noise contains no threat to the infant's well-being. Subsequently, fear of separation and of strangers enters the developmental scene. (Labeling these responses as "anxieties" is irrelevant to the issue at hand.) The mere fact that a mother leaves her infant alone can hardly constitute a threat in modern society; neither can the loving grandmother be regarded as threatening even though she elicits a terrified reaction when she goes to pick up her 8-month-old granddaughter. The threatening nature of the stimulus is in the infant rather than in the environment.

From the toddler period on, socialization enters as an important source of fear. In fact, a primary goal of socialization is to prevent the exploring, expansive toddler/preschoolers from exposing themselves to realistic dangers, such as open wall sockets and oncoming automobiles, by inducing fear reactions that are sufficiently strong to produce avoidance behaviors. Although parental behavior mediates the fear, the dangers themselves are external and realistic. The problem is that, at this early age, the child is apt to be indiscriminate in regard to the parents, adopting their unrealistic fears as well as their realistic ones either by direct tutoring or by modeling. Thus, harmless spiders and mice are apt to be feared as well as sharp knives and boiling water.

Phobias are distinguished from fears on the basis of their intensity (i.e., they are out of proportion to the situation), maladaptiveness, persistence, and age or stage inappropriateness with the last criterion obviously being developmental in nature. However, two criteria might be questioned as being applicable to toddlers and preschoolers; namely, the fear cannot be reasoned away and is beyond voluntary control. Even though both toddlers and preschoolers can be reasoned with, it might be unrealistic to expect conscious control to be sufficiently potent to lead to mastery of any strong affect, such as jealousy or rage. Therefore, the fact that phobias cannot be reasoned away does not mark them as pathological.

One final criterion that is questionable from a developmental perspective concerns the duration of the phobia. Phobic symptoms disappear more rapidly in children than in adults; with or without treatment, they may last no more than two years. Because of this time interval, some clinicians have advocated that a clinically significant phobia should last for two years or longer, perhaps on the basis that it would be wasteful to treat a condition which is self-limiting. Yet 2 years for an 8-year-old is a quarter of his or her lifetime and would be comparable to 10 years in the life of a 40-year-old adult. Adult clinicians might judge the length of suffering inordinately long even if the phobia would disappear of itself. Thus, the intensity of suffering and the disruption of the child's life and of the family might be more appropriate considerations than a duration longer than 2 years.

Summary of Evidence

Before attempting to place the literature on fears and phobias in a developmental framework, the findings themselves need to be summarized briefly. The information is based on the excellent reviews of Graziano *et al.* (1979), Miller (1983),

Morris and Kratochwill (1983), and Rutter and Garmezy (1983). Because many basic methodological problems are unsolved and data are meager, few if any of the findings to be reported are definitive.

1. *Age trends in fears.* Although many studies find that the total number of specific fears decreases with age, some report an initial decrease followed by an increase between 9 and 11 years of age. To complicate the picture further, factor-analytic studies show that, although fears of natural and supernatural dangers decline with age, fear of injury and psychic distress, such as social events and examinations, emerge early and continue into adulthood.

2. *Sex differences in fears.* In the infancy/preschool period, there are no marked sex differences in fears. If anything, boys are more anxious than girls. From middle childhood to adulthood, general fearfulness is marginally more common in girls, but specific fears and phobias are more frequent. However, it is not clear whether this latter finding is due to cultural stereotyping, which dictates that girls more readily admit to and express fear than do boys.

3. *Socioeconomic status (SES) and fear.* The findings in regard to SES differences in fears are mixed and contradictory. A basic problem is that lower-class children report specific fears (e.g., whippings, dope dealers), whereas middle-class children report general categories (e.g., car accidents, dangerous animals). There is some suggestion that the fears of lower-class children reflect the realistic dangers of their environment, whereas the fears of middle-class children are more remote.

4. *Age changes and incidence.* In a population of 7- to 12-year-old children, most fears conform to a J-shaped curve with 84% of the population having no fears, 5% to 15% having normal fears, and fewer than 5% having extreme fears, according to mothers' reports. However, the incidence of phobias has been reported to be as low as 0.5% to 1% of the population and as high as 10% to 20%.

5. *Persistence of fears and phobias.* There is evidence that many fears are transient, around half of them disappearing in 3 months and many of them being "outgrown" in 3 years. For the clinician, it is important to note, however, that even normal fears may become severe or may be a prelude to other neurotic disturbances. Richman *et al.* (1982), for example, found that 33% of "neurotic" 8-year-olds were fearful 5-year-olds. Thus clinicians should not summarily dismiss childhood fears as being temporary and trivial. In addition, referrals to clinics tend to be on the basis of how disrupting the fear is to the child's or parent's life rather than on the basis of severity alone; for example, a fear of the dark would stand a better chance of being treated than a fear of insects.

In childhood, phobias are more tenacious than fears, but even most of these improve in 5 years. Adult phobias are more intractable, with only 20% to 30% showing full recovery. Even though many phobias are outgrown in childhood, therapy can hasten the process. Although childhood phobias, like fears, tend to be self-limiting, there can be both short-term and long-term persistence. An intense fear of novelty in 6-month-olds can lead to a heightened fearfulness in early childhood. Agoraphobia which begins from late childhood on is often preceded by other types of phobic symptoms. Animal phobias and fears of physical injury and of psychic stress may persist as adult problems. For the most part, however, adult phobias begin after puberty. Although childhood phobias may lead to adult disturbances other than phobias, those phobias that begin in adulthood tend to continue in that form.

6. *Etiology of fears and phobias.* The etiology of fears and phobias remains a mystery. A preceding noxious stimulus can be identified in certain instances, but it cannot in others. An event that is terrifying to one child will leave another unaffected. Even a clearly frightening experience, such as an auto accident, can fail to produce a lasting fear or phobia. Almost nothing is known of the variables that are responsible for this state of affairs.

7. *Theories of fears and phobias.* As has happened in other areas, traditional psychoanalytic and learning theories have proved inadequate to account for fears and phobias, being supplemented by ethological considerations on the one hand and cognitive variables on the other.

The classical conditioning paradigm has been criticized for a number of reasons. The original findings of Watson and Raynor have been shown to be more tentative and limited than is commonly believed (Harris, 1979). Their results have been difficult to replicate, while the model does not account for the failure to condition the fear response to such neutral objects as wooden blocks. In addition, fears can be acquired vicariously, and neither age changes nor sex differences fit readily into a classical conditioning model. Explanation in terms of reinforcement suffer from the practical problem of being unable to disentangle changes in parental attention that cause the development of a fear from those changes in attention that are a consequence of the fear. Social learning theory has difficulty in accounting for certain fears in terms of modeling,

for example, separation anxiety, the fear of being alone, and fear of the toilet. Psychoanalysts are updating their theory of affect in a number of ways. For example, affect can exist in the conflict-free sphere of the ego and is a vital element in social relations (Emde, 1980). However, such revisions had not been applied to phobias. In addition, reactivity to environmental influences (Freud, 1965) implicitly allows for the possibility that phobias originate according to learning principles, as well as their being based on unconscious conflicts.

Ethologists point to the "preparedness" of organisms to respond with fear to specific stimuli, such preparedness being due to the organisms' evolutionary history. Bowlby (1973) has the most detailed account of fears and phobias from an ethological perspective, particularly in regard to separation anxiety. (For a critique of the concept of preparedness, see McNally, 1987.) Although not strictly in the ethological camp, two other lines of research underscore the contribution of innate or constitutional factors to the development of phobias. In humans, the universality of facial expressions of basic affects is seen as evidence that affective differentiation is due to neurological changes rather than being based on experience alone, whereas the current interest in temperament suggests an innate predisposition to become fearful and phobic.

Cognition plays two rather different roles in accounting for fear. In the first role, fears are the direct result of certain cognitive activities themselves. The most frequently cited exemplar is the discrepancy hypothesis, whereby stimuli that are moderately discrepant from established schemata produced interest but those that are too discrepant produced fear. Another exemplar is Hebb's hypothesis that fear is due to an unfamiliar element in a familiar stimulus. In the second role, the developing child's increasing understanding of the physical and social environment is used to account for the waxing and waning of stimuli that come to elicit fear; for example, the child's increased ability to think in terms of the future makes it possible for him or her to fear future events in addition to current ones, whereas an increasing understanding of social cues helps to overcome the fear of strangers.

Nature of the Fear Response

Developmental Considerations

Reading the stimulus-charting literature, one would think that there is an invariant relation between various stimuli and the fear response. In reality, the relation is often a contingent one. Jersild and Holmes (1935) stressed the unpredictability of the response: ". . . the data show again and again that a child may be afraid on his first contact with a certain event and show no fear when he meets it a second time" (p. 307). Miller, Barrett, and Hampe (1974) made the same point about phobias; a child who was terrified of riding an escalator with his mother readily rode it with a friendly researcher who was there to observe the phobic behavior!

The contingent nature of the fear response has been explicitly recognized by infant researchers who add an intriguing paradox; namely, the same stimulus that elicits fear may also elicit pleasure or interest. The stimulus of falling, usually regarded as innately producing fear, will elicit great glee if a playful father holds the infant in the air and drops him a little ways before holding him securely again. Most important, strangeness and strangers consistently appear in the list of fear-producing stimuli, yet novelty is a prime lure for interested exploration.

One explanation for such paradoxical responses is the discrepancy hypothesis which states that completely familiar stimuli produce boredom, moderately discrepant stimuli produce interest, whereas stimuli too discrepant from familiar ones produce fear. Note that the emphasis is on the stimulus itself. Although there are many problems with the discrepancy hypothesis that need not be detailed here, what is relevant is that it does not fit the observed facts in many instances. For example, research indicates that stranger anxiety is minimized if the infant is sitting on the mother's lap and that it increases significantly if the infant is 2 feet away from the mother. Such data suggest that the affective response is not to the stimulus *per se* but is determined by the context of the stimulus.

Shifting from stimulus to stimulus-in-context vastly complicates the task of accounting for the data, *context* being such a general term that it is difficult to tease out the significant variables and the manner in which they affect the particular response in question. One attempted solution will be described briefly. Sroufe, Waters, and Matas (1974), in an ingenious maneuver, turn confounds into variables—conditions that others might control in order to obtain an uncontaminated measure of fear now become essential contributors to that response. Their analysis of context variables is addressed to infants in the first year of life. In this period, the following five variables are important in determining the fear response:

1. *The presence or absence of the mother, or, in some cases, nearness to the mother.* This is in-

terpreted as a security variable, with the absent or distant mother increasing insecurity and vulnerability to fear.

2. *The level of cognitive development.* Because newborns have no knowledge of environmental events, categorizing such events as "familiar" or "unfamiliar" requires a gradual accumulation of knowledge as to which events occur with sufficient regularity that they come to be cognized as familiar. Only then can other events be cognized as being unfamiliar or strange.

3. *The setting.* One aspect of the familiar-unfamiliar variable is the physical setting. An infant is more prone to fear when in the laboratory than when at home, and is more prone initially in the laboratory than after a 3- to 10-minute familiarization period.

4. *State and immediately preceeding events.* Just as tickling is noxious rather than pleasurable to an infant in a generally irritable state, so an infant who is in a general state of distress is more prone to fear reactions than one who is alert and happy. In addition to such general background factors, the immediately preceeding events also determine the likelihood of a fear reaction; e.g., a stranger appearing after a distressing event is more likely to elicit fear than one who appears after a pleasant event.

5. *Temperament.* Temperament can be regarded as a highly generalized background factor affecting the appearance of the fear response. Child psychoanalysts have long claimed that certain infants are anxiety prone, the implication being that situations which other infants could master might produce excessive fears and phobias in this specially vulnerable population. Bates, Maslin, & Frankel (1985), for example, found that the infant temperament variables of negative emotion (fussy, socially demanding, difficult) and unadaptability/unsociability were significantly related to mothers' perception of anxious behavior at 3 years of age. Although the concept of temperament and the methodological issues involved in assessing it are far from settled (Goldsmith & Campos, 1982), preliminary data support the hypothesis of an early predisposition to be fearful and to develop phobias.

The developmental course of the context variables listed above remains to be charted. Even though they are still operating in middle childhood, they are probably not as situation specific as in the infant/toddler period—the need for security becomes attenuated and dispersed among many people, the environment is more familiar than strange, both the past and future become available as guides to understanding the present and anticipating what will happen next. However, there is always the possibility that new variables will appear as old ones lose their potency. Although little is known concerning context variables in adults, the fact that phobias persist longer than in childhood suggests that such behaviors are less amenable to change as situational factors change.

The introduction of context has its limitations and problems. It does little to solve the mystery of how fears become phobias—or, better, the mystery of the relation between the two. In fact, the dependency of affect on context accounts for the variability of phobias better than for their generation and self-sustaining qualities.

Developmental Reconstruction

A developmental reconstruction differs from charting changes in eliciting stimuli over time. Rather, it involves integrating such data into general knowledge concerning the development of significant personality variables, such as the self, social relations, cognition, and affect itself.

A major obstacle to a developmental reconstruction is that many categorizations of fear-producing stimuli are logical from the adult researcher's viewpoint but are not necessarily relevant to the children's developmental status. The data of Jersild and Holmes (1935), for example, indicate that the frequently used category of "animal" includes animals both real and imaginary, tame and wild, passive and active, even though a small dog on a leash is different from an attacking lion both as stimuli in their own right and as stimuli for children of different ages. The factor-analytic solution to the issue of categorization of Miller *et al.* (1974) involves the same problem of combining psychologically disparate data, so that "going crazy" and "parents getting a divorce" are found alongside "germs" and "dying" in a factor labeled Abstract Physical Injury.

The developmental reconstruction attempted here will rely heavily on Jersild and Holmes's (1935) research because of the detail with which data are reported and, equally important, because their categories lend themselves more to a developmental interpretation than do those of other investigators. The speculative nature of the reconstruction should again be emphasized. Its purpose is to illustrate a way of thinking that makes "development" something different from stimulus charting over time.

The Infancy/Preschool Period

The first group of stimuli might be regarded as eliciting innate fear reactions since they appear ear-

ly in development with no evidence of their being learned. This group includes loud noise, pain, falling, and sudden, unexpected movement. It is worth noting that such stimuli require little cognitive elaboration, the fear appearing and vanishing with the stimulus itself. Strangeness or unfamiliarity, whether it be in regard to people, objects, or situations, elicits fear in the latter half of the first year. As has been noted, the evaluation of a stimulus as "familiar" or "strange" requires a cognitive elaboration that is not necessary with innate stimuli. At times, the fear conforms to Hebb's concept of a familiar object in an unfamiliar guise, such as a mother's wearing glasses or a hat for the first time; at other times, unfamiliarity *per se* appears to be operating. In the early toddler period, animals begin to elicit fear. In certain instances, fear may be in response to the noise and sudden, unexpected movement characteristic of animals. However, even quiet animals may be effective stimuli, perhaps because of their unfamiliarity, although Jersild and Holmes (1935) speculated that there may be an innate fear of animals.

The change from the toddler to the preschool period is marked by a decline in the primitive fears of noise, falling, sudden movement, and strangeness, although fear of pain and of animals have an irregular upward trend. Memory makes fear less a situational response by strengthening anticipation so that children now respond to the threat or the danger of bodily injury and, subsequently, to warnings that are part of socialization. Thus, children increasingly fear traffic accidents and fires in response to parental punishment and other fear-inducing techniques; subsequently, they come to fear doing things parents regard as "bad."

Fear of imaginary animals and characters and fear of nightmares are now developmentally possible as the early preschooler comes to differentiate reality from fantasy and recognizes the latter as a special realm of cognitive activity. A number of investigators have noted this cognitive achievement but have erroneously assumed that separating fact from fantasy explains the imaginary fears themselves. However, fantasy may be pleasant as well as frightening, so the affect accompanying it still must be accounted for. Jersild and Holmes's (1935) examples suggest that ghosts and fantasy animals are viewed as inflicting pain, although more definitive data are needed to test this speculation. It might also be that the fear of strangeness, which declines in reality, continues in fantasy.

Toward the end of the preschool period, there is an upsurge in the fear of robbers and kidnappers. Such fears may well be learned from adults, either by direct tutelage or by imitation. However, preschoolers may have their own interpretation of the threat such people embody, which might differ from the parents' realistic warnings. There is no evidence, for example, that children at this age are concerned with the loss of property, so the fear of robbers may be due to the child's perception of them as strange individuals who inflict pain. On the other hand, kidnappers may play into the fear of separation, which is more congruent with adults' concerns. In regard to the fear of animals that persists in the preschool period, Jersild and Holmes (1935) speculated that it may be due to adult warnings or to imitation of adults' fearful behavior.

Finally, the end of the preschool period witnesses the appearance of fear of personal inadequacy, failure, and ridicule, the examples cited by Jersild and Holmes (1935) involving learning the academic skills of numbers and writing or fear of other school-related functions. Such fears indicate the development of the concept of an achieving self to the point that anticipation of failure is a source of concern. In all probability social influences are important, as well as advances in the self-concept, parents and teachers making the child ashamed of failing, and peers ridiculing ineptness. Typically, it is only during middle childhood that the image of the achieving and failing self becomes sufficiently internalized and autonomous to operate relatively independent of immediate social feedback.

Not all fears can be accounted for in this reconstruction. Fear of the dark, which Jersild and Holmes (1935) differentiated from fear of imaginary creatures, along with fear of being alone in the dark both increase steadily during the period under discussion for reasons which are obscure. In addition, Jersild and Holmes (1935) note that there are fears that are so idiosyncratic that they defy categorization as well as explanation.

Middle Childhood

By middle childhood, the cognitive distortions that characterize earlier periods have been corrected by and large, and the child's basic grasp of the social and physical environment resembles that of the adult. (The descriptive material in this section is based on Bauer, 1976, Derevensky, 1979, and Miller, 1983, as well as Jersild and Holmes, 1935.) Fears follow this trend toward realism: fears of bodily injury, say, from traffic accidents or fires, increase, but fears of ghosts and imaginary creatures decline. It may well be that the fear of robbers is also based on a more realistic understanding of the threat that they pose than it was previously. Fear

of failure increases, although it is never high in comparison with other fears. The old, primitive fear of sudden movement, falling, and strange people all vanish, although the fear of noise persists in response to thunder.

However, the tilt toward realism is not all encompassing. Although the fear of such remote animals as lions and tigers declines, fear of harmless animals, such as rats and mice, ranks high, whereas the fear of snakes far outstrips their actual danger. Fear of the dark and of nightmares also continue. And at least one study (Jersild & Holmes, 1935) presents evidence that the decline in fear of ghosts is compensated for by an increase in fear of fictional characters from stories, movies, and—extrapolating from their data—from TV. Although the data are in need of replication, the finding itself is plausible. Imagination, by its very nature, is tangential to reality since it does not conform to the same rules as those governing the social and physical environment. Thus, imaginary characters can retain their affective charge—both positive and negative—in the face of an increased cognitive realism. It would be interesting to know at what age children, like adults, realize that their fears are groundless and yet continue to be frightened.

In sum, middle childhood, like all other developmental periods throughout the lifespan, is a mixture of realistic and unrealistic fears, only the proportions are changed compared with earlier periods.

Adolescence

Adolescence brings with it new, age-appropriate fears. (Information on adolescent fears comes from Conger and Peterson, 1984, Horrocks, 1976, Jersild, Brook, & Brook, 1978, and Weiner, 1980. There are no data on changes from middle childhood through adolescence, and Angelino, Dollins, & Mech (1956) are among the few researchers to chart changes within the adolescent period itself.) Sexual fears and concerns over money and work enter the developmental picture as would be expected. Adolescent's increased cognitive ability enables them to grasp and be concerned about a social context broader than home and school as well as making it possible for them to project themselves into the future. Thus, fears centering around war and peace make their appearance and dramatically increase between 14 and 18 years of age, along with more personal concerns about growing up and being able to cope with problems that lie ahead.

Adolescence also witnesses an increase in fears that are present only to a minor degree in middle childhood. The striking example is fear of failure or personal inadequacy, expressed primarily in the large number of school-related fears that are present throughout the entire period. Such fears may be partly due to the increased emphasis on achievement in high school, but they are also due to the adolescent's own increased ability to generate relatively stable standards and to be concerned over living up to them. Fear of success is the counterpart of fear of failure, being particularly linked to adolescent girls' sex-role typing, which dictates that they would not be sexually attractive if they showed their superiority to males. Fear of success may be adolescent specific since it has been used to account for girls' decline in achievement in high school; however, it might also be present in an attenuated form in middle childhood and may have been overlooked. As peer relations take on greater importance in adolescence, so concerns over popularity and having friends begin to rise, especially for girls. And the increased self-consciousness of the adolescent is accompanied by an increased fear of looking foolish.

There is evidence that fear of pain and bodily injury, along with fear of animals, declines in adolescence. The first finding is somewhat baffling since the realistic chances of bodily injury might well increase in adolescence as compared with the more protected middle-childhood period. The fear of death, either of the self or of loved ones, continues. Finally, irrational fears, while infrequent, persist: fear of the dark, of storms and noise, of mice and snakes. Fear of the supernatural is infrequent, but adolescence may bring with it new irrational fears, such as fear of cemeteries and of water (i.e., swimming alone, boating, deep water), which are rarely reported in middle childhood.

In sum, fears continue to reflect significant developments in the self, in social expectations and relationships, in cognition, in physical maturation. Although more definitive data are needed, adolescence may also represent a further diminution of irrational fears, although they are not completely exorcized.

Final Comment. It is a psychological truism that the same behavior can have different meanings just as different behaviors can have the same meaning. Thus, the fear of death may be an amalgam of a fear of separation from loved ones, a fear of the strange appearance of the dead, a fear of pain (which is more a fear of dying), a fear of personal, irrevocable nonexistence, each of which has its own developmental timetable.

Or again, Mooney, Graziano, and Katz's (1985) factor-analytic study found that nighttime

fears in middle childhood centered primarily around children's fear for their life or personal safety, followed by fear of separation from or loss of others and, to a lesser extent, fear of supernatural creatures and inherent characteristics of the situation, such as shadows in the room. Other fears would have their own set of underlying meaning and timetables. In contrast to this pluralistic approach to meaning, psychoanalysts claim that there are only a few basic fears that assume different forms—the fear of loss of love and self-esteem, physical injury, the new and strange, disapproval and censure—so that development would consist of progressive variations on the same basic themes. Regardless of which view that is adopted, understanding fears will not significantly advance until psychologists face the difficult task of understanding their meaning and their progressive change in meaning.

Overcoming Fears

Charting fear-eliciting stimuli over time ignores three basic questions about change: (1) Which fears represent new additions to the developmental picture (such as fear of ridicule)? (2) Which fears represent new variations of existing fears (such as fear of vocational failure being a continuation of fear of academic failure)? (3) Which fears disappear from the developmental scene because they have been mastered? The issue of mastery is particularly pertinent to clinical child psychology because understanding conditions leading to the overcoming of fears may furnish clues to understanding the conditions leading to failure to overcome them and to the psychopathologies that are defined in terms of fixation and regression. Once again, the only researchers who have seriously explored the issue of mastering fears are Jersild and Holmes (1935).

During the toddler/preschool period, mothers used a variety of techniques aimed at helping their children overcome fears: explanation and reassurance; setting an example (such as walking into a dark room or petting a feared dog); providing the child with coping techniques (such as letting a fearful child turn off the lights at night); graduating the approach to a feared object; using distractions or counterstimuli (such as having a child ride horsey on a vacuum cleaner when the frightening motor is not running); and forewarning the child in preparation for a potentially frightening experience, such as getting a shot from a doctor. Thus, the mothers were not only ingenious but their techniques have a surprisingly contemporary ring as well. However, they succeeded only fitfully. More typically, children overcame fears on their own; for example, toddlers' and preschoolers' increased understanding of their environment and their increased ability to manipulate and explore were their greatest assets in overcoming the early primitive fears of noise, falling, and strangeness. In fact, Jersild and Holmes (1935) concluded if these early fears are mild, it is wise to let them run their course since they will be overcome more on the basis of the child's own growth and experience than on the basis of adult interventions.

Jersild and Holmes (1935) came to the same conclusion in regard to middle childhood and early adolescence, although the data here are derived from adult recall rather than from direct observation. Once again, it seems that the growing child's increased experience, information, and skill are more potent in eliminating fears than are aids or influences exercised by other persons. However, the authors are careful to note that a number of fears persist into adulthood and continue to be troublesome. There are also those fears that are so intense and painful that they require special corrective measures, although the number of such fears was low in their study.

In recent times, one of the few studies addressing the issue of coping was conducted by Mooney *et al.* (1985) in regard to nighttime fears. They found that the most frequently used technique was internal self-control, such as thinking to oneself that there is nothing to be afraid of or thinking pleasant thoughts; next came social support, such as calling to parents, or using inanimate objects, such as hugging a pillow; praying and avoidance or escape were also utilized but to a lesser degree. These findings suggest that the study of coping mechanisms and their development might be as complex and fruitful as the study of fears themselves.

Since Jersild and Holmes (1935) found that intrapersonal variables were more potent than interpersonal ones in overcoming fears, their admittedly speculative discussion deserves to be presented in more detail. They list three factors within the child that foster mastery.

1. *Growth and general experience.* This refers to the child's increasing ability to understand the environment that progressively counteracts the distortions of earliest childhood, a cognitive progression that has already been discussed.

2. *Direct habituation.* Here children overcome their unrealistic fears by having direct experience with the feared object. As they increasingly discover the unrealistic nature of their fears, children

become increasingly confident of their ability to face them in order to see if they are justified. Jersild and Holmes's (1935) speculation can be buttressed by advances in psychology that were made after their research was done. The most important advances are the recognition of a basic need to explore and the introduction of a concept that variously has been called competence, efficacy, initiative, or autonomy, all of which stress the child as agent of change, rather than the child as passive recipient of environmental influence (Wenar, 1989). Also favoring mastery is the close relation of fear to interest, especially in regard to novel stimuli; as was noted previously, a strange object can either be frightening or intriguing. Consequently, the tendency to avoid is counterbalanced by the tendency to approach.

Freud (1965) stated that there are innate differences among children in terms of their tendency to face and master anxiety or to retreat and protect themselves against it. The idea that temperamental differences predispose certain children to fearful behavior receives support from the research literature (Rutter & Garmezy, 1983). Morris and Kratochwill (1983) introduced an idea similar to Freud's but without the connotation of temperamental differences when they wrote of courage or persistence in the face of intense fear. They further suggested that the child's behavior is the result of the balance between courage and fear. At a more general level, these authors reflect the current thinking that behavior is the result of the relative strength of forces favoring or impeding mastery.

3. *Deliberate counteracting activities.* Here children deliberately set out to face and master fears, for example, by forcing themselves to go into dark places or to dive into water in order to conquer the fear of darkness or diving, respectively. Such deliberate activities can be observed in the early preschool period, although they are relatively rare throughout the entire childhood period.

From Fears to Phobias

The literature is bleakest when it comes to determining the relation between fears and phobias. Weiner (1982) stated that fears shade imperceptibly into phobias, suggesting that the difference is only a matter of degree, and what is known of one can be applied to the other. By contrast, Graziano *et al.* (1979) stated that we do not know whether fears and phobias operate according to identical principles, and one might add that we do not know whether the principles governing phobias change with age. Even if Weiner's more traditional hypothesis were true, it would still leave unanswered two basic questions: Why do fears become intense, and why do they become persistent so that the factors responsible for openness to change—the context variables and the forces favoring mastery discussed above—fail to serve a growth-promoting function? One might expect, for example, that animal phobias would be the most amenable to the corrective influence of increasing cognitive and emotional maturity, yet just the opposite is the case. One might also expect that the fluidity of personality and the potency of parental influences in the second and third year of life would prevent the appearance of sustained phobias during these early years, yet both animal (Sperling, 1952) and insect (Kolansky, 1960) phobias have been observed. To compound the problems, what empirical data there are often blur the distinction between fears and phobias so that, for example, it is impossible to tell whether what is termed an intense or a clinical fear is the equivalent of a formally defined phobia. Consequently, any discussion of the relation between the two is bound to be tentative.

Although fears are unrelated to deviant behavior in a normal population, phobic children have a variety of other problems, usually of an internalizing nature. Both the Children's Behavior Checklist and the Personality Inventory for Children for example, find a close association between phobias and depressive behaviors. There is some suggestion that even though phobic children are more generally disturbed than normal children, they are not more disturbed than children who are referred to clinics for nonphobic disorders (Poznanski, 1973). Phobias, like fears, may be due to a traumatic event, but in other cases, no actual aversive antecedent can be identified (Morris & Kratochwill, 1983); for example, a preadolescent may be terrified of being alone in the house without ever having a significantly negative experience under such circumstances. Thus, like fears, phobias can be the child's own creation as well as being a reaction to a specific experience.

Although disclaiming any intent of unraveling the etiological mystery of phobias, Jersild and Holmes (1935) did obtain data on 16 preschoolers, representing the extremes of a marked lack of fearfulness on the one hand and unusually high levels of fearfulness on the other. They found that both groups were in good physical condition, that there was no difference in the stability and security of the home atmosphere or in the mothers' understanding of the children's needs, and no difference in general friendliness and enjoyment of peer relations. How-

ever, there was a difference in certain personality traits: the fearful children were more frequently described as dependent on adults for help, as being easily upset emotionally, as being timid, shy, and insecure, and as being unable to stand up for their rights on the playground, whereas the least fearful children were described in the opposite terms. This same theme of dependency on parents plus some infantilization is echoed by Poznanski (1973) and Weiner (1982). Such observations and inferences make sense in light of the previous hypothesis that self-confident exploration and the ability to test out the reality of fears are two of the principal means by which fears are mastered. Lack of confidence and a sense of personal ineffectuality or helplessness may also partially account for the close association between phobias and depressive behaviors. Yet all this speculation fails to pinpoint the necessary and sufficient conditions for producing phobias rather than other kinds of disturbances. As usual, Jersild and Holmes (1935) have the last word when they conclude that "the elements which contribute to causing such individual differences in fear behavior . . . are quite intangible and not often expressed by the type of overt behavior which is easily observed and recognized" (p. 284).

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PART IV

Depression

CHAPTER 23

Depression across the Early Life Span

Dennis P. Cantwell

Introduction

This chapter is concerned with depression throughout the early life span; that is, the infancy, preschool, grade school, and adolescent age ranges. Among the issues to be discussed are the following: the differentiation between depressive symptoms; a depressive syndrome and a depressive disorder; the clinical picture and subtypes of depressive disorders across the early life span; developmental issues of the assessment of the depressive syndrome from infancy through adolescence; etiologic theories of depression; and links between depression in infancy, preschool, grade school, and adult life. Finally, the chapter will conclude with some directions for future research in this area.

Depression: Symptoms, Syndrome, Disorder

Before we begin, distinction must be made between depressive symptoms, a depressive syndrome, and a depressive disorder. Lack of precision in terminology has bedeviled not only some of the early research but also some of the more recent research in the study of depression in the early years. The term *depression* has psychiatric and nonpsychiatric usage. In the latter, for example, it is a common term in economics. However, even when used in the psychiatric sense, the term *depression* has been used alternatively at times to describe depressive symptoms, a depressive syndrome, or a depressive disorder.

Depression as a symptom—a sad mood, unhappiness, feeling miserable, feeling blue, feeling down in the dumps—connotes only one aspect of a depressive syndrome or a disorder (i.e., the dysphoric mood). However, depressive symptoms are relatively common across the entire age span, and they may be transient, are often related to environmental events, and may not be part of any psychiatric disorder. In young children, depressive symptoms are relatively common symptoms across many psychiatric disorders. When depressive symptoms occur in preschoolers, grade schoolers, and adolescents (and, indeed, adults) who have some type of psychopathology, they may not constitute either a depressive syndrome or a depressive disorder.

When depression is used to describe a syndrome, the connotation is more than simply a sad mood or a dysphoric mood. A syndrome is generally recognized by a regular coexistence of symptoms that commonly occur together and are not associated by chance. Thus, a depressive syndrome includes not only mood changes but changes in veg-

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etative and psychomotor areas, cognitive changes, and motivational changes.

The syndrome of depression is less common than depressive symptoms. A depressive syndrome may occur as a primary problem, with no preexisting psychiatric, medical, neurological, or environmental antecedents. However, a depressive syndrome may occur in conjunction with a wide variety of medical disorders, such as Cushing's Disease, rheumatoid arthritis, and hypertension—particularly hypertension treated with certain medications that induce a depressive syndrome as a side affect of the drug.

A depressive syndrome may occur regularly following certain types of life stress. A commonly accepted example of this type of syndrome is a grief reaction which has many of the characteristics of a depressive disorder but is best conceptualized as a depressive syndrome occurring as a reaction to loss. The depressive syndrome may also occur in conjunction with and secondary to a wide variety of psychiatric disorders, such as schizophrenia, anxiety disorders, disruptive behavior disorders, and alcoholism. Then, too, a depressive syndrome may occur as part of a depressive disorder, but when depression is used to connote a depressive disorder, more is usually implied. The implication when the term *depression* is used to describe a depressive disorder is not only that there is a depressive syndrome, but that this depressive syndrome has a minimum duration and has caused a minimal degree of functional incapacity. There is also the implication that this disorder is characterized not only by a characteristic clinical picture or syndrome but also that it has a characteristic outcome, a characteristic response to treatment, and that there may be characteristic etiologic factors or correlates that may be biological, environmental, family, or some combination thereof.

The concept of depression as a syndrome and as a disorder has been well studied in adults. In adults, several different depressive disorders have been delineated on the basis of different clinical pictures, and these disorders differ in natural history, in response to various types of psychopharmacological intervention, in biochemical and neuroendocrinological factors, and in family genetic factors. However, there are controversies even with the concept of depressive syndromes and disorders in adults (Andreasen, 1982).

The limits of the concept of affective disorder and the methods of subclassifying affective disorder remain areas of controversy. On the one hand, there is the problem of discriminating between depression and normality to exclude relatively mild, brief, situational disorders, such as those that occur in response to the death of a spouse, from major affective disorders that constitute true psychiatric disorders. On the other hand, there is the problem of discriminating between major depressive disorders and other psychiatric disorders, such as anxiety disorders and schizophrenic disorders. These discriminations do present clinical difficulty at times, but they are considerably better worked out with adults than they are with children and adolescents.

One question we want to address is the prevalence of depressive symptoms, depressive syndrome, and disorder in infancy, preschool, grade school, and adolescent age ranges. We know the least about depressive conditions occurring in infancy. Although there are individual case reports (Harmon, Glicken, & Culp, 1982) there are very few detailed clinical descriptions of depressive syndromes occurring in infancy. Some years ago, a condition called "anaclitic depression" was described by Spitz (1946). What he had observed were infants in an institution who were between the ages of 6 and 12 months and who presented with a syndrome with many features suggesting a depressive disorder that he felt was due to the loss of a mother figure by these infants. Whether this was the etiologic factor in all cases or whether physical illness and deprivation may have played a role in some is questionable. Nevertheless, the syndrome did resemble a depressive disorder. Spitz thought this condition was relatively frequent in children who were raised in institutions. And the studies of the Hampstead group (Freud & Burlingham, 1974) also reported young children separated from their parents during World War II who had depressive-like syndromes or possibly grief reactions. The protest, despair, and detachment sequence that was described by Bowlby (1980) and Robertson and Robertson (1971) also contained a depressive-like picture in the detachment phase. The role of attachment and breaking of bonds in these conditions has been reviewed by Rutter (1972).

Infant psychiatry is a growing field, and it may be that with more careful study, depressive-like syndromes will be discovered in other populations in infancy. Currently, all that can be said is that these syndromes have been described, but they are probably relatively rare. Whether or not they repre sent the same thing as depression that occurs in grade-school-aged children, adolescents, and adults remains an open question (Rutter, Izard, & Read, 1986).

Likewise, systematic studies of psychiatrical-

ly ill preschoolers suggest that a depressive syndrome analogous to that which occurs in adults is relatively rare (Kashani & Carlson, 1987). Cantwell and Baker (1983), in their epidemiologic study of psychiatric disorder in children with communication disorder, most of whom were preschoolers, found that preschoolers with communication disorder had high rates of psychiatric disorder. However, affective disturbance was relatively rare, although depressive symptomatology occurring in the course of other psychiatric disorders was not as rare.

The bulk of systematic research has been with grade-school-aged children and adolescents. For example, the Isle of Wight Study (Rutter, Tizard, & Whitmore, 1970), which was a total population study of grade-school-aged children of 10 and 11 years of age, showed that, in individual interview settings, 13% of that grade-school-aged population were rated as having a depressed mood. Parents and teachers rated 10% to 12% of the same population to be unhappy and miserable. This was a total population study of a nonclinic, nonreferred population, suggesting that depressive symptoms were relatively common in that age range in a nonpatient population. In several studies of clinic populations, Carlson and Cantwell (1979, 1980a, b, 1982) found much higher rates of depressive symptoms in children in the grade school age range.

When the same Isle of Wight children were seen when they were adolescents (14 to 15 years of age), the individual interview with the adolescents revealed depressive feelings in about 40% of the sample—a major increase from the 13% rate shown when they were grade-school-aged children. In the clinic sample studied by Carlson and Cantwell (1979), the base rate was much higher in the prepubertal age range than it was in the Isle of Wight general population. Nevertheless, adolescents who attended clinics showed higher rates of depressive symptomatology than did prepubertal children. The Isle of Wight study also looked at a definite diagnosis of a depressive disorder and found a rate in the population of about 1.5 per 1,000 in the grade school age range and about 17.5 per 1,000 in the adolescent age range. Carlson and Cantwell (1980a) also looked at the same issue, and their data showed that a diagnosis of a depressive disorder was more common in the adolescent age range than in the grade school age range. It should be noted that the Isle of Wight study was done before more modern methods of classification and assessment measures for depression had been developed. Also, the interview with the adolescent was more fully developed in inquiring about affective symptomatology than was the grade-school-aged child interview.

In a more recent epidemiologic study of a total population of 4,500 high school students in Minnesota, Garfinkel (personal communication, 1988), using more modern methods of measurement and diagnostic criteria, found that 10% had a significant depressive disorder. Thus, these data and others do suggest that depressive symptoms are not infrequent in grade-school-aged children and that depressive disorders are less frequent than depressive symptoms, whether the sample group is in the general population or in the clinic population. These data also suggest that the prevalence of depressive symptomatology and a diagnosable depressive disorder rises up in the post-prepubertal adolescent age range. Not only is there an increase but there is also a shift in sex ratio so that adolescent females are more likely than adolescent males to be diagnosed with a definable depressive disorder, whereas the sex ratio is about equal in the grade-school-aged child. More research in this area is sorely needed.

Clinical Picture and Subtypes

There are several separate issues to be considered when discussing the clinical picture and subtypes of depression across the early life span from infancy, to preschool, to grade school, and to adolescence. The first issue is the delineation of the core clinical picture of depression as it occurs in the four age groups. We will not consider infancy any further because there simply is not enough data to make any generalizations. In the preschool age range, the grade school age range, and the adolescent age range, the questions that arise include the following: Are the essential features of depressive disorder in preschool, grade school, and adolescent children the same as they are in adults? If they are the same, are there differences in associated symptomatology in the different age ranges? If there are differences in the core clinical picture that occur as a result of age, are there definable core clinical pictures that can be reliably and validly delineated for the different age groups? This set of questions deals with the depressive syndrome as essentially a homogeneous syndrome or disorder. However, we know from the study of adult affective disorders that there are meaningful subtypes that differ on the basis of clinical picture and that the different subtypes are associated with differences in other areas, such as outcome, response to treatment, biological

correlates, and family genetic factors. Thus, a related question is: Are there meaningful subtypes of the depressive disorders in the various age ranges?

Thus, one key question revolves around diagnostic criteria for different depressive syndromes at different ages, and the other question revolves around the proper classification of depressive disorders throughout the early life span. Some of these issues have been discussed previously by Cantwell and Carlson (1983a,b) and Carlson and Garber (1986).

There are several sets of operational criteria for the diagnosis of depression in adults that have been in common use. The Feighner criteria (Feighner, Robins, Guze, Woodruff, Winokur, & Munoz, 1972; Spitzer, Endicott, & Robins, 1978) and the criteria contained in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) (American Psychiatric Association, 1980), which have been compared by Cantwell (1983b). In many respects, they are almost identical but differ in duration and exclusion criteria, with the DSM-III criteria probably less strict than the other two sets of criteria. In 1987, the DSM-III criteria were revised, and there were minor changes made in the operational diagnostic criteria for the diagnosis of a depressive disorder in the revised edition. The DSM-III criteria explicitly state that the essential features of a major depressive disorder are similar in infants, children, adolescents, and adults. However, the DSM-III indicated that there were probably age-specific associated features. In prepubertal children, for example, the suggestion was that separation anxiety may be common, whereas in adolescence, negativistic and antisocial behavior may be present. At the time that text was written, there was really no systematic studies to show that in fact the associated features differed with age. But Geller, Cooper, Chestnut, Anker, & Schluchter presented some preliminary data to suggest that indeed that may be true.

Cantwell (1983b) reviewed previous thinking about the clinical picture of depression in young people. The earliest view suggested that depression as a clinical syndrome with a clinical picture identical to that which occurs in adults simply did not occur in prepubertal children.

A second view was that there were unique symptoms that occurred in children who were depressed that were part of the essential clinical picture that occurred in addition to the core clinical features which occurred in adults. This is a reasonable hypothesis since children are developing organisms, and it would be unrealistic to expect that the clinical picture of any disorder, depression included, essentially manifests itself in exactly the same way in preschoolers, grade schoolers, and adolescents. However, there has been a surprising lack of systematic research in this area.

Data produced by Kovacs (1987) do suggest that children who meet DSM-III criteria for depressive disorder also have high rates of social withdrawal. After dysphoric mood, it is the symptom that most distinguishes depressed from non-depressed psychiatrically ill children. Social withdrawal is not part of the essential criteria for a depressive disorder in any of the major criteria, including the DSM-III, for the diagnosis of depression in adults. The data of Kovacs suggest that it probably should be for children.

A third view was that depression manifests itself in a totally different way in young people. That is, the depression did not present with depressive affect and the other coexisting vegetative psychomotor and cognitive symptoms that occur in adults. Rather, the depression was "masked" by a multitude of symptoms, including hyperactivity, learning problems, antisocial behavior, enuresis, and others. Data from Puig-Antich (1982) and from Carlson and Cantwell (1979, 1980a, b, 1982) do suggest that associated symptoms are quite common in prepubertal children with major depressive disorder and in adolescents with major depressive disorder. However, these associated features are often part of another psychiatric disorder that coexists with the depression, such as attention deficit disorder, conduct disorder, or one of the anxiety disorders. Moreover, these symptoms do not mask the depressive picture but rather co-occur with it.

Carlson and Garber (1986) suggested that symptomatic isomorphism across the age ranges in depression probably will not occur. However, they suggested that it might be possible to identify certain signs and symptoms that are age appropriate and that take into account the individual child's level of functioning within the domains that are affected by depression—affective, behavioral, cognitive, and vegetative. Thus, certain symptoms in one age group may be deemphasized in favor of other symptoms that would be considered to be age or phase specific. Certain symptoms, such as guilt, hopelessness, and others, may require levels of cognitive functioning that are not developed yet in younger children and thus would not be expressed in that age range. There may be certain symptoms that are not regularly associated with an adult depressive syndrome but which do occur in preschoolers, grade schoolers, or adolescents. Their solution to this problem was to create a multitiered

diagnostic system. The first tier would be the core clinical signs and symptoms of depression that seem to be consistent across all age ranges. A second tier would be symptoms that occur rarely in children but would be considered signs of a depression if they did exist. A third tier would be symptoms that are found to be highly associated with different age ranges. The actual number of symptoms required for a diagnosis of depression may change with age, and the actual symptoms and signs themselves that are required for the diagnosis may change for different age ranges. Furthermore, Carlson and Garber proposed that affective, cognitive, vegetative, and behavioral domains of depression may be affected across the age ranges but that the symptoms which defined the different domains may differ with age. The correct answer to this problem awaits much more systematic research.

It should be noted that there are investigators who have attempted to develop sets of criteria specifically for the diagnosis of depression in children, and these have been discussed by Cantwell (1983b). An example is the set of criteria developed by Weinberg, *et al.*, 1973, which were modeled after the Feighner criteria with appropriate changes being made for the diagnosis in children. At least one systematic study comparing the Weinberg and the DSM-III criteria suggested that the Weinberg criteria were much broader (Carlson and Cantwell, 1982). More children were diagnosed as being depressed by the Weinberg criteria than were diagnosed by the DSM-III criteria. Almost all children who met the DSM-III criteria met the Weinberg criteria, but the reverse was not true. This fact has important implications for clinical practice and for research. For example, a child may be treated for "depression" according to the Weinberg criteria when he or she may indeed be manifesting another disorder by the DSM-III criteria that would require a different type of therapeutic plan. Obviously, for research studies looking at possible etiologic factors, outcome, response to treatment, and other issues, if different children are entered into different studies by different sets of criteria, the results will be a reflection of the fact that different children make up the patient populations rather than differences in results between two groups of children both of whom were diagnosed as being depressed by the same set of criteria.

Developmental issues in the assessment of the depressive syndrome in children of different ages are obviously related to the clinical picture and the diagnosis of depressive disorder; such assessment issues will be assessed below.

Related to the core clinical picture is the issue of various subtypes of depressive disorder. There have been many meaningful attempts to subclassify affective disorders in adults. Some of these are official classification systems, such as the DSM-I, the DSM-II, the DSM-III, and the various International Classifications of Diseases (ICD). Others have been unofficial systems that were developed primarily for research use. Such dichotomies include the following: primary versus secondary, unipolar versus bipolar, endogenous versus nonendogenous, and melancholic versus nonmelancholic (using DSM-III terms); also included are psychotic versus nonpsychotic versus neurotic, and familial classification schemes, such as that of Winokur (1979) with pure depressive disease with family history of depression only, depressive spectrum disease with family history of other disorders, such as alcoholism and antisocial personality disorder, and sporadic depressive disease with no family history of depression or other disorders.

Klerman *et al.* (1987) suggested that any diagnostic system with various subtypes can be validated by internal and by external criteria. Internal validity is demonstrated by determining consistency of symptoms that comprise the essential diagnostic criteria for various subtypes. External validity is established by validating various subtypes with external criteria that include epidemiological, family aggregation studies, biological laboratory studies, correlation with psychosocial factors, natural history studies, and response to treatment studies. It suffices to say that none of the classification schemes yet devised for the classification of affective disorders in adults has been fully validated with these external criteria. Nevertheless, subtyping has proved valid for both clinical and research reasons.

Which of these adult-oriented subtyping schemes seems to be useful for children and adolescents? The unipolar-bipolar dichotomy is quite useful with adolescents. Full-fledged mania generally begins for the first time in the adolescent age range, although it can occur in prepubertal children. There is an issue whether mania may exist in prepubertal children or whether it manifests itself somewhat differently than it does in the classical adult picture. The melancholic-nonmelancholic dichotomy also has proved to be useful for children and for adolescents, at least with regard to such things as the likelihood of having a positive dexamethasone suppression test. There may be differences in response to somatic therapy as well.

The primary-secondary dichotomy also seems useful. Carlson and Cantwell (1980b) showed that in clinic samples who were primarily outpatients, at least one half of the children and adolescents who present with a DSM-III diagnosable depressive disorder will also have a secondary type of affective disorder. That is, they will present with a depressive episode in the face of a preexisting psychiatric disorder, such as conduct disorder, antisocial personality disorder, or separation anxiety disorder. This finding has implications for both treatment and for research.

Psychotic depression does occur in adolescents with a picture not unlike that which occurs in adults. Psychotic depression also occurs in children in the prepubertal age range, but they are less likely to have delusions than hallucinations. Psychotic depression in children and in adolescents is less likely to be responsive to tricyclic antidepressant treatment, and this also seems to be a useful dichotomy.

Thus, there are some adult-oriented classificatory subtypes that have found utility in children and adolescents. Essentially, these have all been in grade-school- and adolescent-aged children, not in infancy or in preschool children. Attempts have been made to develop specific classification systems for childhood depressive disorders, and the results have been reviewed by Cantwell and Carlson (1983b) and Carlson and Garber (1986). One approach has been based on developmental theory and states that because children have different capabilities at different ages, the phenomenology of depression with different ages must be present in an age-appropriate context (Bemporad & Wilson, 1978; Glaser, 1968; Malmquist, 1971; Phillips, 1978). Although Phillips (1978) included Spitz's (1946) notion of anaclitic depression as an example of infant depression, Bemporad and Wilson (1978) do not agree. Along with Sandler and Joffe (1965), they come to the conclusion that states that look like depressive disorder in infancy are more likely basic psychobiological reactions to deprivation.

There are few systematic studies of depressive disorders in the preschool age range. The general feeling from this literature is that depressions under the age of 7 are more diffuse and not as well defined as they are once one leaves the preoperational period of cognitive development.

Malmquist (1971) proposed a classification of depression in children that used as organizing factors both putative etiology and developmental level. Two of the five major subgroups were based on known etiology, those associated with known organic diseases, and those that were described as reality-based reactions to impoverishing and depriving environments. The other three major subgroups were based on developmental level and were described as syndromes associated with difficulties in individuation, latency age types, and adolescent types.

McConville, Boag, and Purohit (1973b) described an affectual subtype, a self-esteem subtype, and a guilt subtype of depression in children. At one time, these were considered to be independent diagnoses, but later, McConville and Boag (1973a) postulated that these three subtypes were different stages of expression of the same mourning process in children.

Frommer (1968) postulated three groups: enuretic depressives, pure depressives, and phobic depressives; however, she gave no operational criteria for the diagnosis of the subtypes or indeed for the diagnosis of depression in general.

Cytryn, McKnew, and Bunney (1980) proposed a classification scheme based on severity of the depressive disorder and putative etiologic factors. One was a typical depressive syndrome characterized by symptoms that would be considered to be depressive in adults, and these could be acute and chronic. The most common type was a third subtype that they described as masked depression. Later, in 1980, they rediagnosed these children, using the DSM-III criteria, and found that the majority of those with masked depression actually met another DSM-III subtype.

Currently, the issue of subtypes of depressive disorders in preschoolers, grade schoolers, and adolescents is a matter for future research. Although the adult-type classificatory schemes seem to work reasonably well for adolescents, they are not fully validated even for adults. Probably, they work even less well further down the developmental and age level. But search for subtypes will continue to be important because subtyping may lead to improved precision in the clinical picture of depressive disorder in the various age ranges.

Assessment

Recently, the assessment of depression in children and adolescents has been a subject of intense research. The major issues of assessment of depression across the life span have been reviewed by Cantwell and Baker (1987), Petti (1985), Kazdin and Petti (1982), and Strober and Werry (1986).

Assessment involves a diagnostic process of answering a specific set of questions about a child patient, using a particular diagnostic classification scheme, with the disorders in the scheme diagnosed by specific diagnostic criteria. Furthermore, the assessment requires the use of instruments that collect data that allow the researcher to make a specific diagnosis from a specific classification scheme using specific criteria. Finally, the assessment involves the assimilation of the data collected to make a specific diagnostic statement.

Generally, the tools that have been used in psychiatric diagnosis in childhood for all types of psychopathology and for depression, in particular, include parental interviews, child interviews, behavior rating scales, the physical exam, the neurological exam, and laboratory studies. In general, parent interviews, child interviews, and rating scales are the primary instruments used for the assessment of children referred for affective disturbance. Recently, Cantwell and Baker (1987) and Petti (1985) have reviewed the instruments that are currently available for the assessment process in children referred for depression and other forms of affective disturbance. An examination of this literature indicates that the age range has been grade-school-aged children and adolescent populations so that certain instruments will be useful for certain ages and not for others. Few instruments have been developed specifically for the assessment of depression in infants and in preschool children. Some instruments are useful in the screening of normal populations, some in the screening of clinical populations, some are useful in making a specific diagnosis of depression, and some instruments are useful in monitoring therapeutic intervention. A complete discussion of the various instruments is beyond the scope of this chapter, and interested readers are referred to Cantwell and Baker (1989) and Petti (1985) for a detailed discussion of the various instruments. What will be emphasized here is that developmental issues must be taken account of in the assessment process itself.

There are issues of developmental aspects of symptom pattern that already have been reviewed, but the developmental issues in the assessment process itself are somewhat different. If one conceptualizes the assessment process as consisting of a clinician collecting data using certain instruments, such as parent interviews, child interviews, parent and teacher rating scales, and self-rating scales, then it can be seen that developmental issues are very important.

As discussed above, the clinical phenomenology of symptoms may differ across the age range. Thus, rating scales that are useful for early grade-school-aged children may be less useful for adolescents and may not be useful for preschool children.

In interviewing parents to obtain descriptions of behavior in response to questions about specific symptoms of depression, using a structured interview such as the Diagnostic Interview for Children and Adolescents (DICA) or the Diagnostic Interview Schedule for Children (DISC), the parents' responses may not be as useful at wide ends of the age spectrum as those with very young children or with late adolescents. However, it is with the interview with the child that developmental issues in the assessment process are clearly most important, and some of these issues have been discussed by Puig-Antich (1983) and by Kovacs (1986). Kovacs emphasized the general demands the clinical interview places on the child and developmental considerations regarding these demands. The child must recognize that the doctor or the clinician is a helping agent. Even the issue of self-attribution of the child as having a problem is germane. Thus, the child must have trust in the helping agent and must accept verbal exchange as a means of resolution of whatever problem that he or she may have.

There are also specific competencies that are required by the child in the assessment of depressive disorders. These competencies include, but are not limited to, knowledge of emotion, knowledge of mood, understanding of the self, and concepts of time and memory, all of which have developmental implications.

In a detailed discussion of the individual symptoms of the depressive syndrome, Puig-Antich (1983) discussed differences between children and adults in assessing the individual symptoms that make up the core symptoms of the depressive syndrome. For example, in the assessment of depressed mood, eight different labels of depressed mood must be used with a child in order to conclude conclusively that the child does or does not have what would be considered to be the core symptom of dysphoric mood. Children may use different labels to express the same concept, and time estimates are also more likely to be inexact the younger the child. Pragmatic solutions would involve not asking about days or weeks of duration from children but rather if they are depressed from the time they get up in the morning until they eat lunch; or do they feel sad, blue, or down in the dumps from the time they eat lunch until they leave school.

In considering a symptom, such as difficulty in

concentrating or thoughts being slowed down, it is well to take into account that prepubertal children may not understand at all well the concept of thinking being slowed down. Concentration difficulties with all types of psychiatric disorders are hard to assess in prepubertal children without direct input from the parent and from the teacher. A core symptom of the depressive syndrome is excessive guilt. However, in order to assess that symptom in a child, it is axiomatic that the child must understand the concept of guilt.

In summary, thee are important developmental issues in the assessment process that must be taken into account when assessing the depressive syndrome in children across the age span. The de velopmental issues are not simply one of different expressions of depressive symptoms at different ages. There are also different cognitive competencies that the child has at different ages, and these must be taken into account when interviewing the child. Moreover, it must be recognized that the parents may be a better source of certain information, such as overt behavioral symptoms of aggression, hyperactivity, or poor school performance, whereas the child may be a better source of information about "inner" symptoms, such as a sad mood, suicidal ideation, hallucinations, and delusions.

Rating scales to be completed by parents, teachers, the child, and such significant others as peers have been developed for the assessment of depression in children. In general, however, these instruments have been developed with specific populations with specific age ranges, and they may not be generalizable across a wide spectrum from preschool through adolescence. Depending on the purpose of the assessment and the questions to be asked, one set of instruments might be preferable over another set.

Etiologic Theories

Although multiple etiologic theories have been postulated for depressive disorders, most of these have been described for adults. These have been reviewed by Cantwell (1983c), Kashani and Cantwell (1983), and Rutter and Garmezy (1983). Akiskal and McKinney (1975) attempted to integrate 10 conceptual models of depression into a comprehensive framework for the understanding of depression in adults. These 10 conceptual models represented 5 major schools of thought: psychoanalytic, behavioral, sociological, existential, and biological. Four major psychoanalytic theories of depression exist. The earliest is the Abraham-Freud model (Abraham, 1960; Freud, 1950) in which there is a conversion of aggressive instinct into depressive effect, with the aggression being turned inward. Bibring (1965) postulated another psychoanalytic model based on a loss of self-esteem. The patient is described as experiencing helplessness in obtaining goals of the ego ideal. Object loss and factors that prevent the attainment of the ego ideal bring about hostility in Bibring's model, which is secondary and inconstant compared to the hostility and aggression in the first model.

Spitz (1946) and Robertson and Bowlby (1952) proposed a third psychoanalytic model best described as an "object loss model." Here, the mechanisms involved in the genesis of depression are separation and disruption of an attachment bond. But actually, two separate issues are involved: whether separation in adult life is an immediate precipitating stress leading to depression and whether early object loss in childhood, such as that of bereavement, can predispose to the development of depression in adult life. Separation and loss events do seem to act as precipitating events for the development of depression in adult life but are not necessarily specific for depressive illness. Also, there is no one-to-one relationship between loss and depression. Thus, the separation alone is generally not a sufficient cause nor a necessary cause because many depressed individuals do not have evidence of separation. Inconclusive evidence existed for Akiskal and McKinney (1975) to decide whether or not object loss in childhood predisposed to the development of depression in adult life.

Beck (1967) developed a different type of psychoanalytic model by describing a negative cognitive triad that consisted of negative views of the self, negative interpretation of one's own experience, and negative view of the future. Negative cognitions lead to hopelessness, helplessness, and depression.

Akiskal and McKinney (1975) described two major behavioral models: the learned helplessness model of Seligman (1974), and the loss of reinforcement model of Lazarus and Lewinsohn. Learned helplessness is conceptualized as a behavioral state developing because the individual does not recognize the relationship between his or her response and any relief from adversive events. Bart's (1974) sociological model held that loss of role status played an important etiologic role in the genesis of depression, and Becker's (1964) existential school suggested that loss of meaning of existence is an important etiologic factor. It can be seen

that these five major schools overlap to some degree with each other and are not necessarily mutually exclusive and discreet schools. The fifth major school of thought is a biological one emphasizing genetic factors, biogenic amines, and neurophysiological theories.

Depression can be conceptualized as a final common pathway of various processes involving at least four major areas: genetic factors, physiological stressors, psychosocial stressors, and developmental factors predisposing to the development of a depression. Thus, certain genetic factors may predispose some individuals to develop depression. Early object loss or learned helplessness in childhood may act as a developmental precursor and predisposer to depression. Psychosocial stressors, such as object loss, or physiological stressors, such as viral illness, may produce biochemical changes leading to the final common pathway of depression. A complete discussion of the relevance of all of these various schools of thought and models is beyond the scope of this chapter. As noted above, most (if not all) of these models have been developed from the study of depression in adults. Nevertheless, there are some data with grade school children and adolescents with depression to suggest that certain of these models may have relevance. Biological studies of depression in the prepubertal age range have been reviewed by Puig-Antich (1983, 1986, and Chapter 25 in this volume). There are similarities and there are some differences in the biological correlates of prepubertal and adult depression. Some of the differences may be due to developmental factors.

Rutter (1986) pointed out that psychoanalytic theories have emphasized the early mother–child relationships and loss of a loved object, and although many of the details of these particular psychoanalytic models are not necessarily validated, the role of loss as an etiologic factor has received some empirical support that has led to the development of certain alternative theories. Several theories, especially those of Beck (1967) and Seligman (1974), do suggest that negative cognitive sets develop in childhood and that these lead to feelings of hopelessness, helplessness, and depression.

Other theorists, such as Bowlby (1980) and Brown (Brown & Harris, 1978) suggested which conditions may lead to these negative cognitive sets in childhood. Bowlby postulated that a negative cognitive set can arise from insecure mother–infant attachments in early life, actual loss of a parent during childhood, and being brought up in an atmosphere without love. These negative cognitive sets will then cause that child to interpret later losses as further failure either to establish or maintain an affectionate relationship with another individual that is enduring. Thus, in the Bowlby model, early loss leads to a predisposition to depression; later on, losses act as immediate precipitants for the occurrence of depression. Brown's views may be thought of as similar, but loss may not necessarily be used in exactly the same way that Bowlby does, but rather as a loss of sources of value or reward.

The Lewinsohn model (Lazarus, 1968; Lewinsohn, 1974) emphasizes the lack of positive reinforcement from pleasurable activities, especially the lack of positive reinforcement that is contingent on responses of the individual. This is thought of as a predisposing factor to depression.

Thus, many of the theories postulate certain factors that may lead to a vulnerability to depression, which, in turn, may lead to negative cognitive sets or a predisposition to react to loss events. Later losses may then act as precipitating stresses for the development of a particular episode of depression. How early in development children will be able to develop the necessary cognitive set postulated by Beck (1967) and others is open to question. Certain cognitive formulations and certain cognitive functions are necessary developmentally for the child to develop a negative cognitive set as described by Beck. Immature cognitions or changes in the way children respond to failure as a result of age may explain why depressive disorders, at least as diagnosed by adult criteria, are much rarer in preschool and grade school than they are in the adolescent age range.

Rutter (1986) suggested that there are data implying that younger children do not respond to failure situations with helplessness and do not view failure as something that will remain and cause permanent limitations on later functioning. It is unknown whether loss events or stress events, which may specifically predispose to depression in the acute sense, are more frequent in adolescents than they are in preschool or grade school or are more common in adolescent females than they are in males, which would have to be postulated if life stress itself were to be a major etiologic factor. On the other hand, with age, there may be changes in the way children and adolescents perceive stress, and there may be differences in the way different sexes perceive stress with age as well.

Akiskal and McKinney's (1975) summary of the 10 conceptual models into 5 schools of thought with different frames of reference is a masterful synthesis of the available data in adults, taking into

account family genetic factors, neuroendocrine and neurophysiologic factors, psychosocial stress factors, cognitive set factors, and dynamic factors. Less data are available on the relevance of these factors to depression in infancy and preschool, but somewhat more data are seen with prepubertal grade-school-aged children and with adolescents. Although the available data suggest some similarities and dissimilarities between adult depression and depression in young people, further research is needed to establish with model or models is more relevant for what types of depression at what age range and for which sex.

Links between Depression in Infancy, Preschool, Grade School, and Adolescence

One of the major research questions regarding depression in young people, whether they be infants, preschool children, grade school children, or adolescents, is whether or not there is a link between depressive phenomenon in the young age range and depressive disorders in later life. Three types of study designs can be used to attempt to relate depressive phenomena in childhood and depressive phenomena in later life, and these have been reviewed by Cantwell (1983a).

The first study design is a true prospective study, or as it is sometimes called, the real time prospective study. The true prospective study allows the investigator to select infants, preschoolers, grade schoolers, or adolescents with a defined clinical picture and then to follow them up over a period of time and to collect data at various points in time. Thus, the investigator could select "depressed" infants and follow them up through later age ranges, or select depressed adolescents and follow them up into adult life. The advantages of this design are that it allows a particular investigator to select the sample the way he or she wants to select them and study them the way he or she wants to study them. This is the only study design that can truly demonstrate whether or not any type of therapeutic intervention might be effective with depressed children, because it is the only one that allows the investigator to assign children at random either to treatment or no-treatment conditions or to one type of treatment versus another type of treatment. However, there are some major disadvantages to this study design. If the investigator begins with very young children, such as infants or preschoolers, a long time will elapse before they reach adolescence or adult life, and attrition may be a problem over many years. Also new knowledge is being acquired while the children are growing older. Thus, the advantage of having been able to select a particular sample and study them a particular way may be eliminated if the methods that were used initially become somewhat obsolete by the time of follow up. It is clear that our methods of semistructured and structured interviews of parents and children and the development of rating scales over the past 10 years in the study of depression in young people have made early studies rather obsolete, because criteria were not specified, and the instruments used to assess the depression would no longer be considered state-of-the-art at this time. This study design is probably best used to look at short-term stability of psychiatric diagnosis over a relatively brief period of time and for studies that look at the effect of a particular type of therapeutic intervention versus no treatment or some other type of treatment. Studies over multiple age ranges, such as infancy, preschool, grade school, and adolescence, raise the issue of which instruments should be used to measure what domains. Certain instruments will not be appropriate for one age group but will be appropriate for another, and unless they are demonstrated to be comparable measures tapping the same domain, it cannot be assumed that what one instrument measures at Point A, say, in the preschool age range, will be the same thing that another instrument measures at Point B, say, in adolescence.

The second major design that looks at links between depression in young people and depression in later life is the follow-back study. Here, the investigator selects a sample, either in adult life or in adolescence, and then makes use of existing records that were available in infancy, preschool, or grade school to collect early data. Modification of this design would be to use retrospective data collection to paint a picture of what the individual was like in infancy, preschool, and so on. However, this is probably fraught with much more reliability problems than relying on existing records. Thus, if the investigator selected a population of adults with definite depressive disorders, he or she could select several adult control groups—those with no psychiatric disorder and those with such disorders as anxiety or antisocial behavior. Childhood records of all groups would have to be located with relatively complete measures and comparable measures in each group. If a consistent picture were found of a clear-cut depressive disorder in childhood or indeed

any type of childhood disorder that was uniquely associated with the adults who had depression versus the adults who had other psychiatric diagnoses, this would provide evidence of a link between depressive phenomena in childhood and depressive phenomena in later life.

The major advantage to this study design is that the investigators do not have to wait for the population to "age" as they do with the first type of study design. The major disadvantage is that the investigators have to rely on whatever existing records are available for childhood data. Because childhood depression is a relatively new field of research, it is unlikely that the investigator is going to be able to select adults or adolescents who had systematic data collected, which would permit a retrospective diagnosis of a depressive disorder. However, a few studies have attempted this. There are also issues in the selection of the population to be studied in adult life or, say, in adolescence. If the population is selected from the general population, they may not be comparable to adults or adolescents with depressive disorders who are selected from an inpatient setting, and even those from inpatient settings may differ if the investigator selects a private psychiatric hospital inpatient setting versus a university hospital inpatient setting versus a general city or community hospital inpatient with indigent populations.

The third study design that looks at links between childhood and later life is the catch-up prospective study that combines elements of both of the study designs listed above. Here, the sample is identified in childhood rather than in adult life or adolescence. However, the sample is selected in childhood from existing records that have already been collected rather than in selecting a new population of children to be studied by the investigator, which also eliminates the aging factor for the population. However, the investigator must rely on data collected at the time rather than being able to design a study and collect the type of data he or she wants in the way he or she wants it. Robins (1966) provided the best example of this type of research in her follow-up study of children selected from a court clinic in St. Louis, Missouri.

The records that are available to select a population in childhood must be relatively complete, reliable, and valid, and the population should be an unbiased one. The amount of information and the quality of information collected at the point of follow-up, be it in adolescence or in adult life, must also be comprehensive, accurate, and complete.

There are other issues involved in study design that attempt to look at links between depression in childhood and depression in later life. Some of these factors include: selection of the population of depressed young people; selecting the populations that will be control or comparison cases; what type of data to be collected initially in a prospective study; how long the follow-up interval should be; how to measure intervening variables between Point 1 in childhood and Point 2 in adolescence or in later life; what types of variables should be measured that occur between Time 1 and Time 2 at follow-up; what type of outcome measures should be looked for; and what type of statistical analysis is appropriate. Robins (1979) reviewed this study design issue in detail, and Cantwell (1983a) reviewed it with regard to the study of depression in young people.

Some of the important points of study design will be mentioned briefly. In a real time prospective study, the index sample should be selected by operational diagnostic criteria and by the use of measures that are reliable and valid to allow replication by other investigators. Petti (1985) and Cantwell and Baker (1987) reviewed which instruments are useful for this purpose in the study of depression in young people. Careful consideration to study sample should include the purpose of the study. Thus, if the investigator wants to study the predictive validity of the DSM-III diagnosis of major depressive disorder, the population should be as homogeneous as possible and should probably exclude children who have other types of psychiatric disorders so that the sample will be as pure a sample as possible of a major depressive disorder diagnosed according to one set of criteria. On the other hand, if the investigator wishes to look at the predictive validity of different types of diagnostic criteria for making a diagnosis of depression, then the sample will probably be more heterogeneous to allow comparisons between different sets of criteria (e.g., RDC [research diagnostic criteria]), DSM-III, and Weinberg categorical criteria] and possibly dimensional criteria, such as a cutoff score on a particular rating scale.

The source of the index sample is also important if the investigator is to make any generalizations about the data. Thus, selecting only inpatients may restrict follow-up information to only inpatients from that type of a particular setting. Selecting outpatients may provide a broader sample, but the nature of the outpatient population (i.e., taken from the general population vs. taken from a psychiatric clinic population) may affect outcome data so that the generalization about links between child-

hood and later life may apply only to that type of sample.

What type of comparison cases should be selected and when they should be selected are also important issues. It is important not only to determine that depressive phenomenon in young children are associated or are not associated with particular types of outcome compared to normals, but also whether there is anything specific about the outcome compared to children who have psychiatric disorders of a nondepressive type in childhood. Robins (1979) made cogent suggestions about the nature of control and comparison groups that are important to anyone looking at the literature regarding links between childhood and later life depressive phenomenon. Systematic collection of data in the real time prospective study and at follow-up time in all study designs is imperative.

Length of follow-up will be determined by the nature of the question being asked. If the investigator wishes to look at the relationship between depression at a particular time in childhood and in adult life, it is axiomatic that follow-up must be done with an adult population. On the other hand, looking at the natural history over a shorter period of time of children who are diagnosed as depressed by various criteria at various ages will require a shorter period of follow-up and still produce reliable and valid data, although it may reveal nothing about the later life outcome of these children.

What intervening variables to measure during any follow-up study and how to measure them can be problematic. Measuring such things as stress, life events, and the effects of various types of intervention are still somewhat in their infancy. Nevertheless, we must develop reliable and valid measures of intervening variables if we are to make sense of any relationship between depressive phenomenon in early life and depressive phenomenon in later life.

There has been a relatively large amount of work on the natural history of adults with affective disorders. We know that the course of affective disorder in adults is variable and that there are certain subtypes that have different types of outcome. Some individuals have repeated episodes of depression, but the episodes are spaced by many years of no illness and with functioning at a normal level. Other individuals seem to have episodes of depression that cluster much more closely together, and a third group seem to increase in the number of episodes as they get older so that the interillness period gets shorter between each episode. Although it is felt generally that the interillness period is one of normality, it is true that in many adults with affective disorder there is no complete return to normality between clear-cut episodes of illness, and, in some cases, there is a progression, after an episode of major depressive disorder, to a more chronic subsyndromal state of depression rather than to a true normal state.

Approximately half of all adults with an episode of major depression will have another episode, and those with recurring episodes of major depression have an increased risk of developing a manic episode. But certain subtypes do have predictive validity with regard to outcome. Bipolars, for example, differ from unipolars not only because they are likely to develop manic or hypomanic episodes at follow-up, but also, because they have more episodes in their lifetime than those with pure unipolar depression. Those with unipolar depression tend to have somewhat longer episodes than those with bipolar depression—6 to 9 months versus 3 to 6 months. Winokur's (1979) familial classification of unipolar depressed patients also seems to produce meaningful subgroups with regard to outcome. The pure depressive disease group is more likely to have long periods of chronicity than those with depressive spectrum disease, who are much more likely to have a variable illness period with more interpersonal difficulty. Those with secondary versus primary affective disorder also differ in that the primary group regards their depressive episodes as discrete and quite different from normal, whereas those with secondary depression do not have as discrete episodes but rather more transient or more chronic periods, and many might qualify for a diagnosis of DSM-III dysthymic disorder.

Although not much research has been done with regard to the links between depression in adolescence and depression in later life, there are some data that are suggestive. All of the various study designs listed above have been used in one form or another. True prospective study designs have been used by Poznanski, Krahenbuhl, and Zrull (1976) and Herjanic (1976). Poznanski's data suggested that children diagnosed as depressed were likely to manifest depressive symptomatology in adolescence and that the clinical picture in adolescence resembled more the adult type clinical picture than it did in childhood.

Herjanic (1976) found that those subjects who met operational criteria for depression as described by Weinberg *et al.* (1973) in a hospital setting were more likely to have a diagnosis of depression on

follow-up than those who were clinically diagnosed as depressed but did not meet operational research criteria.

Other studies using a prospective design were reviewed by Cantwell (1983a). Probably the most significant study of this type is that by Kovacs (1984a,b) of prepubertal children who were diagnosed by operational criteria as being depressed in the outpatient clinic at the Western Psychiatric Institute and Clinic in Pittsburgh. The prepubertal children were given a diagnosis of either major depressive disorder, dysthymic disorder, adjustment disorder with depressed mood, or had a psychiatric disorder that was nondepressive in nature. The later group was a heterogeneous mix of various types of psychiatric disorders. The data indicated that adjustment disorder with depressed mood remitted relatively rapidly and was not associated with an increased risk of the development of a depressive episode over a 5-year period of follow-up. The nondepressive psychiatrically ill children also did not have elevated rates of depressive disorder over the period of follow-up. Major depressive disorder remitted more rapidly than did dysthymic disorder. However, both have relatively long courses of illness. Moreover, both were associated with the risk of the development of a subsequent episode of major depressive disorder during the period of follow-up.

Thus, the data suggest that major depressive disorder and dysthymic disorder diagnosed by DSM-III criteria in prepubertal children are associated with an increased risk of depressive disorders over the next 5 years. Longer term follow-up will be necessary to see if this group with an early onset has a more severe outcome in adolescence and in adult life than those with an adolescent onset or with an adult onset.

One major problem with the Kovacs data is that the great majority of children with major depressive disorder and dysthymic disorder also had other psychiatric disorders, including anxiety and disruptive behavior. Thus, they would be considered to be secondary depressives. Although it looks as though secondary depression and primary depression in adult life are associated with different outcomes, the data cannot be generalized to children with pure major depressive disorder or pure dysthymic disorder.

Strober (personal communication, 1986) produced data on systematically diagnosed adolescents from the UCLA Adolescent Inpatient Service. All subjects met DSM-III criteria for major depressive disorder, and more than 70% were primary depressives. The course of the individual episode was relatively protracted with the nonmelancholic and nonpsychotic types having shorter durations than the other two, and the nonmelancholic and nonpsychotic types also having a less of a likelihood of relapse over time. Overall, approximately 45% of the adolescents had a relapse within 3 to 4 years after discharge from the Adolescent Inpatient Service. Twenty percent of the major depressive disorder adolescents developed a bipolar course over the 3- to 4-year follow-up. They were characterized by a rapid onset of their depression, psychotic symptomatology during the depressive phase, the presence of psychomotor retardation, an increased family history of affective disorder (especially with a multigeneration family history) tending to become hypomanic with tricyclic antidepressant treatment during the course of their hospitalization.

The UCLA data also suggest that bipolar illness in adolescents is frequently preceded by clinical symptoms of a severe conduct disorder, hyperactivity, and labile mood in the prepubertal age range. This prepubertal syndrome predicts a significant increased rate of familial affective disorder (bipolar illness in particular), psychotic symptoms during the manic episode, poor response to lithium therapy, and earlier age of onset of illness in affected relatives. The Strober data suggest that, at least for adolescent inpatients, a complex interaction of clinical phenomenologic features, age of onset, and family features predict different courses of outcome.

In a UCLA sample that consisted primarily of outpatients, who were studied earlier by Carlson and Cantwell (1984), Carlson demonstrated that depressed adolescents who had an adolescent onset versus depressed adolescents who had a prepubertal onset had different outcomes. A follow-up over a period of 6 to 7 years after they were seen at UCLA suggested that nearly 90% of the adolescents who had had at least one episode in the prepubertal age range were still having significant problems with depressive symptomatology versus less than 40% of those who had an adolescent onset. The prepubertal onset group were more likely to be male, had increased rates of learning disorders and other psychiatric disorders, such as disruptive behavior, and an increase in family history of alcoholism and antisocial personality disorder as well as of depression. These data (Carlson, 1984) suggest a link between depressive disorders in the prepubertal age range and depressive disorders in adolescence. It

also suggests that the continuity may be greater for those children who have other types of psychopathology in childhood in addition to pure depression.

Zeitlen (1972, 1985) used a study design similar to the catch-up prospective design but with certain modifications. All of the patients Zeitlen studied had been seen at the Maudsley Hospital in London, England, as both children and as adults. A comparison group of children who were not seen at the Maudsley as adults and a comparison group of adults seen at the Maudsley who had no childhood disorder was also collected. A large number of cases of depressive disorders in adults did not have any type of childhood psychopathology, and if they did, many of them did not have disorders that could be diagnosed as depressed by the usual criteria. Children who were seen at the Maudsley who had depressive disorders not diagnosed by operational criteria did not turn out to have depressive disorders in adult life in the majority of cases. When operational criteria were used to diagnose depression in childhood and in adult life, 31 childhood cases out of 37 had an adult depressive disorder. The continuity between childhood depression and adult depression, when diagnosed in this way in this sample, was greater for depressive disorder than the continuity for any other neurotic state except obsessive-compulsive disorder. It should be pointed out that this continuity did not exist when the clinical diagnoses that had been made at the time the individuals were seen in childhood and adult life were used. The continuity only occurred when diagnoses were made by Zeitlen himself, using specified operational criteria in childhood and in adult life. Many of these children, when seen at the Maudsley, also had multiple symptomatology and probably would have met criteria for other disorders. These results are similar to Herjanic's (1976) findings in patients who were seen in the St. Louis Children's Hospital.

The follow-back study design of adults with a diagnosis of depression has been used by several investigators, and these data were reviewed by Cantwell (1983a). No unique clinical picture in childhood generally was described with these groups of adult patients by the investigators in their studies.

In summary, the data that are available thus far suggest links between depression in young people and depression in their later adult life. The data are probably strongest for depression in adolescents and weakest for depression in infants and preschool children. Kandel's (1986) work with adolescents suggests not only that depressive disorders have some continuity but that depressive symptomatology does as well. The researchers saw adolescents, aged 15 and 16, in the general population who were rated as depressed on a dimensional basis rather than on a categorical basis. At ages 24 and 25, this population of adolescents did have similar experiences of dysphoric mood in their adult life.

Directions for Future Research

A good deal of progress has been made in the study of depression in young people, despite the fact that this area of systematic investigation is relatively new in child psychiatry. However, much future work needs to be done, and some suggested areas of important research will now be presented for consideration.

1. *Development of criteria for the diagnosis of depressive episodes.* This might be the starting point for all future research. Whether the research is of a clinical nature or of a biological nature, who gets into the study is probably the most crucial factor determining the results. Criteria are going to have to be developed for the diagnosis of depressive disorders at various ages: infancy, preschool, grade school, and adolescence. The suggestion of Carlson and Garber (1986) that different criteria may need to be developed for different age groups needs systematic study. Their idea of a several tier approach warrants merit, as does their suggestion that there may be a set of core criteria that are necessary for the diagnosis of depression across all age spans. On the other hand, certain symptoms that are common at one age may not be present at all at another age; but other symptoms may be important that are not necessarily present in adult depression. Although not systematically investigated, the question of sex-related criteria might also be an interesting avenue for approach. We know that childhood depression diagnosed by adult criteria is essentially equally common in males and females in the prepubertal age range but that it is much more common in females in the adolescent age range. Many prepubertal males with depressive disorders also meet criteria for other disorders, especially disruptive behavior disorders. Whether these disruptive behaviors should be considered part and parcel of the depressive syndrome in males is a question that awaits future research. It is of interest that studies of successful suicides that occur primarily in the ado-

lescent age range suggest that those most at risk have a mixture of disruptive behavior and of depressive symptomatology.

In the development of criteria, some reconsideration should be given to the question of "masked depression." Are there nondepressive clinical pictures in childhood that predispose to the development of depressive disorders in adolescence and/or adult life? If so, could they be considered "formes fruste" of depressive disorders? The limited data that are available suggest that that indeed might be true for manic disorder. As mentioned above, a significant number of adolescent depressives who have a prepubertal history of severe disruptive behavior disorders go on to develop full-fledged mania in adolescence. This raises the question as to whether this disruptive behavior in the prepubertal age range is actually a childhood manifestation of what later may become clear-cut manic behavior.

2. A second set of studies are related to the development of criteria, that is, *classification and subtyping studies*. A thorough study of the various subtypes that have been found useful in adults is necessary across the age range to see if indeed these subtypes occur at all frequently in different age ranges and whether they have any external validity. However, subtyping should not simply be a manifestation of what we know about adults with affective disorder and a downward extension. Indeed, there may be meaningful subtypes in infancy, preschool, and childhood that have not yet been discovered in adolescence or adult life, because the adult classification schemes work well in adolescents and adults. When subtypes are developed on the basis of phenomenology, family history, or some combination of other factors, the subtypes need to be tested for their external validity using the criteria outlined above.

3. The third set of studies has to do with *assessment instruments*. Obviously, certain types of assessment instruments are going to be more useful with different age ranges than with others. Interviews and observations are probably more useful for the late grade school and adolescent patient as opposed to the preschooler and the infant. Interviews with parents may also have a differential usage across the age span. Various types of self-rating scales, parent-rating scales, teacher-rating scales, and rating scales completed by significant others such as peers also may have differential utility across the age span, and these findings need to be checked.

Also, comparison of individuals who were diagnosed as depressed by different instruments at different ages would be a fruitful area of investigation. Children diagnosed as depressed by parental interview but not by child interview versus children diagnosed by child interview but not by parent interview would be one such comparison. Likewise, children diagnosed as depressed on the basis of a cutoff score on a dimensional rating scale, such as a self-rating scale or a clinician-rating scale versus those made by categorical diagnoses, would be another such comparison.

These three sets of studies (development of criteria, development of various subtypes of the disorders, and assessment instruments useful at various age ranges) are really crucial for development of the field. The latter three sets of studies can only be completed when the first three sets have produced useful information.

4. The fourth set of studies would be *correlates of depression at different ages*. These might be biological correlates, such as neuroendocrine, biochemical, neurophysiological sleep studies, or psychosocial correlates, such as different types of life stress, and family correlates, including family genetic factors and family interaction factors. Instrumentation would be necessary for some of these studies that is not now readily available, and some of the readily available instrumentation may have developmental features, such as the biological correlate studies, which may render them less useful at various age ranges.

5. Once meaningful subtypes with different diagnostic criteria and possibly different correlates have been developed, *outcome studies* need to be done. Comparisons with outcome of normal controls and also nondepressed psychiatrically ill controls are still necessary. Predictors of outcome are also vitally necessary. We know very little about predictors of relapse and predictors of the likelihood of a subsequent episode in either children or adolescents. We know almost nothing about this area in infants and preschool children.

6. Finally, little has been said above about *treatment per se*. Nevertheless, from the clinical standpoint, this area is obviously the most important. Diagnosis and assessment are essential, and it is hoped that correlate studies will lead to a better understanding of etiology in different types of depression in children. But we do need to know what to do with different types of depression in different children at different ages. The area of psychopharmacologic intervention, which has only recently

been tapped in adolescents and in prepubertal children, has not been tapped at all in infants and in preschool children with various types of depressive disorders. Likewise, there is a growing literature of the effectiveness of different types of psychotherapy, such as cognitive therapy and interpersonal psychotherapy, and their interaction with psychopharmacological intervention in adults with depression. But there is almost no literature on this area in depressive disorders in children and adolescents.

Over the next 10 years, future investigations in these areas should advance the field to an even greater degree than it has advanced by the studies of the past 10 years.

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CHAPTER 24

Etiologic Perspectives on Depression in Childhood

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What makes children depressed? Although there is evidence that bipolar depression is strongly influenced by genetic factors (e.g., Meyersberg & Post, 1979), the evidence is far less clear for unipolar depression. A number of theories have been postulated to account for the development of depression (see Chapter 23 in this volume). In this chapter, we focus primarily on unipolar depression in children and attempt to identify and evaluate those factors that may contribute to its onset.

Early causes of depression are mainly of interest if depression is not simply a transient and common phenomenon and if depression in childhood is related to depression in adulthood. Traditionally, the existence and nature of a distinct clinical syndrome of childhood depression has been questioned (Anthony, 1975). Recently, however, there has been an increased awareness and understanding of the phenomenon that has led to an appreciation of its prevalence, course, and consequences. For example, Poznanski, Krahenbuhl, and Zrull (1976) conducted a longitudinal study in which they reevaluated 10 subjects (aged 12 to 23) who were not in treatment, but who had previously shown depressive symptomatology. Approximately six and one-half years after the initial evaluation, 50% of the children were still described as clinically depressed. The data were not analyzed separately for sex or age.

In a more methodologically sound study, Tesiny and Lefkowitz (1982) examined the stability of depressive symptomatology in fourth- and fifth-grade students, over a 6-month period. Depression was measured by peer (Peer Nomination Inventory) (Lefkowitz & Tesiny, 1980; Tesiny & Lefkowitz, 1983), self (Children's Depression Inventory [CDI]) (Kovacs, 1981; Kovacs & Beck, 1977) and teacher (5-point scale based on clinical definition of depression) ratings. All three measures of depression showed significant stability over time, although analyses were not reported separately for sex and age.

Taken together, these data provide preliminary support for the view that depression in childhood is a distinct entity, which persists over time. Larger scale longitudinal studies, following children of different ages across varying developmental periods, are clearly called for in order to help untangle issues relevant to continuity and discontinuity (Cytryn, McKnew, Zahn-Waxler & Gershon, 1986).

Depression and Loss

The role of loss in the development of depression has received considerable theoretical attention, particularly the role of early loss (Bowlby, 1980;

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Brown and Harris, 1978). Although several studies have demonstrated a relationship between early loss and depression in adulthood (Brown & Harris, 1978), this effect is not always obtained (see reviews by Crook & Eliot, 1980; Tennant, Bebbington, & Hurry, 1980). Brown, Harris, and Bifulco (1986) suggested that the failure to find effects is due to methodological limitations, which overlook the contributions of both early loss and a current adverse experience. When an individual experiences an ongoing provoking agent, it sets the stage for an episode of depression to occur. Such an agent is typically a severe event that involves loss, including not only the loss of a person but also the loss of a role or even an idea. Whether or not depression is then precipitated is dependent on the presence or absence of certain "vulnerability" factors. These factors, which include foundational experience with early loss as well as a current lack of social support, increase risk only in the presence of a provoking agent.

In a pioneer study, Brown, Harris, and Copeland (1977) provided some support for this model. They assessed women living in Camberwell, an inner-city area of London, using a clinical-type interview (Present State Examination) (Wing, Nixon, Mann, & Leff, 1977). In order for a woman to be considered a "case," there had to be both depressed mood and four or more of the following ten symptoms: hopelessness, suicidal ideas or actions, weight loss, early waking, delayed sleep, neglect due to brooding, poor concentration, self-depreciation, loss of interest, and anergia. The results showed that the loss of a mother before age 11, by either death or separation (not due to war but for at least 1 year), had a clear association with depression. Of those with depression, 22% had lost a mother before the age of 11, compared with 6% of other women. Loss of the father before or after the age of 11 was unrelated to depression. However, early loss of a mother increased risk only when a provoking agent had also occurred in the year before onset. Indeed, not 1 of the 15 women who had experienced early loss without a current provoking agent developed depression. In contrast, 7 of the 15 women with early loss and a provoking agent became depressed.

Although Brown and his colleagues have retrospectively explored the consequences of early loss of a mother for adult depression, few researchers have attempted to determine what the immediate effects of parental loss are for childhood depression. In addition to bereavement, other, more common forms of loss include parental psychiatric illness (especially depression) and the occurrence of parental life events more generally (Brown *et al.*, 1986). These events can disrupt the family, interrupt the parent's psychological and/or physical availability, and deprive the child of important protective buffering. In addition to, and especially in the presence of, parental factors, the child may be directly exposed to provoking agents. This chapter explores the implications of a subset of these factors for depression in childhood. First, it reviews evidence on the influence of foundational parental factors, including parental depression, associated life events, and family interaction patterns. Second, it reviews evidence for direct, adverse experiences in the lives of depressed children, including the occurrence of life events and negative family interactions.

Parental Influences

Rate of Psychological Disturbance in Children of Depressed Parents

Evidence

The few studies that have investigated the effects of parental depression on infants have focused on the children of parents with bipolar depression (Cytryn *et al.*, 1984; Gaensbauer, Harmon, Cytryn, & McKnew, 1984; Zahn-Waxler, McKnew, Cummings, Davenport, & Radke-Yarrow, 1984). Although these data suggest that parental depression may have an important impact, studies are needed which explore these effects on the infants of adults with unipolar diagnoses.

More evidence is available regarding school-aged children. In a pioneer study, Welner, Welner, McCrary, and Leonard (1977) compared the presence of depression in children (aged 6 to 16, $n = 75$) of inpatient mothers who met criteria for primary depression, with a control group of children ($n = 152$) whose mothers had no history of mental illness. None of the husbands had a history of mental illness. Mothers and their children were interviewed separately. The interview assessed growth and development, physical illness, academic performance, discipline problems at school and home, deviant behavior, and other symptoms of psychopathology. Of 32 relevant variables analyzed, only 12% reached significance: depressed mood, death wishes, and frequent fighting were all more common in the probands than in the controls, according to both the mother and the child. Children of de-

pressed parents reported more frequent unexplained headaches and showed more loss of interest in usual activities. Depressed mothers reported that their children were more withdrawn than nondepressed mothers did.

Twenty-five percent of the depressed mothers had at least one child with five or more (out of a possible eight) depressive symptoms. None of the children of the controls had five or more depressive symptoms. Seven percent of the probands met diagnostic criteria for definite or probable depression, according to the Feighner criteria (Feighner, Robins, Guze, Woodruff, & Winokur, 1972). None of the controls met this criteria. One major problem with this study is that the interviewers were not blind to the depressive status of the parent, nor were analyses reported separately for age and sex of the child.

McKnew, Cytryn, Efron, Gershon, and Bunney (1979) assessed 30 children between 5 and 15 years of age, whose parents were hospitalized for either a unipolar ($n = 5$) or bipolar ($n = 13$) primary affective disorder. The children were interviewed and observed twice, at 4-month intervals. The diagnosis of a depressive disorder was made if the child met the Weinberg criteria (Weinberg, Rutman, Sullivan, Penick, & Dietz, 1973). A high rate of depressive diagnoses was found in the children of adults suffering from an affective disorder: 30% were depressed at both interviews and 47% were depressed at one interview. Although there was no significant relationship between depression rating and the age or sex of the child, boys'—but not girls'—scores were significantly correlated at the two interviews. Analyses were not conducted separately for sex or diagnosis of the parent.

One study compared children, ages 6 to 18, whose parents were either severe, hospitalized major depressives ($n = 44$), or mild major depressives never hospitalized ($n = 89$) with normal, never psychiatrically ill ($n = 82$) (Weissman, Prusoff, Gammon, Merikangas, Leckman, & Kidd, 1984a). A best-estimate diagnosis was made by a psychiatrist, based on information from parents' reports and medical records, as defined in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) (American Psychiatric Association, 1980). Children of depressives were found to be at an increased risk for psychological symptoms, treatment for emotional problems, school problems, suicidal behavior, and DSM-III diagnoses. Indeed, children of depressives were three times as likely to carry any DSM-III diagnosis. The most common diagnoses included major depression (13.1%), attention deficit disorder (10.3%), and separation anxiety (10.3%). In addition, suicidal behavior was reported in the children of depressives, but not in the children of controls. Nine percent of the children of depressives actually had made a suicide attempt, and 6.5% reported suicidal ideation.

Age-corrected rates of depression by sex were calculated, using an estimation of lifetime risk (Kaplan & Meier, 1958). The earliest age of onset of depression was age 6 and there were no significant sex differences in patterns of age of onset. Several proband characteristics were found to increase risk, including early age of onset of depression, increased number of first-degree relatives depressed or with any psychiatric illness, and parents' marital status. The proband characteristics that did not increase risk were: age, sex, social class, number of children in the family, childhood history of stuttering, sleep walking or enuresis, and separation from parent as a child. A linear relationship was found between number of parents ill and children's psychiatric status: children with two parents ill evidenced the greatest depression and children with no parents ill evidenced the least depression. Although DSM-III diagnoses were made by a blind psychiatrist based on multiple information sources (parents' reports and medical records), they did not include direct interviews with the children, teachers' reports, or assessments of the children's social functioning and social supports. Analyses were not conducted separately for parental diagnosis.

In a methodologically sophisticated study, Billings and Moos (1983) compared children of parents ($n = 33$) with a diagnosis of major or minor depression (according to Research Diagnostic Criteria) (Spitzer, Endicott, & Robins, 1978) to children of matched, nondepressed parents ($n = 135$). Patients were recruited at the treatment facility (inpatients, $n = 29$) or at home (outpatients, $n = 104$). The child's health and functioning for the last month were reported by a parent on the following indices: (1) physical health problems (the number of 9 possible physical problems, such as allergies, asthma, frequent colds, and the like); (2) psychological distress (the number of 5 possible symptoms, such as sad or blue, anxious, nightmares, nail-biting, and emotional problems); (3) total health problems (a summary index composed of the 14 possible physical and psychological problems); (4) behavioral problems (the number of 3 possible problem areas including academic and discipline problems at school and difficulty getting along with other children); and (5) health-risk factors (the

number of 3 possible factors, such as smoking, alcohol use, and drug use).

Results showed that the children of depressed parents had significantly more physical and psychological problems, including greater depression and anxiety, as reported by a parent. Parents also reported significantly more behavioral problems, such as problems in academic adjustment and peer interaction as well as a tendency for greater use of alcohol, drugs, and cigarettes. Specifically, 49.2% of the probands exhibited depressed symptoms as opposed to 32.3% of the controls; 44% as opposed to 19.4% had anxiety; 34.7% had academic problems as opposed to 17%; 23% had school-discipline problems as opposed to 14.1%; and 18.1% had peer-interaction problems as opposed to 7.4%. In order to evaluate whether the chronicity of a parent's depression had an effect, chronically depressed parents ($n = 47$) who had received treatment in the previous year were compared with a group of nonchronically depressed parents ($n = 86$) who had not received treatment during that time. Children of nonchronic parents had a greater number of psychological, physical, and total health problems than children of chronic parents. Children's functioning was compared among families in three age groups: preschool (< 6 years old); grade school (6 to 13 years old) and high school (14 to 18 years old). Comparisons of children's functioning were also made among families with all girls and among families with all boys. No significant age- or gender-related differences were found, nor were there effects as a function of the gender of the depressed parent.

In a 1-year follow-up, children of originally depressed parents whose symptoms had remitted were compared with children of depressed parents whose symptoms had not remitted, and a matched control group whose parents had shown no signs of psychiatric impairment (Billings & Moos, 1985). Overall, control children fared better on indices of psychological, physical, and behavioral functioning than both the remitted and nonremitted groups, as assessed by parent reports. However, children of remitted parents functioned at a higher level than did children of nonremitted parents. Nonremitted parents were two times as likely to report significant child disturbance as remitted parents (52.2% vs. 26.5%). When the reports of depressed parents were compared with their nondepressed spouses, moderate to strong correlations were found among them on all indices of their child's health and functioning. Further, agreement was as high among depressed parents as among control parents.

Weissman, Leckman, Merikangas, Gammon, and Prusoff (1984) examined 6- to 17-year-old male children whose parents fell into the following diagnostic groups: (1) depression and no anxiety, (2) depression and agoraphobia, (3) depression and panic disorder, and (4) depression and generalized anxiety disorder. Depressed probands were the children of primary unipolar major depressives, drawn from a community sample, and were matched with children of a normal, never psychiatrically ill parental control group. Children of parents with depression and panic disorder or depression and agoraphobia were at a higher risk for disorders in general. The results showed that the children's diagnosis followed the parents' diagnosis: (1) when the parent did not have an accompanying anxiety diagnosis, neither did the child; (2) there was increased anxiety disorder in the children of parents with depression plus panic disorder; (3) increased rates of phobia were observed in the children of parents with depression plus agoraphobia and among the children of parents with depression plus panic disorder; (4) there was increased co-occurrence of depression plus any anxiety disorder in children whose parents also had a co-occurrence of depression and an anxiety disorder.

When age-corrected rates of depression and anxiety were calculated, the earliest onset age of an anxiety disorder was 3 years, while the earliest onset age for depression was 6 years. There were no significant differences in rates of anxiety or depression by sex of the child. Several proband characteristics were examined in order to determine whether they increased risk of major depression, any anxiety disorder, or any DSM-III diagnosis in the child. Number of episodes of depression, childhood history of enuresis, and the number of first-degree relatives with major depression and anxiety all increased risk for DSM-III diagnoses. Characteristics that did not increase risk were: current age, sex, social class, number of children in the family, marital status, childhood history of stuttering or sleepwalking, and separation from the parent as a child.

Hammen, Adrian, Gordon, Burge, Jaenicke, and Hiroto (1987) evaluated the separate contributions of maternal depressive history, as measured by the Research Diagnostic Criteria (RDC) and number of hospitalizations and episodes, and current maternal depressive symptomatology, as measured by the Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Parents were recruited from inpatient facilities, outpatient clinics, and private referrals. Chil-

dren (ages 8 to 16) whose mothers were either unipolar depressives, bipolar depressives, medically ill, or normal were compared. Hierarchical multiple-regression analyses were run to test the independent contributions of maternal symptomatology and history on their child's functioning, as measured by the Child Behavior Checklist (Achenbach, 1978; Achenbach & Edelbrock, 1983), the Kiddie-Schedule for Affective Disorders and Schizophrenia (K-SADS) (Puig-Antich & Chambers, 1978), the CDI, and the Conners Teacher Rating Scale (Conners, 1969). Although maternal depressive history did not contribute significantly to the child's current functioning, current maternal depressive symptomatology was a significant predictor, as assessed by all measures.

Children's K-SADS diagnosis indicated a very high lifetime rate of psychopathology, including minor disorders, in the children of unipolar (74%) and bipolar (92%) depressives. Moderately high rates of psychopathology were found in the children of medically ill (50%) parents, whereas significantly lower rates (29%) were found for the children of normal parents. Rates for affective disorder (including minor depression) in the children showed a similar pattern: unipolar, 74%; bipolar, 67%; medically ill, 44%; and normal, 17%. Children of unipolar depressives actually showed higher rates of major depression (42%) and dysthymic disorder (32%) than did children of bipolar depressives (25% and 8%, respectively). Analyses were not reported separately for age or gender of the child.

Summary

Despite methodological limitations, children of affectively ill parents appear to be at an increased risk for a variety of disorders, particularly depressive symptomatology and clinical depression (Beardslee, Bemporad, Keller, & Klerman, 1983; Hammen *et al.*, 1987; Orvaschel, 1983). There also appears to be a high rate of other psychiatric diagnoses, such as attention deficit disorders and separation anxiety (Weissman *et al.*, 1984a), particularly when the parent has a dual diagnosis (Weissman *et al.*, 1984b).

In addition, there is evidence that the detrimental effects of parental depression are related more to current maternal depressive symptomatology than to maternal depressive history (Hammen *et al.*, 1987). In line with this, children of nonchronic, but untreated, parents have more psychological and behavioral problems than children of chronic, but treated, parents (Billings & Moos, 1983). Further, children of remitted parents function at a higher level than do children of nonremitted parents (Billings & Moos, 1985). The few studies that report analyses separately for age and gender of the child (Billings & Moos, 1983, 1985; McKnew *et al.*, 1979; Weissman *et al.*, 1984a,b) find no significant differences. Proband characteristics that significantly increase risk for psychiatric diagnosis are: the number of first-degree relatives with a diagnosis, as well as early age of onset of depression, and parent's marital status (Weissman *et al.* 1984a,b).

When parents of depressives are compared with parents of other disabled groups (e.g., the medically ill), both groups show increased rates of childhood psychopathology (Hammen *et al.*, 1987). However, being the child of a depressed parent puts one at even higher risk for a variety of problems, particularly depression. Even though these data suggest that parental depression may be an important vulnerability factor, the question of etiology remains unanswered. Although it is difficult to tease out genetic from environmental influences, it is interesting that as many as 60% of the children of affectively ill parents seem to function normally, without evidence of psychopathology. Recently, then, there has been a burgeoning interest in identifying the particular parental circumstances or characteristics that moderate or intensify the impact of parents' psychopathology on their offspring, including the occurrence of life events and impoverished or faulty family interaction patterns.

Life Events Associated with Parental Depression

Evidence

Billings and Moos (1983) compared 135 children whose parents were either diagnosed with major or minor depression (according to the RDC), with 135 children of nondepressed parents. Subjects were contacted for data collection at a treatment facility (inpatients, $n = 29$) or at home (outpatients, $n = 104$). Stressors were assessed in six areas: (1) negative life events (the number of 15 possible events, such as income decreased substantially, job loss, death of a spouse, marital separation that occurred during the previous 12 months); (2) parental illness; (3) spouse illness; (4) negative physical home environment; (5) family arguments; and (6) work stress. Family resources were evaluated on three indices: (1) quality of significant relationships; (2) family support; and (3) joint family activities.

Results showed that depressed parents reported significantly more family stressors and significantly less family support and resources than did normal parents on all dimensions, except negative aspects of work stress and home environment. Number of family stressors had a negative impact on the child's physical and psychological health, in both the depressed and nondepressed groups. Conversely, family support was associated with fewer child symptoms. Stepwise multiple-regression analyses were conducted, in which parental depression was entered first, acute versus chronic status second, stress third, and support last. High family stress and low family support added incrementally to the prediction of childhood disturbance, beyond that accounted for by having a depressed parent. In general, family resources and stressors were more strongly related to the child's health when parental depression was acute than when it was chronic. There was no evidence that the effects of parental depression varied with the age or gender of the child or with the gender of the parent. Analyses were not presented separately for parents with major and minor depression.

In a 1-year follow-up, families of remitted parents reported significant improvements in family support and lower levels of family conflict than did families of nonremitted parents (Billings & Moos, 1985); yet, in spite of this, their children continued to display higher levels of dysfunction in comparison with controls. This may be due to the fact that remitted families reported greater levels of stressors and parental dysfunction than did controls. However, children of remitted parents showed consistently lower levels of psychological, physical, and behavioral problems than did children of nonremitted parents. In general, parental functioning, family stressors, and resources were all linked significantly to children's health problems, in all groups, as indexed by a total health-problem composite (a summary index of 14 possible physical and psychological problems).

Webster-Stratton and Hammond (1988) investigated maternal depression and its relation to life stress and child conduct problems. Depressed (n = 46) and nondepressed (n = 49) mothers and their 3- to 8-year-old children, referred to a clinic for conduct disorder, were examined. Results from the child domain of the Parenting Stress Index (PSI) (Abidin, 1983) indicated that both the depressed and nondepressed mothers reported extremely high (99th percentile) levels of stress because of their children's difficult temperament. These scores were significantly higher for mothers than for fathers, across both groups. In addition, depressed mothers reported significantly more problems related to attachment, depression, role restriction, competence, social isolation, and health (on the PSI parent scale). They also reported significantly more negative life events over the previous year than nondepressed women did (as assessed by the Life Experiences Survey) (Sarason, Jenson, & Seigel, 1978).

Finally, an interview assessing family background measures revealed more frequent abuse (by their parents, spouse, or former partner) in depressed mothers. However, there was no significant difference in how supportive they perceived their fathers or mothers to be. In general, mothers' reports of stress on the PSI parent domain and negative life events were the most potent variables discriminating depressed from nondepressed mothers. However, maternal depression was not linked to increased child conduct problems. In fact, children of depressed mothers were found to be less deviant than children of nondepressed mothers. This result may reflect a ceiling effect, since all the children were already displaying high rates of deviant behavior at a very young age. However, it is also possible that these children were experiencing high rates of depression or other forms of psychopathology that were not assessed.

Hammen *et al.* (1987) compared four groups of children (29 boys and 29 girls with a mean age of 12.38 years) whose mothers were either unipolar depressives, bipolar depressives, medically ill, or normal. Mothers were recruited from inpatient facilities, outpatient clinics, and private referrals. Chronic strains were assessed and coded on the basis of an interview in which role content areas (marital, social, employment, finances, relationships with children and family) were assessed and rated. For example, if a family had more than enough money, their finance rating was coded as a 5; 4 indicated that the family was comfortable; 3 meant that the family had enough money for essentials; a rating of 2 signified "meager" conditions; and a rating of 1 indicated poverty, lack of housing, and so forth. Interrater reliability and test–retest scores were high. Unipolar depressives reported higher levels of stress than the other three groups, which did not differ significantly from one another. Moreover, chronic strain was found to contribute significantly to the predictive value of several of the children's outcomes (self-ratings of depression, teacher ratings of depression, psychiatric diagnosis), beyond the effects attributable to maternal depression alone. Age and gender effects were not reported.

Hirsch, Moos, and Reischl (1985) compared three groups of adolescents: 16 with a clinically depressed parent, 16 with a parent suffering from rheumatoid arthritis, and 16 with a parent free from psychological or physical disabilities. All depressed parents were in treatment at a psychiatric facility and were diagnosed with either major or minor unipolar depression (according to RDC criteria), with no manic symptoms and no indication that the depressive symptoms were due to neuropsychological or metabolic disorders. Four of the depressed parents were treated as inpatients, and the others received outpatient care.

Only the adolescents of a depressed parent reported more total symptoms, as assessed by the Hopkins Symptom Checklist (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974). More detailed analyses of the 5 subscales of the Hopkins checklist revealed significant differences on the obsessive-compulsive and depressive subscales, but not on the somatization, interpersonal sensitivity, or anxiety subscales. Children of the depressed group and the arthritic group of parents reported significantly poorer self-esteem (Rosenberg, 1965). Adolescents in the normal control group reported the fewest number of negative life events, as measured by the Life Event Checklist (Johnson & McCutcheon, 1980), and adolescents with a depressed parent reported the largest number of negative life events. Negative life events were significantly correlated with number of symptoms in the depressed and in the arthritic groups, but not in the normal group. Positive life events were negatively correlated with number of symptoms in the normal group, but positively correlated in the depressed and in the arthritic groups. Separate analyses for gender and age were not reported.

Summary

In general, depressed parents report significantly more family stressors and significantly less family support and resources than do normal or medically ill parents (Billings & Moos, 1983, 1985; Hammen *et al.*, 1987; Hirsch *et al.*, 1985). Further, negative events in the lives of depressed parents appear to have an adverse effect on their children's health and functioning (Billings & Moos, 1983, 1985; Hammen *et al.*, 1987). Children with an acutely depressed parent, high family stress, and low family support are the most likely to exhibit problems in the areas of academic achievement, school discipline, and peer relations (Billings & Moos, 1983). In addition, they display more physical and emotional health problems (Billings & Moos, 1983). Even though parental chronic strains contribute significantly to children's outcome measures, they do not necessarily put them at risk for conduct disorders (Webster-Stratton & Hammond, 1988). Finally, as compared to adolescents with a normal or medically ill parent, adolescents with a depressed parent report the largest number of negative life events and the greatest number of symptoms (Hirsch *et al.*, 1985).

Overall, being in a family with a depressed parent appears to put one at an increased risk for stress. Indeed, remitted parents report significant improvements in family support and lower levels of family conflict than nonremitted parents (Billings & Moos, 1985). There is no evidence for age or gender effects (Billings & Moos, 1983, 1985). Further work needs to be conducted in order to clarify (1) the way in which parents' stressful life experiences affect the child, and (2) the separate contributions of specific stressors (Hammen *et al.*, 1987).

Family Interaction Patterns of Depressed Parents

Evidence

A parent is the primary environment of an infant. Quality of early parent–child attachment has been associated with the child's later functioning: insecurely attached children have been found to be less social, more fearful of strangers, and more prone to behavior problems than are securely attached children (Radke-Yarrow, Cummings, Kuczynski, & Chapman, 1985). Infants of affectively ill parents appear to be less securely attached than infants of normal, never psychiatrically ill parents. For example, Radke-Yarrow *et al.* (1985) studied patterns of attachment in 2- and 3-year-olds in normal families ($n = 31$) and families with a mother with major bipolar depression ($n = 14$), major unipolar depression ($n = 31$), and minor depression ($n = 31$). The mothers, recruited from advertising, included 7 mothers who had been hospitalized (3 during their child's lifetime), 25 who had received drug treatment (4 currently), and 51 who had sought professional help (19 were currently seeing a professional). The "Strange Situation" procedure was employed, which involves 8 brief episodes in which the child is separated and reunited with the mother (Ainsworth & Wittig, 1969).

Insecure attachments, observed during the session, were most common in families with a unipolar

depressive mother (79%) as compared with normal families (24%). A number of moderating variables increased significantly the likelihood of insecure attachments, including severity of the mother's illness, absence of father, and percentage of child's lifetime that the mother was ill. The multiple correlation between attachment classification and maternal depression variables was .38, accounting for 15% of the variance. Stepwise multiple-regression analyses revealed that mother's diagnosis was the best predictor of attachment classification, followed by the severity of her worst episode (see also Davenport, Zahn-Waxler, Adland, & Mayfield, 1984). Mothers' self-assessed mood during the experiment was unrelated to diagnosis. In contrast, composite scores of negative and positive emotions expressed during the experiment showed that mothers of securely attached children expressed more positive, and less negative, affect than did mothers of insecurely attached infants. Mothers' expressed emotions during the testing periods contributed significantly (5%) to the variance accounted for in attachment classification.

In addition to attachment, studies show that the parenting behavior of individuals with an affective disorder is impaired. McLean (1974) found that 68% of depressed parents indicated that they were parenting ineffectively. Weissman, Paykel, and Klerman (1972) studied 40 outpatient depressed women, judged by their psychiatrist to have moderate to severe depression, and compared them to a group of 40 normal women. The mean age of the children was 11 years. During an acute phase of maternal depression, mothers were found, on the Social Adjustment Scale (SAS) (Gurland, Yorkston, Frank, & Stone, 1967), to be significantly more impaired than their matched counterparts were. Highly significant differences in parental role functioning were found between the depressed and normal parents, on seven of the eight SAS items that assessed maternal role performance. Specifically, depressed women felt only moderately involved in their children's daily lives, reported difficulty in communicating with their children, were less affectionate, reported having considerable friction with their children, moderate degrees of guilt, and considerable resentment and ambivalence toward family members. In contrast, normal women reported that they were relatively harmonious, involved and affectionate with their children, and did not report significant guilt or resentment. The interviewer's global ratings were consistent with these reports. The average rating for maternal role performance in normal women was in the nonimpaired range, whereas the depressed women were judged to have moderate degrees of impairment.

More detailed observations, derived from clinical records and narrative notes of the psychotherapists, were also examined. Five stages of the family life cycle were retrospectively evaluated: (1) care of the newborn—the postpartum stage; (2) nurturance of the infant—the infancy stage; (3) enculturation of the young child—the childhood stage; (4) emancipation of the offspring from the family—the adolescent in the family; and (5) termination of the child-rearing process—"the empty nest." Of the 40 mothers who were evaluated, 35 had children and 3 of them currently had an infant in the postpartum stage. All three women were experiencing postpartum depression and were immobilized in carrying out the maternal role. They felt helpless, overwhelmed, and inadequate. Eleven women had 16 children in the nurturance stage. Seventy-three percent of the depressed mothers were either helpless and guilty or directly hostile. Eighty-one percent of their children were more vulnerable and displayed more tyrannical behavior than did their nondepressed counterparts.

Ten of the 20 depressed mothers with children in the enculturation stage had problems: the mothers were emotionally and physically uninvolved or overinvolved, and displayed higher rates of friction, irritability, self-preoccupation, withdrawal, and ambivalence than did the nondepressed mothers. The children displayed increased peer and sibling rivalry and hyperactivity, and had more school problems, higher rates of enuresis, and other symptoms, and more feelings of isolation or depression than did the children of nondepressed mothers. Eighty-one percent of the depressed mothers and 65% of their children experienced difficulty with the emancipation stage. Mothers reported impaired communication, increased friction and withdrawal, and higher levels of worry, guilt, and resentment. Their adolescents were more deviant, rebellious, and withdrawn. Sixty percent of the depressed mothers and 38% of their children were experiencing problems in the termination stage. The mothers reported higher rates of friction whereas their children were rebellious and guilty. In general, acute depressive illness was found to impair significantly the depressed woman's mothering ability as well as the child's coping ability.

Billings and Moos (1983) compared 133 parents with major or minor depression (43 men and 90 women) with 135 matched nondepressed control parents. Twenty-nine inpatients and 104 outpatients were recruited for data collection. Depressed

families were characterized by less cohesion, expressiveness, independence and organization, and more conflict than were nondepressed families, as measured by the Family Environment Scale (FES) (Moos & Moos, 1981). No differences were found between the depressed and nondepressed families in terms of achievement orientation, intellectual orientation, or control. Of interest, more severe depression and lack of parental self-confidence in the control group was associated with a greater number of physical and psychological health problems (as assessed by a summary index composed of 14 possible physical and psychological problems) in the child. In contrast, no relation emerged in the patient group, which may reflect a ceiling effect. Finally, the functioning of both the depressed parent's spouse and the control's spouse was significantly related to children's health. There was no indication that gender or age of the child or gender of the parent had an impact.

In a 1-year follow-up, remitted families reported higher levels of cohesion and expressiveness (as assessed by the FES) than did nonremitted families (Billings & Moos, 1985). Nonremitted patients' families continued to report lower levels of cohesion, expressiveness, independence, recreational orientation, organization, and higher levels of conflict than did control families. In contrast, the family milieus of remitted parents did not differ significantly from controls, except that they were higher in intellectual-cultural orientation. The children of remitted parents continued to evidence more dysfunction than controls, but fared better than did the children of nonremitted parents.

The study by Hirsch *et al.* (1985) discussed above is also relevant here. Among adolescents of a depressed parent, those with higher self-esteem (as assessed by the Rosenberg self-esteem scale) had a more cohesive, expressive family that encouraged independence and had low conflict (as measured by the FES). Among the normal group, higher self-esteem was correlated with a more cohesive, intellectually oriented active family, with low conflict. No significant family variables were related to self-esteem in the arthritic group. Surprisingly, in both the depressed and the normal groups, family social climate was unrelated to symptomatology (as measured by the Hopkins Symptom Checklist). In contrast, a more cohesive and expressive family with low conflict was significantly associated with decreased symptomatology (as assessed by the Hopkins checklist) in the arthritic group. Separate analyses were not reported for age or gender.

Rogers and Forehand (1983) examined the relationship between maternal depression and each of three dimensions of interaction between parents and their clinic-referred children: child behavior, parent behavior, and parent perceptions of their children. Three groups were created, according to the level of maternal depression on the BDI (low, medium, and high). Surprisingly, there were no differences in maternal behavior observed during coded parent–child interactions at home, across the BDI levels. Furthermore, observers (using a Behavior Coding System) found no differences in measures of child compliance and deviant behavior. In contrast, parental perceptions of their child's behaviors on the Parent Attitude Test (Cowen, Huser, Beach, & Rappaport, 1970) yielded significant differences: the more depressed the parent was, the more negatively they viewed their child. These results suggest that parents' perceptions may not be based on the actual behavior of the child, but are rather a result of their own depressed state. No age or gender analyses were reported.

Webster-Stratton and Hammond (1988) investigated maternal depression and its relationship to life stress, perception of child behavior problems, parenting behavior, and child conduct problems. Forty-six depressed mothers (as measured by the BDI) and 49 nondepressed mothers and their children (3 to 8 years old), referred to a clinic for conduct problems, were assessed. Several researchers have theorized that maternal depression leads to negative perceptions of children, increased commands and criticisms, and finally to increased child conduct problems (Anthony, 1983; Patterson, 1982; Rogers & Forehand, 1983). Depressed mothers perceived their children to be more disturbed and were much more critical of them than nondepressed mothers were. In contrast, home observer behavior ratings, conducted on two evenings during the week (usually dinner time), revealed no significant differences between the behavior of children of depressed and nondepressed mothers. The last link in the chain, increased child conduct problems, was not supported. In fact, children of depressed mothers were found to be less deviant than were children of nondepressed mothers.

Summary

The fact that maternal depression reduces the likelihood of a secure attachment indicates that an affectively ill mother's ability to relate to her infant may be impaired (Radke-Yarrow *et al.*, 1985). Such maternal characteristics as responsivity and emotional availability may attenuate the impact of

parental depression (Radke-Yarrow *et al.*, 1985), whereas other behaviors such as maternal disorganization, tension, and unhappiness may intensify the impact of maternal depression (Davenport *et al.*, 1983). Further research is needed in order to determine specific qualities of the rearing environment created for the child by parental pathology (Radke-Yarrow *et al.*, 1985).

In depressed families with school-aged children, there is evidence of poor communication, increased parental resentment and ambivalence, and lower levels of affection and involvement (Weissman *et al.*, 1972). Results also show less cohesion, expressiveness, and organization (Billings & Moos, 1983). Importantly, parental interactions seem to have a negative impact on the child's health and functioning (Billings & Moos, 1983; Weissman *et al.*, 1972). These impairments appear to be evident at all stages of the child's development (Weissman *et al.*, 1972). Compared with adolescents of a medically ill parent, the self-esteem of adolescents with a depressed parent is buttressed when the family environment is more cohesive and supportive.

Finally, parental perceptions of children's behavior seem to be highly related to the parents' depressive status (Rogers & Forehand, 1983; Webster-Stratton & Hammond, 1988) and to current parental depressive symptomatology (Billings & Moos, 1985). Future research should investigate what behaviors mediate the relation between parents' depressive mood and children's outcomes, by analyzing and directly observing the interactions between depressed parents and their children (Hammen *et al.*, 1987).

Children's Factors

Life Events Associated with Childhood Depression

Evidence

Mullins, Siegel, & Hodges (1984) examined the correlation between self-reported depression (as measured by the CDI) and self-reported life events, measured by Coddington's Life Events Scale for Children (Coddington, 1972) in 134 fourth-, fifth-, and sixth-grade nonclinical school children. Children's age and sex did not differentiate between the two groups, nor did the parent's socioeconomic or marital status. The number of negative life events (e.g., death of parent, suspension from school, illness) that occurred in the preceding year was positively correlated with current depression. The number of positive life events was not significantly related to depression. Subjective weightings of the positiveness and negativeness of the events did not change the findings. However, because life events were assessed by retrospective and subjective reports, the results should be considered cautiously.

Nolen-Hoeksema, Girgus, and Seligman (1986) explored the relationship between depression, life events, and attributional style in a sample of 308 nonclinic, grade-school children (aged 8 to 11, 87 male and 81 female). Testing took place every 2 to 3 months over a 1-year period. Children were asked to indicate which life events occurred since the previous administration of the questionnaire. The number of life events reported by the child was significantly correlated with depression at each testing period, indicating that the number of events occurring prior to an administration was related to the level of depression reported at that administration. In addition, for each pair of adjacent testing periods, multiple regression analyses were run to test whether negative attributional style and life events together interacted to predict depression better than either variable alone. In two of the four analyses (administrations 2 and 3, and administrations 4 and 5), the interaction between attributional style and life events significantly predicted levels of depression, after partialing out the terms for initial levels of depression, life events, and explanatory style. In the other two analyses, the interaction term was not significant. Analyses were not reported separately for age or sex of the child.

Cohen-Sandler, Berman, and King (1982) explored the relationship between total life stresses ever experienced and psychological disorders, in a sample of 76 children aged 5 to 14, discharged from an inpatient psychiatric unit. Based on descriptions of symptomatology, behavior, and family history contained in the Screening Intake and Ward Summaries, the sample was divided into three groups: suicidal, depressed, and psychiatric control. Using information in the child's chart, five developmental periods were created: infancy, preschool, early childhood, later childhood, 12 months prior to admission, and total. Suicidal children did not differ from either depressed or controls in age, sex, race, previous treatment history, weeks in current treatment, or family psychiatric and suicidal history. However, unlike the depressed and control groups, the suicidal group experienced an increasing amount of stress as they matured, particularly during the year prior to admission.

Particular types of stressors appeared to differ-

entiate suicidal from other children, across the different developmental periods. During infancy and preschool, psychiatric controls were separated from their parents significantly more often than were the depressed and suicidal groups. In contrast, the suicidal group usually remained at home, but was joined by a third adult (typically the maternal grandmother). By early childhood (age 4½), suicidal children more frequently experienced the birth of a sibling, divorce of their parents, or medical hospitalization of their parents. Depressed children, on the other hand, experienced more school failure than did the suicidal or control groups. From the time suicidal children entered school to just prior to their admission, they experienced a disproportionate number of losses of all kinds, including birth of a sibling, death of a grandparent, hospitalization, separation, divorce or remarriage of a parent, a broken home, and psychological trauma (e.g., witnessing death). In contrast, depressed children experienced more rejection by their peers. This pattern persisted during the 12 months prior to admission.

Summary

Only a handful of researchers have attempted to determine what the immediate effects of negative life events are for depression in childhood. The above studies provide preliminary evidence linking the occurrence of negative, but not positive, life events with ongoing and subsequent depression in childhood (Mullins *et al.*, 1984; Nolen-Hoeksema *et al.*, 1986). High levels of life stress are also associated with suicidal behavior (Cohen-Sandler *et al.*, 1982). Moreover, certain types of stressors appear to differentiate depressed from suicidal from normal children. Specifically, suicidal adolescents experience a greater number of losses (i.e., death and illness), whereas depressed children experience higher rates of peer rejection (Cohen-Sandler *et al.*, 1982). In the two studies that analyzed separately for sex and age, no differences were found (Cohen-Sandler *et al.*, 1982; Mullins *et al.*, 1984).

Research on the children of affectively ill parents has also found that parental depression is associated with high levels of family stress and low levels of family support. Further, children with high family stress are significantly more dysfunctional than are children with low family stress (Billings & Moos, 1983). Some theorists (Bowlby, 1980; Brown & Harris, 1978) suggest that early loss situations lead to the development of negative cognitive sets in childhood, which cause the child to interpret later losses as failure and thereby lead to feelings of hopelessness, helplessness, and depression (see Chapter 23 in this volume). Future research is needed in which stressful life events are prospectively and objectively assessed, teasing out the separate contributions of specific stressors and moderating factors.

Family Interaction Patterns of Depressed Children

Evidence

In an early study, Poznanski and Zrull (1970) found that parents of depressed, psychiatric inpatient children were often angry, punitive, detached, and belittling in their relations with their children. Parental neglect was noted in 2 of the 14 cases. More recently, Puig-Antich, Lukens, Davies, Goetz, Brennan-Quattrock, and Todak (1985) compared depressed children who were outpatients, nondepressed psychiatric controls, and normal, never psychiatrically ill children. According to the Psychosocial Schedule for School-Aged Children, depressed children had poorer communication and less affectionate relations with their mothers (see also Burbach & Borduin, 1986; Puig-Antich, Blau, Mark, Greenhill, & Chambers, 1978).

Cole and Rehm (1986) studied families of clinically depressed outpatients, clinically nondepressed outpatients, and normal, nonclinic children (as assessed by the K-SADS, the DSM-III, and the CDI) in a family interaction task. The children ranged in age from 8 to 12 years old. The experimenter instructed the child to roll a ball through a maze and try to get it into the highest numbered hole. Parents were told to encourage their child while they were playing the game. Mothers of depressed children expressed less positive affect during the session than did nonclinic mothers, at all levels of the child's performance. These results could not be accounted for by the mother's level of depression. Fathers of depressed children increased positive affect only at highest performance. Although parents rarely expressed negative affect, depressed children expressed much more negative affect during the task than did the nondepressed clinic group. Self-report data indicated that mothers of depressed and nonclinic children set more stringent standards than mothers of nondepressed clinic children. Further, depressed children reported higher standards for their own performance than did nondepressed children. Separate analyses for sex and age were not reported.

Amanat and Butler (1984) compared an outpatient sample of 12 moderately to severely depressed children (aged 7 to 14 years) with 11 children suffering from overanxious reaction of childhood (according to a psychiatric evaluation and scores on the Child Center Depression Inventory). Parents and their children were interviewed and asked to respond to questions from an "oppressive behavior" inventory. These measures were then combined by two independent raters. The parents also filled out a self-rating scale to assess depressive tendencies and authoritarian attitudes.

Results revealed that depressed families tended toward absolute control and dominance whereas anxious families tended toward chaos. In families of depressed children, children's self-expression and autonomy was suppressed in 75% of the decision-making situations; families of overanxious children revealed these oppressive attitudes in only 38% of the cases. In 90% of the families with overanxious children, projection and external blame were frequently used. In contrast, 70% of the depressed children used introjection and self-blame. No differences were found in self-expression of parents, invasiveness and intrusion, empathy with child's needs, attitudes toward chores, time needs and space, and provision of comfort. Analyses were not reported separately for different ages or sex of the child.

Kaslow, Rehm, and Siegel (1984) classified 108 elementary school children in Grades 1, 4, and 8 as depressed and nondepressed, according to the CDI. Although depression scores were lower for the younger than the older children, the differences obtained were not significant. A family questionnaire was administered, containing 8 items that asked the child about (1) how much he or she enjoys being with his or her family; (2) how well he or she gets along with his or her parents; and (3) how well his or her parents get along with each other. Analyses were conducted for the entire sample and for each grade separately. Across all age groups, depressed children reported more overall family dysfunction than did nondepressed children.

Summary

Few attempts have been made to study the family relations of depressed children. Evidence exists that depressed children perceive their family environment to be more distressed and dysfunctional than do control children (Kaslow *et al.*, 1984). In addition, families of depressed children appear to display a variety of oppressive attitudes. Specifically, they set more stringent standards, display less affection, and tend toward absolute control and dominance (Amanat & Butler, 1984; Cole & Rehm, 1986). During a family interaction task, mothers of depressed children express less positive affect than do mothers of nondepressed children, whereas fathers of depressed children increase positive affect only at the highest performance level (Cole & Rehm, 1986). Results further suggest greater suppression of self-expression and autonomy and more use of introjection and self-blame in the families of depressed, but not overanxious, children (Amanat & Butler, 1984). In the only study that analyzed separately for age and sex of the child (Kaslow *et al.*, 1984), no significant differences were found.

These results are consistent with the literature examining the family interaction patterns of depressed parents and their children. Families with depressed parents report less cohesion, expressiveness, independence, and organization, as well as poorer communication and increased resentment (Billings & Moos, 1983; Weissman *et al.*, 1972). Taken together, impaired family interactions may be an important component of depression in children.

Conclusions

The evidence reviewed here suggests that there is a relationship between early loss and childhood depression as indicated by (1) a high rate of psychological dysfunction, particularly depression, among the children of depressed parents; (2) an increased rate of dysfunction in the children of depressed parents with high levels of stress; and (3) impairment in the family interaction patterns of depressed parents. There is also evidence for direct provoking agents in the lives of depressed children, in terms of both the experience of negative events and a high rate of family conflict and dysfunction (suggesting a lack of social support). These data are consistent with the perspective of Brown *et al.* (1986) on the development of depression.

Future work should address the methodological limitations that currently exist in the literature. For example, studies often do not include a control group (e.g., McKnew *et al.*, 1979; Poznanski & Zrull, 1970; Puig-Antich *et al.*, 1978) or do not match the control and experimental groups on relevant factors, such as socioeconomic status, that may impact on depression (McKnew *et al.*, 1979; Poznanski & Zrull, 1970; Puig-Antich *et al.*,

1978). The generalizability of investigations is also limited by small sample sizes (e.g., Cohen-Sandler *et al.*, 1982; Hirsch *et al.*, 1985; McKnew *et al.*, 1979; Weissman *et al.*, 1972; Welner *et al.*, 1977). Much of the evidence reviewed (including Amanat & Butler, 1984; Hammen *et al.*, 1987; McKnew *et al.*, 1979; Welner *et al.*, 1977) evaluates a wide age range of children, without presenting the results separately for different age groups (see Cohen-Sandler *et al.*, 1982; Kaslow *et al.*, 1984; Mullins *et al.*, 1984; Weissman *et al.*, 1972 for exceptions).

With regard to sex of the parent, many of the studies assess only the mother's depression (e.g., Hammen *et al.*, 1987; Radke-Yarrow *et al.*, 1985; Rogers & Forehand, 1983; Webster-Stratton & Hammond, 1988; Weissman *et al.*, 1972; Welner *et al.*, 1977; vs. Billings & Moos, 1983, 1985). Some studies do include depressed fathers, but they usually present data irrespective of the sex of the parent (e.g., Hirsch *et al.*, 1985; McKnew *et al.*, 1979; Weissman *et al.*, 1984a,b).

In addition to the above, studies of both depressed parents and children sometimes include a mixed population, grouping depressed inpatients and outpatients of varying levels of severity together (Billings & Moos, 1983, 1985; Hammen *et al.*, 1987). It may well prove important to consider distinct diagnostic groups separately and to explore the implications of change in diagnostic status (Billings & Moos, 1983, 1985). It would also be useful to obtain information from multiple sources (e.g., direct child interviews, parent interviews, and teacher interviews). Finally, assessment and criterion measures vary from study to study, making it difficult to draw cross-study comparisons. Careful consideration of these factors, along with a focus on differentiating specific types of foundational life stress and current adverse experiences, should help to refine and clarify our etiological models.

ACKNOWLEDGMENTS

We are indebted to D. Durbin, F. Quintos, B. Bogdnoff, and W. Shadel for their assistance. This research was partially supported by Temple University Research Incentive Fund and Temple University Grant-In-Aid of Research to Suzanne M. Miller.

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CHAPTER 25

Psychobiology of Childhood Depression

Patrick Burke and Joaquim Puig-Antich

Over the past decade, childhood depression has gained increasing acceptance as a clinical entity (Puig-Antich, 1986). Earlier debates as to the existence or nonexistence of the syndrome in youth have given way to a variety of empirical studies investigating methods of assessment (Chambers *et al.*, 1985; Kovacs, 1986), phenomenology (Ryan, Puig-Antich, Ambrosini, Rabinovitch, Robinson and Nelson, 1987; Mitchell, McCauley, Burke, and Moss, 1988; Mitchell 1988), outcome (Kovacs, Feinberg, Crouse-Novak, Pauluskas, & Finkelstein, 1984), and family history (Strober, 1984).

In addition, there has been a substantial increase in research on biological aspects of childhood depression (Puig-Antich, 1986). Investigators are interested in delineating similarities and differences in neurotransmitter functioning between the childhood and adult forms of depression. Moreover, childhood depression may provide a unique opportunity to study potential markers of depression. Evidence to be reviewed below suggests that childhood depression is associated with an unusually high family loading for affective illness in comparison to depression of later onset. Thus, alterations in biological indices reflecting underlying trait markers might well be evident in this age group. Finally, the study of childhood depression provides a unique opportunity to examine developmental aspects of depressive phenomena.

In this chapter, we review family history studies, neuroendocrine and sleep research, and treatment of childhood depression using antidepressants.

Family History Studies of Childhood Depression

In general, two study designs have been used to determine if children and adults with affective disorders cluster in the same families. In one design, the lifetime psychiatric diagnoses of first- and second-degree relatives of children and adolescents with major depression are ascertained. Data may be collected by the so-called family history method, in which parents are interviewed about other relatives, or by the family study method, a more sensitive technique in which first- and second-degree relatives are directly interviewed (Thompson, Orvaschel, Prusoff, & Kidd, 1982). In the second design, the offspring of adults with unipolar or bipolar affective disorders are studied.

A number of studies have shown that the age-corrected lifetime morbidity risk for major depression in the first-degree relatives of adults with major depression is between 0.18 and 0.30 (Gershon, Bunney, Leckman, van Eerdewegh, & DeBauche,

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1976; Gershon, Targum, Kessler, Mazure, & Bunney, 1977; Weissman *et al.*, 1984), with the risk being lowest for late onset unipolar probands and highest for bipolar and schizoaffective probands. Weissman *et al.* (1984) reported a large inverse relationship between age of onset of major depression in a family study of 133 adult probands and controls and the risk of major depression in their relatives. The highest risk occurred in relatives of probands with an age at onset of depression under 20 years of age compared with relatives of probands with a later age of onset, or with relatives of controls. There was little difference in the risk for relatives of probands with an age of onset of 40 years or over than for the relatives of control subjects.

There are also complex relationships between affective illness and other diagnoses, for example, alcoholism and anxiety disorders. Leckman, Weissman, Merikangas, Pauls, and Prusoff (1983) found that relatives of adult probands with panic disorder in addition to major depression had greater risks of alcoholism in addition to depression, panic disorder, and phobia than probands with depression only. Probands with depression and panic also had an earlier age of onset of depression, more alcoholism, and more suicide attempts than probands with depression only. Merikangas, Leckman, Prusoff, Pauls, and Weissman (1985) found that adult probands with depression, but not alcoholism, and probands with depression and alcoholism showed different patterns of transmission of alcoholism.

Studies of family history in children and adolescents are in some respects similar to the above. In adolescents, the age-corrected morbidity for depression in one study of relatives of inpatient probands with 0.35 (Strober, Burroughs, & Salkin, 1989), with a higher risk in relatives of bipolar rather than unipolar probands. In a study of outpatient depressed adolescents, the risk for depression was 0.37 among adult first-degree relatives (Puig-Antich, Ryan, & Larson, unpublished manuscript). In prepubertal depressed probands, Puig-Antich, Goetz, Davies, Kaplan, Ostrow, and Asnis *et al.* (1989) found that the age-corrected morbidity risk for first-degree relatives over 18 years of age was 0.53. Thus, there appears to be an increased risk or greater familial aggregation in the younger age groups, although the child and adolescent data were largely collected by the less sensitive family history method. Mitchell, McCauley, Burke, Calderon, and Schloredt (1989) similarly reported high rates of depression in the parents, particularly mothers of depressed probands. Although further study is needed before definitive conclusions can be drawn, the data are consistent with the studies in adults reviewed above, that show increased familial aggregation in probands with a younger age of onset (Weissman *et al.*, 1984). Furthermore, in depressed child and adolescent probands, the risk in relatives is increased for alcoholism (Puig-Antich, 1987; Strober, 1984), and anxiety disorders (Puig-Antich & Rabinovich, 1986; Puig-Antich *et al.*, 1989), and mothers of younger individuals may have more marked histories of substance abuse and suicidality (Mitchell *et al.*, 1989). These studies in conjunction with the findings in adults suggest complex relationships between age at onset of depression, which is likely associated with increased familial aggregation, and associated diagnoses of alcoholism and anxiety disorders.

Offspring of Adult Probands

A number of studies have shown that the children of adults with depression are at considerable risk for depression and other disorders (Keller, Beardslee, Dorer, Lavori, Samuelson, & Klerman, 1986; Weissman, Gammon, John, Merikangas, Warner, & Prusoff, 1987; Weissman, Leckman, Merikangas, Gammon, & Prusoff, 1982; Welner, Welner, McCray, & Leonard, 1977). Studies suggest that for children under 18 years of age, the morbidity risk increases as a function of parental depression, including recurrent depression, family history of psychiatric disorders, and parental and assortative mating. Moreover, an associated diagnosis of panic disorder or agoraphobia in parents increases the risk of separation anxiety as well as depression in offspring. Akiskal, Downs, Jordan, Watson, Daugherty, and Pruitt (1985) found in a 3-year follow-up study that there was an increased risk of bipolar disorder or cyclothymia in the offspring or younger siblings of adult bipolar probands. Over half the sample showed either mania, hypomania, a mixed disorder, or cyclothymia.

Twin and Adoption Studies

As yet, neither adoption nor twin studies of childhood depression have been reported. In adults, higher concordance rates for affective illness have been reported in monozygotic rather than in dizygotic twin pairs, and are higher for bipolar rather than for unipolar disorders (Bertelsen, Harvald, & Hauge, 1977; Gershon *et al.*, 1976; Zerbin-Rudin, 1972). Data from adoption studies support the importance of both genetic and environmental factors.

Mendlewicz and Rainer (1977) found that bipolar illness in adoptees was associated with affective illness in biological but not in adoptive parents. However, in other studies, evidence for environmental transmission was found. Cadoret, Gorman, Heywood, and Troughton (1985) reported that depression was not correlated significantly with affective illness in biological relatives of adoptees. Factors in the adoptive families were also significant, and had different effects on males and females. In adopted males, the risk of depression was increased if another individual in the adoptive home had alcoholism. For females, the risk of depression was increased if an adoptive parent died before 19 years of age, and if another individual in the adoptive home had a behavior problem. Von Knorring, Cloninger, Bohman, and Sigvardsson (1983) found a similarly nonsignificant relationship between depression in adoptees and affective illness in biological relatives.

Neuroendocrine Studies

Cortisol Secretion Patterns

In concert with the tremendous growth in studies of cortisol dynamics in adult depression, most researchers on childhood depression have focused on cortisol secretion patterns, the dexamethasone suppression test, and other challenge tests. Puig-Antich, Blau, Marx, Greenhill, and Chambers (1978) reported that two of four endogenously depressed prepubertal children showed cortisol hypersecretion in a pattern typical of that described for endogenously depressed adults. Subsequent more extensive studies have failed to confirm these findings. Puig-Antich, Dahl, Ryan, Novacenko, Goetz, Goetz, *et al* (in press) measured plasma cortisol concentrations every 20 minutes for 24 hours in 45 depressed prepubertal children and controls. There were no significant differences among the groups in plasma cortisol concentration as measured by 24-hour mean, peak, nocturnal rise, or nadir. There were also no differences among subgroups of depressed children based on endogenicity, psychotic symptoms, or suicidality. Hypersecretion of cortisol occured in only four depressed children, and occured also in one normal control. Identical results were obtained in a similar study of depressed adolescents (Dahl, Ryan, Nelson, Novacenko, *et al.* (in press). These results contrast with the typical finding in adults that 40% of endogenously depressed individuals show a pattern of hypersecretion with more secretory episodes, more minutes of active secretion, and continued high secretion of cortisol into the late evening and early morning hours (Sachar, Hellman, Roffwarg, Halpern, Fukushima, & Gallagher, 1973). There is also no significant change in the latency of cortisol secretion in young subjects in contrast to adults where cortisol latency is shortened (Jarrett, Coble, & Kupfer, 1983). These differences in cortisol secretion patterns during an episode of depression between children and adults are consistent with data showing a positive correlation between cortisol hypersecretion and age in endogenously depressed adults (Asnis, Sachar, Halbrich, Nathan, Novacenko, & Ostrow, 1981; Stokes *et al.*, 1984) and in normal adults (Pfohl, Sherman, Schlechte, & Winokur, 1984; Pfohl, Sherman, Schlechte, & Stone 1985) and suggest that age may be an important variable in understanding the relationship between cortisol secretion and depression.

The studies of Puig-Antich *et al,* (in press) and Dahl *et al,* (in press) were carried out in a relatively low-stress environment. Stress has been shown to influence cortisol response to dexamethasone in healthy young adults (Brier, Albus, Pickar, Zahn, Wolkowitz, & Paul, 1987), and the stress of hospitalization may be a factor in nonsuppression of cortisol in depressed adults (Coccaro, Prudic, Rothpearl, Nurnberg, 1984). Thus, stress may also be an important variable in accounting for the differences in the studies to date of cortisol secretion in children, adolescents, and adults.

Dexamethasone Suppression Test

The dexamethasone suppression test (DST) has received considerable attention as a simple potentially useful test for endogenous depression (Carroll, 1985). Typically, 1 mg dexamethasone is given at 11 P.M., and cortisol is measured at 8 A.M. and 4 P.M. the next day. Cortisol values greater than certain cutoffs are said to discriminate endogenous depression. Although initial results were very promising, further studies have shown that the test is less sensitive and less specific than originally thought (Arana, Baldessarini, & Ornsteen, 1985; Carroll, 1985). Relatively few researchers, however, have studied the utility of the test in childhood depression. In preadolescent children, studies to date have shown variable sensitivity (percentage of depressed subjects showing a positive test) and specificity (percentage of nondepressed subjects with a positive test) for the test. The sensitivity of the DST has varied from 14% (Geller, Rogal, &

Knitter, 1983), 26% (Burke, Mitchell, McCanley, Smith, & Moss (unpublished data), and 56% (Posznanski, Carroll, Banegas, Cook, & Grossman, 1982) in primarily outpatient groups, to 70% in an inpatient sample (Weller, Weller, Fristad, & Preskorn, 1984). Specificity has also varied. Posznanski *et al.* (1982), Burke *et al.* (unpublished data) and Doherty, Mandansky, Kraft, Carter-Ake, Rosenthal, and Coughlin (1986) reported high specificities in the range of 88% to 89%. Others, however, have reported lower specificity. Livingston, Reis, and Ringdahl (1984), reported nonsuppression in 3 of 5 children with severe separation anxiety. Petty, Asarnow, Carlson, and Lesser (1985) reported that 5 of 6 nondepressed psychiatrically ill children showed nonsuppression.

Results are similarly variable in adolescents. Although some studies (Extein, Rosenberg, Pottash, & Gald, 1982; Robbins, Alessi, Yanchyschyn, & Colfer, 1982) report good sensitivity and specificity, Targum and Capodanno (1983) reported nonsuppression in 7 of 17 (41%) depressed adolescents, 7 of 38 (18%) adolescents with dysthymia, 7 of 47 (15%) adolescents with conduct disorder, and 4 of 15 (27%) adolescents with schizophreniform disorder. The predictive value of the DST for major depression was only 28%. Burke *et al.* (unpublished data) found that the DST had a low sensitivity (19%) but a high specificity (100%) and predictive value (100%) for major depression in outpatient depressed adolescents. However, the predictive value for endogenous depression was only 40%.

The data, therefore, on the dexamethasone suppression test in depressed children and adolescents are mixed but not dissimilar to adults. A number of factors are known to influence DST results (Carroll, 1985; Rubin, 1985) and might also contribute to the variable results that have been reported in young subjects. Merkle (1982) showed that serum dexamethasone concentrations following a 1-mg dose can be highly variable. Meltzer and Fang (1983) found that differences among laboratory assay methods may contribute to discrepancies between results. Moreover, there is considerable variability in circulating cortisol levels as shown by studies in which cortisol was sampled at 20-minute intervals (Sherman, Pfohl, & Winokur, 1984). Thus, the use of a single or even two or three blood samples might well give rise to spurious results. The clinical characteristics and diagnostic categories of the patient groups studied are also important variables, as there is often poor agreement among clinicians for diagnoses of depression (Carroll, 1985). This is likely an important issue in studies of childhood depression, especially in younger children in whom the diagnosis of endogenous depression can be difficult. Secondary diagnoses, such as separation anxiety or conduct disorder, occur in depressed children and adolescents, thus further contributing to diagnostic heterogeneity.

The data on children and adolescents do not allow definitive conclusions regarding the relationship between age and the DST. A number of studies in adults have shown that age is positively correlated with postdexamethasone cortisol levels (Asnis *et al.*, 1981; Lewis, Coryell, Pfohl, Schlecte, & Corvell, 1984; Stokes *et al.*, 1984). Age and depression might be independent factors interacting additively to produce higher cortisol levels, or age might be a permissive factor such that older subjects are more sensitive to depression-induced changes in the HPA axis. If depression is a heterogeneous disorder with different subtypes having different age distributions, then subtypes of depression might interact with age such that subtypes in which nonsuppression occurs might be more likely in older subjects.

Finally, there is a complex relationship between stress and DST response. Recent studies (Roy, Rickar, Linnoila, Doran, & Paul 1986) have shown that DST nonsuppressors experienced fewer recent life events and had lower cerebrospinal fluid (CSF), HVA, and 5-HIAA serotonin metabolites than did suppressors. Ceulemans, Westenberg, and van Praag (1985), however, found a high rate of nonsuppression in nondepressed men who were about to undergo surgery. Further research on the relationship of recent life events and stress to DST response in children and adolescents is therefore warranted. Interestingly, Siefert, Foxx, and Butler (1980) found that CSF, HVA, and 5-HIAA levels were higher in normal healthy children than in adults.

Cortisol Response to Provocative Challenge

In adults, a number of researchers have studied the cortisol response to provocative tests. Carroll *et al.* (1980) showed that physostigmine, which stimulates cholinergic transmission, induced nonsuppression in normal adults. Thus, cholinergic overactivity might be implicated in at least some depressed patients who are nonsuppressors. Sachar, Asnis, Nathan, Halbreich, Tabrizi, and Halpern (1980) and Sachar *et al.* (1985) found that intravenous amphetamine, which induces signifi-

cant cortisol release in normals, either lowered or produced little increase in cortisol in depressed adults, particularly those with endogenous depression. Because amphetamine acts by increasing adrenergic transmission, the results suggest that noradrenergic underactivity occurs in depression. Further converging evidence that a functional noradrenergic deficit is present is shown by the finding that intramuscular desmethylimipramine (DMI) (Asnis *et al.*, 1985) and clonidine (Siever, Uhde, Jimerson, Port, Lake, & Murphy, 1984) also result in decreased cortisol release in depression. Amphetamine, desmethylimipramine, and clonidine all result in increased stimulation of postsynaptic alpha-2-adrenergic receptors. Thus, abnormal cortisol regulation in depression may reflect a functional adrenergic deficit, possibly secondary to lower alpha-2-receptor sensitivity.

In children, provocative challenge studies are now being attempted. Puig-Antich (unpublished data) found that cortisol in depressed prepubertal children or control subjects showed no response to DMI given in a single oral dose in the morning. It is not clear why such negative effects were found. One possibility is that hypersecretion of cortisol had not occurred in these subjects and thus could not be "corrected," or that DMI may have a different effect on presynaptic uptake mechanisms or adrenergic receptors in prepuberty.

Thyrotropin

In adults with endogenous depression, a number of research groups have reported that the thyroid-stimulating hormone (TSH) response to thyrotropin-releasing hormone (TRH) is blunted (Gold *et al.*, 1981; Kirkegaard, Bjorum, Cohn, Faber, Lauridsen, & Nerup, 1977; Loosen & Prange, 1980). Puig-Antich (1986), however, found no differences in TSH response to TRH between prepubertal depressed children and controls. Similar results have been reported for depressed adolescents (Greenberg *et al.*, 1985). Further research is needed as there is evidence that the TSH response to TRH is influenced by age and steroids (Baumgartner, Hahnenkamp, & Meinhold, 1986; McGrath, Quitkin, Stewart, Asnis, Novacenko, & Puig-Antich, 1984).

Growth Hormone Secretion

Growth hormone secretion may be studied in the waking state, in response to pharmacological challenge, and during sleep (Mendelson, 1982). Research on growth hormone (GH) secretion and recovery in depression has focused on provocative tests of GH secretion during depression as well as studies of sleep-related GH secretion without provocative challenge.

Growth Hormone Response to Provocative Challenge

Approximately half of unipolar endogenously depressed adult hyposecrete GH in response to insulin-induced hypoglycemia (Gregoire, Branman, DeBuck, & Corvilain, 1977; Gruen, Sachar, Altman, & Sassin, 1975). The effect is most pronounced in postmenopausal women, probably because of diminished sex steroids. Attenuated GH response to other pharmacologic agents, for example, desmethylimipramine (Laakman, Hinz, Neulinger, Strauss, and Wittman, 1986), clonidine (Charney, Heninger, Sternberg, Hastad, Giddings, & Landis, 1982; Checkley *et al.*, 1981), and D-amphetamine (Langer, Heinze, Rein, & Matussek, 1976) has also been noted. These agents all increase noradrenergic activity, and the results therefore are consistent with the hypothesis of a functional noradrenergic deficit in depression. Mendelson, Sitaram, Wyatt, Gillin, Jacobs (1978) and Takahashi, Kondo, Yoshimur, and Ochi (1974) found that L-5-hydroxytryptophan (L-5 - HTP), a serotonin precursor, produced significant differences in GH response between depressed adults and controls.

Puig-Antich, Novacenko, Davies *et al.* (1984) found that hyposecretion of GH in response to insulin-induced hypoglycemia occurred in prepubertal endogenously depressed children when compared with nonendogenously depressed or psychiatrically ill, but not depressed controls. In contrast to the above, in an ongoing study of adolescents (Puig-Antich, 1986) preliminary analysis did not reveal a clearly significant difference between endogenously depressed subjects and controls. However, girls secreted significantly more GH than boys, probably indicative of estrogen potentiation of growth hormone response (Frantz & Rabkin, 1965). These results, showing an effect in prepubertal but not adolescent subjects, are consistent with a pubertal effect on GH secretion. The results in conjunction with data showing that GH hyposecretion is most pronounced in postmenopausal subjects (Gruen, Sachar, Altman, and Sassin, 1975) suggest that GH response to ITT reflects the neuroregulatory mechanisms of depression only in prepuberty and following the menopause (Puig-Antich, 1986).

The growth hormone response to either 50 or 75mg desmethylimipramine has also been studied in prepubertal children by Puig-Antich and colleagues (Puig-Antich, 1987). Hyposecretion of GH did not occur and no significant differences in GH secretion, whether measured by maximum increase from baseline or area under the curve, were found between depressed children and controls. These results are again similar to those found in postmenopausal women (Matussek & Laakmann, 1981). In contrast, Ryan *et al.* (1988) found that depressed adolescents given 75 mg i.m. desipramine secreted significantly less growth hormone over the next two hours than did normal adolescents. A substantial proportion of the difference was accounted for by adolescents who were suicidal. Thus, the GH response to ITT and DMI may be similar in depressed prepubertal and postmenopausal subjects, and different from that found in adolescents and premenopausal adults.

Growth Hormone Secretion during Sleep

Growth hormone secretion increases within 80 minutes of sleep onset and occurs primarily in conjunction with delta sleep (Takahashi, Kipais, & Daughaday, 1968). Similarly, in prepubertal children, most GH secreted during sleep is secreted during the first few hours (Finkelstein, Roffwarg, Boyar, Kream, & Hellman, 1972; Mace, Gotlin, & Beck, 1972) and is also secreted in conjunction with delta sleep. Delta sleep and the associated GH release decrease with age (Finkelstein *et al.*, 1972).

Puig-Antich, Goetz, Davies, *et al.* (1984) reported significantly greater GH release during sleep in depressed prepubertal children, regardless of subtype, than in controls. No increase occurred in daytime GH release. In contrast, endogenously depressed adults may hyposecrete GH during sleep, when delta sleep is controlled (Jarrett & Kupfer, personal communication, 1985). Mendlewicz *et al.* (1985) similarly found that hypersecretion of GH did not occur in depressed adults during sleep, but increased daytime GH release was present. In an ongoing study of depressed adolescents, Puig-Antich (unpublished data) finds no significant differences in GH release during delta sleep between depressed adolescents and controls. Thus, the results in depressed adolescents may occupy a position intermediate between the results in prepubertal children and those in adults. It is reasonable, therefore, to hypothesize (Puig-Antich, 1987) that age and puberty interact in controlling GH release during sleep and also daytime GH release.

Growth hormone release during sleep and during the day are probably regulated by different mechanisms (Mendelson, 1982). Hypersecretion of GH during sleep might arise through three possible mechanisms. First, Methysergide, which blocks serotonin receptors, increases sleep growth hormone secretion (Mendelson *et al.*, 1975). Second, sleep growth hormone secretion is inhibited by methoscopolamine, an antimuscarinic agent (Mendelson *et al.*, 1978). Third, piperidine, which is a nicotinic receptor agonist, increases sleep GH secretion (Mendelson, Latigna, Wyatt, Gillin, & Jacobs, 1981). Thus, a functional deficit of hypothalamic serotonin and increased cholinergic activity either through increased acetylcholine release or muscarinic receptor hypersensitivity might be responsible.

Growth Hormone Secretion during Recovery

Of further interest is the finding that in prepubertal children both the hyposecretion of GH in response to insulin and the hypersecretion during sleep persist during recovery when subjects are drug-free and in remission. Puig-Antich, Davies, Novacenko, *et al.* (1984) performed follow-up insulin tolerance tests in 18 medication-free prepubertal children who had shown at least 4 months of sustained recovery from an episode of depression and 16 control subjects with neurotic disorders. Subjects who previously experienced an endogenous depression showed significantly decreased GH release. Moreover, GH response during illness and recovery were significantly correlated. Growth hormone response to insulin-induced hypoglycemia may thus be a biological marker for a previous episode of depression or a trait marker for endogenous depression.

Polysomnography

EEG Sleep during Depression

There is now considerable evidence that EEG sleep abnormalities occur in adults with major depression. A number of studies have shown that in depressed adults there is a decrease in the first REM period latency (Gillin, Duncan, Pettigrew, Frankel, & Snyder, 1979; Kupfer, 1976; Vogel, Vogel, McAbee, & Thurmond, 1980). There is also evidence for decreased slow wave sleep time (Coble, Kupfer, Spiker, Neil, & Shaw, 1980; Kupfer &

Foster, 1979), increased REM density (Coble *et al.*, 1980; Gillin *et al.*, 1979; Vogel *et al.*, 1980), decreased sleep efficiency (Gillin *et al.*, 1979; Kupfer & Foster, 1979), and abnormal temporal distribution of REM throughout the night (Vogel *et al.*, 1980).

Studies of EEG sleep in childhood depression, however, reveal no changes in EEG sleep parameters before puberty, but there is evidence for an effect of age. Puig-Antich, Goetz, Hanlon, Tabrizi, Davies, and Weitzman (1982) studied 54 depressed prepubertal children and controls and found no decrease in slow wave sleep, no decrease in sleep efficiency, no shortening of latency to the first REM episode, no increase in REM density, and no abnormality in the temporal distribution of REM sleep during the night. Similar results were reported by Young, Knowles, MacLean, Boag, and McConville (1982), and Kupfer and Foster (1979). In contrast, Lahmeyer, Poznanski, and Bellur (1983) reported decreased REM latency in 13 depressed late adolescents/early adults (most subjects were over 17 years of age) in comparison to 13 control subjects. Goetz *et al.* (1987), in a preliminary analysis of an ongoing study, found that in depressed adolescent outpatients disturbances in sleep continuity become evident as the depressive group enters adolescence, but that REM latency only becomes abnormal in late adolescence. The overall major sleep variables studied were highly correlated with age in that abnormalities are more likely toward later adolescence. Puig-Antich *et al.* (1982) hypothesize that the negative EEG sleep findings in prepubertal children in comparison to adults reflect not differences in the neurobiology of sleep, but in the neurobiology of the initiation and maintenance of depressive episodes, or in the regulation of the first non-REM period. There is also evidence from studies of depressed and nondepressed adults that age and EEG sleep variables are highly correlated (Coble *et al.*, 1980; Gillin *et al.*, 1979; Ulrich, Shaw, & Kupfer, 1980; Williams, Karacan, & Hursch, 1974). It may thus be hypothesized that the lack of changes in EEG sleep during major depressive episodes in prepubertal children are secondary to maturational factors which modify the neuroregulation of the first non-REM episode, or in the neurobiology of the initiation or maintenance of the depressive episode in predisposed individuals.

EEG Sleep Measures during Recovery

Of further interest are findings that though depressed prepubertal children show no change in EEG sleep measures, changes in the direction of those shown in depressed adults become evident during recovery from depression. Puig-Antich, Goetz, Hanlon, Tabrizi, Davies, and Weitzman (1983) restudied 28 prepubertal endogenous and nonendogenously depressed children who were fully recovered from the depressive episode for at least 4 months and were drug free for at least 1 month. During recovery, significantly decreased first REM latency and a significantly greater number of REM episodes were evident in comparison to the children's own status while they were actively depressed. Moreover, there was no relationship between change in sleep onset and change in REM sleep latency, although depressed children fell asleep somewhat earlier during their recovery phase than when they were ill. Sleep continuity during recovery also improved significantly on most measures compared to the time when the children were clinically depressed. Thus, it appears that the shortened first REM latency period may be a marker of an underlying trait or of a past episode. Comparable data on adolescents who have recovered from an episode of depression are not yet available. Similar studies on recovered drug-free adult depressives provide mixed results. Although there is some evidence that REM latency normalizes in recovered adult depressives (Hauri, Chernik, Hawkins, & Mendels, 1974; Sitaram, Nurnberger, Gershon, & Gillin, 1982; Schulz & Trojan, 1979), others have found that REM period latency tends to remain the same or only show slight improvement (Kupfer & Foster, 1979; Mendels & Chernik, 1975).

Biological Mechanisms

There is considerable evidence that cholinergic systems are important in regulating REM sleep. Physostigmine and archoline decrease REM sleep latency in normal adults and recovered depressives (Sitaram, Nurnberger, Gershon, & Gillin, 1980), and this effect is blocked by scopolamine, a muscarinic receptor blocker (Sitaram, Moore, & Gillin, 1978). Changes in EEG sleep variables similar to those observed in depression can be induced in normals by scopolamine (Sagales, Erill, & Domino, 1975; Sitaram, Moore, & Gillin, 1979). Muscarinic supersensitivity is a likely mediator of these effects. Puig-Antich (1987) advanced the hypothesis that central muscarinic receptor supersensitivity is the common factor underlying the findings or REM sleep latency across age groups. Other systems may also be involved. Alph-

methylparatyrosine, which blocks tyrosine hydroxylase and thus depletes CNS norepinephrine, also decreases REM latency (Sitaram, Gillin, & Bunney, 1984). McCarley (1982) proposed that the regulation of REM sleep is dependent on both cholinegic and aminergic systems based on a review of data from animal studies.

Biological Treatment of Childhood Depression

Children

Frommer (1967) reported the first controlled double-blind study of antidepressant treatment in depressed children. She compared the combination of phenelzine and chlordiazepoxide for 2 weeks to phenobarbital for 2 weeks in 32 children. Although 78% of the children receiving the combination improved, only 50% of those on phenobarbital alone improved. The design of the study, however, is such that one cannot tell if improvement was due to the combination, to phenelzine alone, or to chlordiazepoxide alone. The study is also flawed in that 2 weeks may be insufficient time to allow the full therapeutic effects of phenelzine, or to rule out carry-over effects of the medications.

Puig-Antich, Perel, Lupatkin, *et al.* (1987) studied 38 prepubertal children meeting RDC criteria for depression and diagnosed by two independent interviews given 2 weeks apart. In a 5-week double-blind placebo-controlled study, subjects were given imipramine on dosages which ranged to 5 mg/kg/day or placebo. Sixteen subjects were given medication, and 22 were given placebo. Fifty-six percent of subjects given imipramine and 68% of the placebo group responded. Kashani, Shekim, and Reid (1984) in a randomized double-blind crossover design compared amitriptyline and placebo in nine depressed children. After 3 days of medication or placebo at 1 mg/kg/day, subjects were maintained on a fixed schedule of 1.5 mg/kg/day. The daily dose ranged from 45 mg to 1.0 mg/day. Subjects were given placebo or amitriptyline for 4 weeks and were then given the other for a further 4 weeks. Overall, six children responded favorably to amitriptyline. Improvement also occurred in the placebo condition, and the difference between the two approached but did not reach statistical significance.

The relationship between plasma levels of medication and clinical response is an important issue that has been addressed in three studies. Preskorn, Weller, and Weller (1982) examined plasma level of imipramine and clinical response in 20 depressed prepubertal children over a 6-week study period. During the first 3 weeks of study, subjects were given 75 mg/day of imipramine. At the end of the 3 weeks, four subjects had responded. All of the responding children had plasma levels between 125 and 225 ng/ml. Nonresponders had plasma levels outside this range. Imipramine dosage was then adjusted. At 6 weeks, 11 of 12 children with plasma levels between 125 to 225 ng/ml had responded. Of the four other children who completed the protocol, one responded and had a plasma level outside this range. The authors concluded that there is a curvilinear relationship between clinical response and plasma level of imipramine. However, the proposed relationship is based on only four cases at 3 weeks, and two cases at 6 weeks in the upper plasma range. A curvilinear relationship has been suggested also for plasma levels of nortriptyline and clinical response in a small sample of depressed children (Geller, Perel, Knitter, Lycak, & Farook, 1983). In adults, a curvilinear relationship has been shown for nortriptyline (Asberg, Cronholm, Sjoquist, & Tuck, 1971; Kragh-Sorensen, Hansen, Baastrup, & Hvidberg, 1976), but not imipramine (Reisby *et al.*, 1977).

Puig-Antich, Perel, Lupatkin *et al.* (1987) also studied the relationship between plasma level and clinical response in 30 depressed prepubertal children over a 5-week period. The sample in this study overlapped with the sample described above. Maintenance plasma levels of imipramine plus desipramine were significantly higher in subjects who responded than in those who did not respond. Less severely depressed subjects responded more than more severely depressed individuals. Subjects with a psychotic depression also showed poor response. Almost all subjects with a plasma level greater than 155 mg/ml responded. However, subjects had a lower response rate than placebo, although the difference was not significant. In contrast to the report of Preskorn *et al.* (1982), there was no evidence of a curvilinear relationship between imipramine and desipramine plasma level and clinical response.

Adolescents

There are two studies of antidepressant use in adolescents. Kramer and Feiguine (1983) reported that placebo was equally effective as amitriptyline (200 mg/day) in a double-blind, 6-week, placebo-controlled study in depressed adolescent inpatients.

However, only 10 subjects were included in each group thus precluding definitive conclusions. Ryan, Puig-Antich, Cooper *et al.* (1986) studied the relationship between imipramine and desipramine maintenance plasma level and clinical response at 6 weeks in 34 depressed adolescents. Overall, only 44% of subjects responded. Thirty-three percent of those with endogenous depression responded. There was no relationship between clinical response and plasma level of medication. Ryan, Puig-Antich, Rabinovitch, Fried, Ambrosini, and Meyer (1988) also reported an open clinical trial of monamine-oxidase inhibitor therapy in 23 adolescents, all but two of whom had previously failed to respond to a tricyclic antidepressant. Seventy-four percent showed a good or fair response.

Puig-Antich (1987) has attempted to put the above findings in a developmental perspective. Puig-Antich reviewed studies of imipramine effectiveness in adult depression and noted that adults with endogenous depression who responded tended to be older than 45 years of age, whereas responders with nonendogenous or atypical depression tended to be younger than 45 years of age. However, it was not clear from the studies reviewed if age or depressive subtype was the most important factor influencing response. Similarly, a clear positive relationship between plasma level and clinical response was found primarily in older subjects with endogenous depression. Integrating these data with the above results in prepubertal and adolescent subjects, Puig-Antich (1989) hypothesized a U-shaped function with a strong relationship between imipramine and desipramine plasma level and clinical response in prepuberty, and during and past middle age, and a weak relationship during adolescence and young adulthood. Puig-Antich further hypothesized that negative effects of sex hormones, after puberty and during young adulthood, on the antidepressant effects of imipramine might account for these findings. Estrogens may inhibit monoamineoxidase (MAO) activity (Klaiber, Kobayashi, Broverman, & Hall, 1971; Klaiber, Broverman, Voga, Kobayashi, & Moriarty, 1972), or methylation of norepinephrine by catechol-o-mothyltrensterase (COMT) (Ball, Knuppen, Haupt, & Breuer, 1972), thus increasing central adrenergic function. Bhavsar, Dumal, and Kelkar (1983) have demonstrated that estradiol reversed the effects of imipramine but not those of electro convulsive therapy on norepinephrine-mediated behavior in female rats but did not effect serotonin-mediated behaviors induced by either treatment. Moreover, decreases in 5-HT_2 receptor binding in rat cerebral cortex was abolished by bilateral ovariectomy, but ovariectomy did not change other effects of imipramine on 5-HT content or adrenergic receptor binding. Replacement of estradiol, progesterone, or both reversed the effects of bilateral ovariectomy. Estrogens may therefore be important mediating variables for the antidepressant effects of IMI.

Conclusion

Considerable strides have been made in the past decades in exploring biological parameters of childhood depression. The research reviewed in the present chapter shows that age and puberty are important influences. Further exploration of the potential biological markers that were reviewed will substantially aid in high-risk research, treatment studies, and ultimately in elucidating the biological underpinning of depression.

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CHAPTER 26

Cognitive Components of Depression

Lynn P. Rehm and Alice S. Carter

Current thinking regarding childhood psychopathology stresses the necessity of viewing psychopathology from a developmental perspective. Cicchetti and Schneider-Rosen (1984), for example, argue that psychopathology in children should be looked at in terms of failure to negotiate important tasks of social-cognitive competency in the sequence of childhood development. Stage-relevant task mastery is seen as the mechanism by which children progress to new levels of cognitive organization and differentiation. Cognitive reorganization is seen as a process whereby prior levels of organization are subsumed under new hierachies of cognitive structure. As such, failure to negotiate one developmental task has relevance for mastery of subsequent stages and thus implications for later social-cognitive competence on into adulthood.

Modern conceptions of childhood psychopathology thus warn that it may be dangerous simply to translate downward theoretical models that were developed to explain adult forms of psychopathology. These models may not be applicable or may apply in different ways to children at different stages of development. Despite this caution, it is also important to recognize that new models of adult disorder may have heuristic value in elucidating psychopathology in children. Despite controversy regarding children's ability to become clinically depressed because of developmental limitations in cognitive capacity (Lefkowitz & Burton, 1978; Rie, 1966; Rochlin, 1959), the majority of research on childhood depression has emphasized adult cognitive models. This body of research has been criticized for adopting "downward" extensions of adult models with disregard for the developmental considerations (Malmquist, 1977). In this chapter, we will take the position that cognitive models of adult depression can be applied usefully to understanding depression in children, but that they should be examined and evaluated with attention to what is known about the normal development of relevant cognitive factors. Thus, for example, though comparisons of symptoms and correlates of childhood depression with adult depression may provide useful information, the essential comparisons must be between depressed and nondepressed children.

The purpose of this chapter is to review some of the recent findings in the literature on normal and depressed children's cognitive processes in an effort to improve our understanding of the cognitive component of childhood depression. As the developing child's cognitive structures undergo qualitative changes, these changes will influence how the child construes and organizes the world. This, in turn, should influence both the child's affective experience and its expression. This is not to ignore the

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important contribution of biological, environmental, and interpersonal influences to cause, characteristics, maintenance, and recovery in depression. Rather, interpersonal, environmental, and biological constraints are viewed as interacting with cognitive processes and as providing the context within which cognitive processes develop.

Although traditionally depression has been viewed as an affective disorder, currently the predominant models of adult depression have emphasized cognitive variables. Three models in particular will be emphasized in our review: Beck's cognitive model (Beck, Rush, Shaw, & Emery, 1979), Seligman's learned helplessness model (Abramson, Seligman, & Teasdale, 1978), and Rehm's self-management model (Rehm, 1977). Beck's cognitive model posits the existence of schemas through which individuals filter and interpret their experience. Depression-prone individuals may develop distorting, negative schemas in childhood that later may coexist with more positive and realistic schemas. When activated, the depressive schemas distort experiences of the self, the future, and the world in a negative direction. Seligman's model posits a form of depression in which individuals perceive themselves helpless to prevent negative events. This form of depression occurs when the individual makes an overgeneralized interpretation of the uncontrollability of events following an important negative event that is perceived as uncontrollable. A contributory cause to the likelihood of drawing such a conclusion is a depressive attributional style. Borrowing from attribution theory in social psychology, a depressive attributional style is a traitlike tendency to attribute negative events to causes that are internal (i.e., due to the individual), stable (i.e., generalizable over time), and global (i.e., generalizable over domains of events). Conversely, the depressive style involves the tendency to attribute positive events to external, unstable, and specific causes. The style is a vulnerability factor for helpless depressions. Rehm's self-management model was originally an attempt to subsume factors identified by other models of depression under a more comprehensive theoretical framework. The framework employed was Kanfer's (1970, 1971) theoretical model of self-control behavior. The self-management model of depression posits six specific deficits in the way in which individuals manage their efforts toward long-term goals: (1) a tendency to attend to negative events in personal experience to the exclusion of positive events, (2) a tendency to attend to the immediate as opposed to the delayed consequences of personal behavior, (3) a tendency to set stringent standards for personal behavior, (4) a tendency to make depressive attributions of the causes of personal experience, (5) a consequent tendency to self-administer insufficient contingent reward to sustain efforts toward long-term goals, and (6) a consequent tendency to self-administer excessive self-punishment.

Each of these models has contributed to depression research in adults and, as a consequence, each has undergone revisions and elaborations. Many of the cognitive phenomena of depression are dealt with in different but overlapping ways by these models. Rehm (1988) has argued that these phenomena can be viewed collectively as ways in which individuals process their experience to draw inferences and judgments, which are then used as a basis for decisions regarding future actions. Following this logic, we will review the childhood depression literature with regard to inference processes relevant to self-evaluation, personal causation, future expectations, and distortion of present or past events.

Self-Evaluation

Negative self-esteem is held to be a central factor in depression by a number of theorists. For example, Beck (1967) presented a negative view of self as one of the essential features of depression. In Rehm's (1977) self-management model of depression, negative self-evaluation is presented as one of the several critical factors in depression. The self-management model suggests that negative self-evaluation in adults is based on stringent standard setting as well as being influenced by negative self-monitoring and a depressive attributional style. In the study of adults, it has become evident that self-evaluation is a multidimensional construct (Rehm, 1988). Performance expectancies, comparisons to others, task importance, standard setting, and generalization are all relevant variables.

Self-evaluations become more differentiated and less global with age (McCandless & Evans, 1973; Mullener & Laird, 1971). Young children are likely to think of themselves in absolute unqualified terms, such as "I am good" or "I am bad." Secord and Peevers (1974) observed several transitions that occur in children at around age 7. Prior to approximately age 7, children tend to evaluate themselves in absolute terms, that is, in terms of some internal standard rather than in relation to what others do. After age 7, however, children begin to evaluate

their own competence by comparison with others. They also begin to shift from thinking of themselves in terms of habitual actions, that is, what they are wanting to do at various times and places to thinking in terms of "action competence," or what their actual abilities are. Cicchetti and Schneider-Rosen (1984) noted that the transition from physicalistic to a psychological conception of the self is relevant to the dimension of stable versus unstable attribution. If the child prior to age 8 conceives of the self principally in terms of actions or such physical factors as possessions, then any attribution of noncontingency to features of the self would be unstable, since these physicalistic factors are usually transient.

Several empirical investigations have documented a major change in self-understanding that occurs at about 8 years of age, which most likely reflects when the child enters concrete operational thinking (Harter, 1985; Ruble & Rholes, 1981; Suls & Sanders, 1982). Prior to this time, the very concept of "personness" as a generalization about the self is not yet firmly established (Harter, 1985). As a result, young children probably cannot make inferential judgments of global self-worth, although they can evaluate their performances in particular domains of their lives. Harter (1985) reported that items tapping specific abilities, such as doing well in sports or school work, have meaning to children below age 8, whereas items describing the degree to which one likes oneself as a person or is happy being the way one is do not have meaning for the young child. With the emergence of concrete operational thinking, children begin to develop the capacity to reflect on their own cognitive processes, that is, to engage in metacognition.

The capacity for engaging in negative evaluation of the self occurs at the age at which McConville, Boag, and Purohit (1973) found a transition from "affectual" depression to depression characterized by negative self-esteem.

A number of investigators have now addressed the developmental emergence of social comparison processes and how these impact self-description (Masters, 1971; Ruble & Rholes, 1981; Suls & Sanders, 1982). Within the domains of academic and athletic competence, pupils during the middle elementary grades begin to use social comparison as a basis for making judgments that have implications for the self. Prior to that age, children have the ability to compare themselves to others; however, these comparisons are not employed for the purpose of self-evaluation (Masters, 1971; Ruble, Boggiano, Feldman, & Loebl, 1980; Suls & Sanders, 1982). Glasberg and Aboud (1981) asked 5- and 7-year-old subjects to give liking ratings to hypothetical children who had undergone a painful life event and to give evaluative ratings of themselves in the same situations. Older children evaluated a hypothetical other and themselves negatively, whereas the 5-year-olds could not extend their negative evaluation of a sad other to themselves.

The majority of studies of adult depression suggest accurate and equivalent expectancy or prediction of performance by depressed adults. Furthermore, results are fairly consistent in suggesting that it is higher standard setting by depressed adults that leads to more negative evaluations of themselves but not of others, even when others demonstrate equivalent performance (Rehm, 1988). Studies of adult depressives focus on level of aspiration on a task (Golin & Terrell, 1977; Golin, Terrell, & Johnson, 1977) or level which would yield satisfaction (Mercatoris, Cole, Lewis, & Leonard, 1980). In her work with normal children, Harter (1986) focused on importance of success or relevance. If it is important that one succeed in a given domain (i.e., that domain is relevant), success should lead to high self-esteem. If the dimension is irrelevant, negative judgments of one's performance will not threaten one's sense of self-esteem in terms of one's global self-worth.

Harter and Mayberry (1984) asked fifth and sixth graders to complete separate ratings of competence and importance in five domains of performance (scholastic competence, athletic competence, social acceptance, physical appearance, and behavior or conduct) and an additional rating of global self-worth. The discrepancy score between children's competence/adequacy judgment and the importance of success ratings was correlated with self-worth. They inferred that children were weighing their competence in relation to the importance of success in these domains, and that the outcome of this evaluative procedure provided the basis for their overall feelings of worth. These findings were replicated in a second sample of 30 high, 30 medium, and 30 low self-worth pupils in the fifth and sixth grades. In addition, the higher the self-worth, the greater was the congruence between importance scores and competence scores. It appeared that children in the three self-worth groups differed in their ability to discount the importance of areas they did not feel competent in. Using vignettes, Harter and her colleagues asked sixth graders to make judgments about whether or not a given story character would discount or continue to maintain the importance of an activity for the subject's lowest compe-

tence domain. There was a clear relationship between the percentage of discounting responses and children's level of self-worth. They found that 80% of the high self-worth subjects indicated the story character would decide this domain was no longer important, whereas 55% of the low self-worth subjects felt the story character would discount the importance of a domain in which his or her incompetence had been demonstrated. It must be noted that the low self-worth students were, in fact, among the least competent pupils. Furthermore, due to the fact that the domains under study are highly valued by society, it may be impossible for these pupils to employ discounting.

Comparing children's judgments of competence with the teacher's evaluation revealed that the self-judgments of the high self-worth group are higher than the teacher's ratings; that is, the group tends to see itself as slightly more competent than did the teachers in both highest and lowest domains. In contrast, the low self-worth group showed no tendency to inflate their feelings of competence and, in fact, for their least competent domain, their judgments were somewhat lower than the teachers' judgments.

Several studies have found some evidence that academic and social incompetence are associated with children's depression (Asher, Hymel, & Renshaw, 1984; Kaslow, Rehm, & Siegel, 1984; Lefkowitz & Tesiny, 1980; Seagull & Weinshank, 1984; Strauss, Forehand, Frame, & Smith, 1984; Tesiny, Lefkowitz & Gordon, 1980) and that children who are both academically and socially incompetent have the highest levels of peer-nominated depression (Blechman, McEnroe, Carella, & Audette, 1986). Causal relationships between depression and social and academic competence have not been studied. Thus, depression may cause incompetence, incompetence may cause depression, or depression and incompetence may reflect some other effect. Skills deficits have been associated with a variety of severe behavior problems (Blechman, Tinsley, Carella, & McEnroe, 1985). Thus, skills deficits in academic and social functioning may be more cautiously viewed as risk factors for psychopathology, with little implication for specificity to depression.

Harter and her colleagues also looked at children whom teachers identified as manifesting the affective and motivational symptoms of depression, that is, sadness or depressed mood, despondency, and/or lack of energy and interest in age-appropriate activities. Self-worth scores among the 44 children identified in this manner were extremely low, and scores were low in at least two of the three specific competency domains that were tapped (cognitive, social, and physical). These children reported that success in their lowest competence domain was just as important as success in their highest domain. Interestingly, when children's scores were examined in terms of congruence with teacher ratings, there was considerable agreement, suggesting that these children were quite realistic in appraisals of their strengths and weaknesses. Thus, these children who were identified as depressed on the basis of teacher ratings of affect and motivation are less competent than their peers, accurately evaluate their strengths and weaknesses, and continue to aspire for success in areas of weakness. It is possible that these children have unrealistic standards for their success given their limited competence and that, similar to depressed adults, this discrepancy leads to a sense of failure, which promotes depressed mood.

Studies from our research program at the University of Houston also illustrate some additional points with regard to self-evaluation and depression in children. Kaslow, Rehm, and Siegel (1984) examined correlates of depression, as measured by the Children's Depression Inventory, in samples of first, fourth, and eighth graders. A global measure of self-esteem, the Coopersmith Self-Esteem Inventory (Coopersmith, 1967), correlated .72 with depression scores. A finer analysis of self-evaluation was obtained by asking children a series of questions before and after a design-copying task. Depressed children anticipated poorer performance on the task and said they would take longer and get fewer correct than did nondepressed children. Although there were no differences in standard setting for a good score, depressed children set more stringent standards for what they perceived to be a poor score. Similarly, depressed children did not differ in self-administered reward but were more likely to self-administer punishment, and they endorsed the use of punishment as a motivator more frequently. Following the task, depressed children evaluated their performance as poorer on a number of dimensions despite an absence of any objective difference. The emphasis on punishment over reward is a consistent finding in our work (cf. Cole & Rehm, 1986).

We have also been interested in the relationship between self-evaluation and parental evaluation. Depressed children in the study above perceived their mothers as setting similarly stringent standards for them with regard to failure. Cole and Rehm (1986) found that mothers (but not fathers) of

depressed children in a guidance clinic sample set more stringent standards for their children's performance. This difference was corroborated by actual observations of the interaction of the child and both parents as the child played a game. Mothers of depressed clinic children were less likely to make positive comments about their child's performance than were mothers of nonclinic children. Mothers of nondepressed clinic children fell in between. Fathers of depressed children showed a similar effect at lower levels of performance. There were no differences between parent groups on negative comments.

In general, efforts to assess self-esteem in children have not attended to the dimensional complexity and changing nature of children's abilities to self-evaluate at different stages of development. As an example, Leventon (1983), using the Kaslow, Rehm, and Siegel (1984) data, noted that high scores on the Children's Depression Inventory reflected different item patterns at different grade levels. Depressed first graders were more likely to evaluate themselves as bad, whereas depressed eighth graders endorsed sadness items more frequently. Improved assessment instruments would match question types to the cognitive level of the child.

Personal Causation

Four dimensions of perceived causality have been studied in children and have been related to depression. A primary dimension is the distinction between *internal* and *external* causation. This differentiation distinguishes between causes located within the person—intelligence, skill, aptitude, effort, and personality—and causes located outside the person—difficulty of the task, skill of one's opponent, help from others, and luck. Rotter (1966) viewed the tendency to attribute causes to internal or external factors to be a generalized expectancy and thus a consistent difference among individuals. A shortcoming of this one-dimensional taxonomy became evident when it was observed that causes classified on the same dimension elicited disparate responses. For example, in achievement-related contexts, expectations of success are lower when failure is perceived as resulting from a lack of ability than when failure is believed to result from a lack of effort (e.g., see Weiner, Nierenberg, & Goldstein, 1976). Labeled causal *stability* (Heider, 1958; Weiner, 1980), this dimension differentiates causes on the basis of their relative endurance. A third dimension of causality, *controllability,* was proposed when it became evident that some causes identically classified on both locus and stability dimensions yielded dissimilar reactions (e.g., Weiner, 1979). Thus, when adults assume that the actor who failed "could have done otherwise" (Hamilton, 1980), greater punishment is elicited. Attributions can also be classified as *global* versus *specific,* depending upon the breadth or generality of the inferred cause.

The reformulated learned helplessness model of depression (Abramson *et al.,* 1978) states that the nature of the attributions a person makes about a major aversive event will determine whether or not a depression is produced. The dimensions of internality, stability, and globality are employed by this model. Furthermore, the specific dimensions of attribution will determine the characteristics of the depression. An internal attribution implies self-blame and thus depressed affect. An attribution to a global cause will yield a generalized depression influencing all areas of the person's life, and a stable attribution will yield enduring depression. The theory further postulates that individuals have consistent attributional styles and that a person may be prone to depression because of a particular attributional style, comprised of making attributions for negative events to internal, stable, and global causes and making attributions for positive events to external, unstable, and specific causes. The assertion that attributional styles influence the characteristics of depression has important implications for the development of depression in children. Seligman (1975) speculated that children's generalized sense of mastery is formulated at a very early age and that failure to acquire this characteristic would lead to helplessness and depression proneness.

Early studies of the internal-external dimension found that externality was associated with high depression and internality was associated with low depression (Lefkowitz, Tesiny, & Gordon, 1980; Moyal, 1977). Leon, Kendall, and Garber (1980) surveyed parents of 138 third through sixth graders and, based on parental reports of depressive symptoms, formed depressed and nondepressed groups of 21 children. Depressed children attributed positive events to external causes significantly more than did nondepressed children. There were no differences between depressed and nondepressed groups on measures of attributions for negative events. However, when children's self-reports of depressive symptoms were analyzed, children who scored high on a measure of severity

of depressive symptoms attributed negative events to internal factors significantly more than did children who scored low on depressive symptomatology.

Dweck and Repucci (1973) identified a group of children who did not perform a response required to succeed even though they were motivated and fully capable of doing so. These children had poor academic performance, were more likely to give up in the face of failure, took less personal responsibility for their successes and failures, and attributed negative outcomes to stable personal qualities, such as ability (versus effort) when compared to more persevering, mastery-oriented children. Dweck (1975) trained these children to attribute failure to effort and demonstrated that such training tended to improve later performance on a series of arithmetic tasks.

A number of studies have found that the helpless attributional style correlates with depression in normal and mildly depressed samples (Blumberg & Izard, 1985; Craighead, Smucker, & Duchnowski, 1980; Friedlauder, Philips, Morrison, & Traylor, 1981; Kaslow, Rehm, & Siegel, 1984; Leon, Kendall, & Garber, 1980; Seligman, Peterson, Kaslow, Tanenbaum, Alloy, & Abramson, 1984). Studies with clinical samples have yielded less consistent results. Kaslow, Rehm, Pollack, and Siegel (1987) used the Children's Attributional Style Questionnaire (Seligman *et al.*, 1984) to derive a total helplessness score across internal, stable, and global dimensions. Depressed clinic children score more helpless than either nondepressed clinic or normal children. In contrast to Seligman *et al.* (1984), this study did not find a relationship between the child's attributional style and the attributional style of either parent.

McCauley, Burke, Mitchell, and Moss (1986) studied cognitive attributions of depression in children (ages 7 to 17) from the psychiatric services of a children's hospital. Forty-four met criteria for Major Depression, 24 had an episode of depression within the prior 12 months but were not depressed at the time of the evaluation, and 24 had other psychiatric disorders. Although attributional styles have been conceptualized to be a risk factor for subsequent depressive episodes, in the present study, the nondepressed and depressed-resolved groups had comparable scores on the attributional style questionnaire for children. When the nondepressed groups were collapsed into one and then compared to the depressed group, there was a significant difference between the groups with the depressed group showing a more depressive attributional style. The depressed group indicated a greater tendency to attribute success to external, unstable, specific factors. The groups did not differ in responses to failure situations. In order to investigate developmental changes in cognitive attributions, the sample was divided into pre- and postadolescence using 12 years as the cutoff. There was a cumulative trend for adolescents to endorse responses reflecting lower self-esteem, more hopelessness, and a more depressive attributional style than preadolescents, but a more internal sense of control than the younger group.

Weiner and Graham (1984) proposed an attributional approach to the study of affective development that is guided by the belief that causal thoughts precede or change the experience and understanding of emotions, such as pride, pity, guilt, and anger. Consistent with the principle of orthogenesis in broader areas of child development, emotions are generally believed to proceed from a state of general excitement, the undifferentiated emotion present at birth, to one of more differentiated feelings (Bridges, 1930; Izard, 1977). Weiner and Graham (1984) speculated that outcome dependent affects, in addition to their reported high intensity, may be of relatively short duration whereas internal, dimension-linked affects may have great longevity and thus be important in life adjustment. This speculation is consistent with claims that young children cannot maintain negative affective states because of their limited cognitive capacities.

Weiner, Graham, Stern, and Lawson (1982) found that 5-year-olds believe that the communication of anger from a teacher toward a failing pupil indicates the pupil is not trying, in contrast to being low in ability. In a similar manner, two studies have demonstrated that attribution-affect linkages between, for example, insufficient effort-anger and illness-sympathy are exhibited by children as young as 6 years of age (Graham, Doubleday, & Guarino, 1984; Weiner *et al.*, 1982). For this reason, the understanding that internal reasons for rejection influence self-esteem, and that controllable causes for a broken social contract generate anger, may also be manifested by the age of 6 (Weiner & Handel, 1985). As Weiner and Handel (1985) pointed out, these investigations into attribution–emotion relations among younger children stand in contrast to other developmental findings in the attribution area that focus on inferences about the causes of success and failure, particularly ability and effort causes. The latter investigators have concluded that children do not fully distinguish "cannot" (lack of ability) as causes of achievement failure prior to age

9. Yet investigations in the area of attribution–emotion relations generally find that children as young as 5 can distinguish the underlying property of controllability.

Time Concepts

Children cannot truly conceive of temporal order (i.e., the succession of events) and temporal duration (the length of interval between events) until concrete operational thinking is well established, usually between 8 to 9 years of age (Friedman, 1978; Wessman & Gorman, 1977). Children's conception of time continues to develop, as school-aged children have a "shorter" future orientation than teenagers or adults (Doob, 1971). Only at the stage of formal operational thinking (as defined by Piaget), which usually occurs in adolescence, does the ability to assess probabilities emerge. Children's ability to conceptualize time is important in terms of their ability to become hopeless, or to have negative expectations about the future, which is considered to be a central feature of depression and suicidality (Beck, 1976; Melges & Bowlby, 1969). Harris and his colleagues focussed specifically on children's insight into the time course of emotions. Harris (1983) and Taylor and Harris (1983) demonstrated that younger (ages 6–7) and older (ages 10–11) children appear to appreciate that an initial emotional reaction will vary in intensity depending on the nature of the precipitating situation. They also appreciate that intense reactions persist well beyond the precipitating situation, and that such reactions typically wane gradually over time. Harris, Guz, Lipian, and Man-Shu (1985) found that 6-year-olds consistently judged that an initially strong emotional reaction wanes gradually over time for story episodes and for negative experiences recalled from their own lives. Results for 4-year-olds were less consistent, but when adequately trained in the use of the rating scale, they, too, produced a waning pattern of judgment for various types of episodes. Furthermore, 4- and 6-year-olds judged that the story character would be happier the following day if she or he thought about the earlier positive episode rather than forgot it. In addition, 6-year-olds but not 4-year-olds, judged that the story character would be sadder the next day if she or he thought about the earlier negative episode rather than forgot it. This finding suggests that young children appreciate the impact of mental processes on emotion. They did not see current emotion as a simple function of the current external situation. Insight into the time course of emotions is important for diagnosing the causes of emotion in both self and others and is also important in recognizing that children as young as age 4 have expectancies for duration of affective states. Although children may not experience hopelessness in a qualitatively similar manner to adults, hopelessness may appear in the form of a generalized negative expectancy about the duration of a negative affective state in oneself or others. Depressed and nondepressed children could be compared in terms of their expectation for immediate affective reactions and reactions at succeeding points in time.

Despite many authors' challenging children's capacity to become hopeless (Bemporad & Wilson, 1978; Rie, 1966; Siomopoulos & Inamdar, 1979), Kazdin and his colleagues developed a self-report measure of hopelessness, the Hopelessness Scale for Children (Kazdin, Rogers, & Colbus, 1986), which they have validated for use with children ages 6 to 13. The hopelessness scale correlated positively with depression and negatively with self-esteem and social behavior. These findings closely parallel similar work with adults.

Distortion of Present and Past Events

Two questionnaires have been developed to assess cognitive bias in children. Beck (1967) argued that cognitive distortion, or the tendency to misconstrue or distort significant life events in a negative way, plays a central role in the development and maintenance of depression. The Cognitive Bias Questionnaire for Children (CBQC) (Haley, Fine, Marriage, Moretti, & Freeman, 1985) consists of 10 brief vignettes describing a school, home, or social situation familiar to children and adolescents. Each vignette is followed by four response options that exemplify one of the following categories: depressed-distorted, depressed-nondistorted, nondepressed-nondistorted, or nondepressed-distorted. Within a psychiatrically disturbed sample of 39 children and adolescents (ages 8–16), the depressed-distorted scale from the CBQC was significantly correlated with psychiatric and self-reported ratings of depression and could significantly discriminate affective from nonaffective disorders.

A self-report questionnaire, the Children's Negative Cognitive Error Questionnaire (CNCEQ), was developed to measure in children four types of negative cognitive errors derived from Beck's cognitive theory of adult depression: (1) overgeneraliz-

ing predictions of negative outcomes, (2) catastrophizing the consequences of negative events, (3) incorrectly taking personal responsibility for negative outcomes, and (4) selectively attending to negative features of an event (Leitenberg, Yost, & Carroll-Wilson, 1986). In a study of children in fifth through eighth grades, comparing children scoring above 17 (depressed group) to those scoring below 2 (nondepressed group) on the Children's Depression Inventory (CDI) (Kovaks, 1981), Leitenberg and his colleagues found a significant main effect for depression on the CNCEQ, indicating that depressed children were found to endorse each type of negative cognitive error in each content area significantly more often than did nondepressed children. In addition, the depressed group scored highest on overgeneralizing, whereas the nondepressed group, as in the normative sample, scored highest in selective abstraction. However, this pattern was not unique to depressed children. In subsequent studies, low self-esteem and high-evaluation anxiety children endorsed the same negative cognitive errors. This is not surprising given that in school-aged children low self-esteem, depression, and evaluation anxiety are to some extent correlated with each other (cf. Hammen & Zupan, 1984; Moyal, 1977).

Complaints of memory problems are a common symptom of adult depression. Interestingly, this symptom is not typically discussed in the childhood depression literature. One strategy in exploring memory and depression in children has been to investigate cognitive self-structure as a biasing effect in memory. The basic argument here is that there exists in the structure of memory a cluster or schema made up of organized information about oneself. This schema can then be employed for encoding new information. Information that is "about me" or "true of me" can be organized with the aid of the existing self-structure that resides in long-term memory. It is hypothesized that in depressed persons, this structure contains a great deal of negative content. Therefore, depressed persons should show deficits in memory when personally relevant information to be encoded is positive but should show enhancement when information to be encoded is negative. Hammen and Zupan (1984) studied 7- to 12-year-old children's ability to use the self as a schema representing a relatively deep or elaborated encoding principle. They also compared depressed and nondepressed children to determine whether or not nondepressed children showed enhanced recall for adjectives judged to be self-referent and negative in content. All children showed greater recall as a function of encoding with respect to the self, and this capacity appears to improve between ages 8 through 12. As predicted, the nondepressed children showed greater recall for yes-rated, self-reference, positive-content words and no depth of processing effect for the negative-content words. Contrary to prediction, however, depressed children did not show any content-specific self-schema effects. Depressed children's recall was approximately equal for negative and positive content self-referent words, whereas nondepressed children displayed clear superiority of recall for positive content. Hammen and Zupan (1984) proposed a developmental approach in which depressed individuals must have a sufficient depressive history to establish a consistent negative-content self-schema. Assessing the duration and history of depressive episodes would be necessary to explore this hypothesis. Alternatively, the normal children in this sample, though describing themselves in more negative terms than did their peers, may not have been depressed enough to show the content-specific recall effects. Replication in a clinical sample would be necessary to explore this alternative.

Concluding Comments

In this chapter, we have attempted to examine in children several cognitive constructs that have been identified in the adult literature as theoretically or empirically relevant to the development of depression. We have tried to review the available research on the depression construct in children and relate it to relevant work in the normal developmental literature. It should be clear that there is much to be done before these areas of research can be thoroughly integrated. Childhood depression research is a fairly new field, and developmental issues are influencing it only in some respects.

As in any new field in psychology, assessment of the construct of depression is one of the first problems addressed. There has been a proliferation of new instruments for assessing depression in children (cf. Kaslow & Rehm, 1985; Rehm, Gordon-Leventon, & Ivens, 1987). Most of these are direct downward translations of adult instruments and are used for children from the primary grades to the teenage levels. Developmental considerations would suggest that different scales should be constructed for children of different stages of development. Such scales could then match the cognitive capacities of the child and reflect cognitive symptoms relevant to the stage.

Some research is moving in this direction. One strategy has been to look at age differences in response patterns on these standard instruments (e.g., Garber, 1984; Leventon, 1983). Another strategy has been to group children by developmental stage and to examine symptom patterns within groups (Kovacs & Paulauskas, 1984).

Research on the etiology of depression in children is just beginning. It is well established that children of depressed mothers are at risk for childhood depression. Hammen and her colleagues (Hammen *et al.*, 1986) have begun a longitudinal study of the children of depressed and control mothers that is assessing stress, quality of mother–child relationships, and the acquisition of depressive cognitive constructs (e.g., self-esteem, efficacy, and causal attributions for negative events) in an attempt to track the course of the development of depression in children. Zahn-Waxler and her colleagues (Zahn-Waxler, Cummings, Iannotti, & Radke-Yarrow, 1984) have reported on research following depressed parents and their children from 1 year of age. The study is exemplary in the use of direct observational assessment of the children's mastery of social and cognitive developmental tasks. They find, for example, that children of bipolar depressed parents at 1 year are less securely attached, and show poorer problem-solving and role-taking abilities. The adult theories of depression suggest that cognitive components are vulnerability or risk factors predisposing the individual to depression in the face of stress. Understanding the development of these cognitive components in children will have implications not only for the etiology of depression in children, but also for the vulnerability of those children for later depression as adults.

Treatments and prevention of depression in children may also focus on correcting cognitive development. Few studies of therapy with depressed children *per se* have been reported (Kaslow & Rehm, 1983, 1985), although there are studies in the child therapy literature that have targeted cognitive change relevant to depression, as, for example, Dweck's (1975) efforts at alleviating learned helplessness in children. Stark, Kaslow, and Reynolds (1985) reported on a group design study of the treatment of depression in 9- to 12-year-olds. Adaptations of adult therapy programs, the Rehm's self-management therapy program, and a behavior therapy program were effective in comparison to a waiting list control. The self-management program, in particular, targets cognitive self-control skills. The field of childhood depression research is developing rapidly, and much of the research does attend to cognitive components and developmental issues.

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PART V

Specific and Pervasive Disorders

CHAPTER 27

Borderline Disorders in Childhood

Dante Cicchetti and Kurt Olsen

Introduction

Since the mid-1950s, clinicians, theorists, and, more recently, empirical researchers have been involved in an ongoing process of differentiating, defining, and understanding the borderline disorders of childhood. The major emphasis of differentiation and definition has been the clinical explication of the differences among borderline children and psychotic or severely neurotic or acting-out children. Only recently have researchers turned toward the creation of empirical, valid diagnostic criteria and research instruments (Bemporad, Smith, Hanson, & Cicchetti, 1982; Bentivegna, Ward, & Bentivegna, 1985; Greenman, Gunderson, Cane, & Saltzman, 1986; Vela, Gottlieb, & Gottlieb, 1983). Understanding borderline conditions in children has been aided by the various etiological conceptualizations from ego-psychology and psychoanalytic theory. The current view seems to be based on the assumption that children with borderline disorders are on a continuum between neurosis and psychosis, with some characteristics of both less and more severely disturbed children manifested at different times. The prevailing belief is that the position of a borderline child on the neurosis–psychosis continuum is relatively stable—that is, there is not a general progression toward psychosis, although there are psychotic-like episodes.

Although borderline disorders of childhood are not currently part of the official psychiatric nomenclature in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) (American Psychiatric Association, 1980), their adult counterpart is considered to be a personality disorder (i.e., located on Axis II). The DSM-III suggests that Identity Disorder in children is a disorder that, in some cases, is continuous with borderline personality disorder in adulthood. That is, after age 18, the label can change if the symptoms remain. Most recent theoretical formulations suggest that borderline disorders of childhood are phenotypically somewhat similar to adult borderline personality disorders. However, there is not a consensus on whether or not the childhood disorders constitute a personality disorder or should be viewed in some other way. Aarkrog (1981) emphasized that it is important to determine whether these children become adult borderlines, schizophrenics, or have any later pathology. Thus, it needs to be demonstrated whether borderline children are more disturbed than children who become borderline adults.

History

The history of borderline disorders in childhood overlaps considerably with the history of borderline personality disorder in adulthood (see Mack, 1975, and Stone, 1980, for reviews of the

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adult borderline literature). Much of the early theoretical and clinical descriptive literature about adult borderlines (even before the term *borderline* was in vogue) has had a direct influence upon the conceptualization of borderline disorders in children. Like early clinical descriptions, the majority of the early theoretical literature about borderlines refers to childhood even when written about adult dysfunction. This is because most theorizing has come from the traditional psychoanalytic and ego-psychology schools, emphasizing early disturbances in the "separation-individuation process" (Mahler, 1971), "the need-satisfying object relationships" (Freud, 1969), or "pre-oedipal aggressive needs" (Kernberg, 1967).

Two related streams of thinking converged to create the early literature on borderline disorders of childhood. First, clinical descriptions of case studies of severely disturbed children and especially descriptions of "atypical" psychotic children (Geleerd, 1946; Weil, 1953a) led to the idea that there was a unique constellation of symptomatology in some children that did not fit within the framework of neurosis and psychosis that was allowed by traditional psychodynamic theory. Second, analytic and ego-psychology theorists began to speculate that there existed some patients with particular early childhood disturbances, who demonstrated ego deficiencies which did not fit well into the traditional structure of diagnosis either.

One notable early adult study that has major relevance to our contemporary thinking about borderline children was conducted by Deutsch (1942). She wrote of some adult patients who displayed serious character disorders, especially a false relatedness that she termed "as-if personalities." These adults were characterized by a lack of individuality and demonstrated shallow imitativeness. Deutsch felt that these people probably fell between neurosis and psychosis; however, she maintained the possibility that such a personality might have been a step in the process of becoming schizophrenic. In 1946, Geeleerd, in her work with psychotic children, described a subgroup of less severely ill children. She assumed that these children were incipient psychotics and described them as being aggressive, having poor self-control, and fluctuating in their mood and levels of functioning. They were also noted to have poor interpersonal relations.

In 1949, Hoch and Polatin developed one of the early conceptualizations of adult patients who are now called *borderline*. Their term, "pseudoneurotic schizophrenia," was intended to convey the idea that these patients have a less severe form of schizophrenia (with a possible progression toward a full schizophrenic syndrome). Hoch and Polatin felt that because these patients usually had neurotic defense mechanisms and symptoms, they were often misdiagnosed as neurotics. They argued that the presence of disturbed cognitive processes in these individuals warranted their being called schizophrenics. Finally, Hoch and Polatin emphasized that the diagnosis of these patients could be justified retrospectively if the course of the disorder turned out to have been progressing toward schizophrenia.

In 1953, Knight described "borderline states" between psychosis and neurosis in adult patients. The observation of borderline adults continued the interest in this type of psychopathology, but two studies by Weil (1953a,b) helped change the focus of study in the childhood disorder. Weil described children with "certain severe ego-development disturbances" and became the first person to compare these children to adult borderlines and to integrate developmental principles by acknowledging that the disorder would manifest itself in children through different external behaviors than those exhibited by adults. However, Weil believed that the underlying meaning of the behaviors was similar for borderline children and adults. She felt that the ego had not developed sufficiently to give the "characteristic imprint of reasonableness, attempt at control and integration to children of that age" (Weil, 1953a, p. 277). Weil saw these children as having developmental problems in life history as well as in personality organization.

Weil also noted in these children the neurotic symptoms that may be seen when the overflow of anxiety leads to sleep problems, hyperactivity, persistent fears, phobias, and obsessions. However, Weil differentiated these children from neurotics in several areas, including, perhaps most importantly, the changeability of their symptomatology; symptoms appear and disappear and vary in their type and severity. For this reason, an understanding of the whole clinical picture was viewed as necessary for making the diagnosis.

Based upon their clinical experiences, Ekstein and Wallerstein (1954) suggested that borderline children manifest dramatic shifts in their functioning. Neurotic defenses fail and primary process thinking arises during stress. They suggested that autistic and symbiotic interpersonal relations were common in such children. They saw these children as lying between neurosis and psychosis with differentiation possible from childhood schizophrenia

in that borderline children have more control over their fantasies.

Around this time, Mahler (1952; Mahler & Gosliner, 1955) suggested that children with symbiotic infantile psychosis (an early conceptualization of borderline disorders of childhood) had problems with the formation of stable internal object representations. She believed that there were no gross breaks with reality and no primary orientation toward psychotic mechanisms. She also found a group of children who did not fit well into traditional categories and who did not respond to classical psychoanalytic therapy. Mahler (1971) later wrote that borderlines were developmentally arrested somewhere between symbiosis with the mother and object constancy, forever lost in the separation–individuation process and always using "splitting," the inability to integrate such conflicting emotions as anger toward and longing for the mother or the attachment object.

Along with Ekstein and Wallerstein (1956), Anna Freud also stressed the need to differentiate borderline children from neurotic children qualitatively. Although written in 1956, her article "The Assessment of Borderline Cases" was not published until 1969. Her developmental assessment of the topic involved observation of several different characteristics that she felt were important in making a differential diagnosis. She felt that the borderline child manifested deeper levels of regression than the neurotic and also exhibited more massive developmental arrests. She saw these children as withdrawing their libido from the object world and displacing it onto the body or the self. Borderlines demonstrate an inability to receive comfort from others and exhibit several ego defects, including defects in reality testing, poor age-appropriate defense mechanisms, primary process thinking, and concrete thought processes and unstable ego boundaries leading to a confusion between self and others.

In 1958, Geleered indicated that she believed that early disturbances in the relationship with the mother were to blame for borderline patients' ego disturbances. In the next year, Schmideberg (1959) was the first researcher to suggest that borderline was a clinical entity, and not a step along a deteriorating progression. Because adult borderlines seem to remain largely unchanged by time or therapy, she coined the term "stable in their instability."

In 1963, Rosenfeld and Sprince reviewed the literature and pointed out the importance of deviation in all areas of function, with a basic, decisive failure in borderline children's ability to maintain true emotional contact in relationships. Because of their arrest at an earlier stage of development, the majority of these children have confused sex role identities. Many of these children, in spite of their faulty internal organization, can achieve a considerably high level of ego function and often have particular gifts for learning or performing; however, this achievement is characterized by instability and may disintegrate easily under stress.

The fantasy life of borderline children, usually highly defended in neurotic children, is relatively undefended and is easily accessible during times of even slight stress. Their anxiety, which this too easily accessible fantasy life arouses, is characterized by primitive feelings of disintegration and annihilation. Moreover, signal anxiety, a preanxiety warning, is itself perceived as the overwhelming threat. Borderline children experience certain events as traumatic which in other children would not be perceived in that way.

Engel (1963) felt that the strong impact that stress had upon the borderline child's behavior was of great importance and expanded upon Rosenfeld and Sprince's (1963) findings by using projective tests to examine the child's behavior and thought. Because of its unstructured format, a projective test constitutes a stressful experience. Engel used her results to describe a characteristic psychological test profile that could help differentiate the borderline child from the neurotic or psychotic child. In this description, she included the borderline's use of primary process fantasy on projective tests which help reduce the anxiety; the borderline's more intense involvement of the examiner than either neurotic or schizophrenic children; and, in contrast to the psychotic, the borderline's capacity to express massive amounts of anxiety in realistic fantasy. These fantasies are not the same as the grossly distorted fantasies of psychotic children.

Engel also pointed out several themes that ran throughout the tests. One such theme was the theme of "survival." Although borderline children could express anxiety through realistic fantasy, these fantasies were scattered with themes of survival and fear of annihilation. Engel mentioned an issue of "struggle for reality control," which can be interpreted to mean something similar to Ekstein and Wallerstein's (1954) fluctuations in ego state. Borderline children never completely lose contact with reality; rather they "wax and wane" in their reality testing. In order to avoid anxiety, Engel claimed that these children use "distance devices" and "pivotal interruptions" during testing. Distance devices involve the use of fantastic distortions of time and space in order to avoid the mounting anx-

iety produced by the test material. Often, borderline children would interrupt the testing, by asking the examiner a question, in order to avoid the anxiety-producing materials. Therefore, they appeared to be involving the examiner in an effort to relieve anxiety.

Otto Kernberg (1967) is perhaps the most articulate dynamic theorist to describe adult borderline disorders. He emphasized that "borderline personality organization" was the preferred terminology as these disorders are not transitional. He described "splitting" as the essential defensive operation of borderlines and suggested that splitting underlies much of the other pathology and interpersonal dysfunction of borderlines. He hypothesized that premature development of Oedipal conflicts play an important role in the etiology of borderline personality organization. Pregenital aggressive needs are said to motivate splitting.

Frijling-Schreuder (1970) adopted a separation–individuation pathology model of the development of borderline states in children. Differentiation between borderlines and either neurosis or psychosis was said to be a matter of degree of severity of pathology. She argued that borderlines display an ability to use internal speech in self-thinking, which differentiates them from psychotics. Furthermore, borderlines display extreme overt anxiety, which may be the single clearest differentiation from neurosis. Frijling-Schreuder believed that this anxiety was based on a fear of becoming someone else and regarded this anxiety as being symptomatic of a relatively differentiated level of ego functioning. She also noted the tendency of borderline children to have "micropsychotic" episodes and an extreme form of loneliness that leads to their engaging in social conformity. She claimed that borderline children felt "like toddlers whose mothers were permanently out of the room" (p. 312).

Pine (1974), who was one of Mahler's students, integrated the divergent aspects of the literature on borderline children and wrote about a number of aspects of the borderline syndrome that might be parts of different borderline children's personality organizations. The core idea of Pine's important conceptualization of the disorder was developmental.

Although the borderline domain possesses many diverse qualities, the external differences are given unity by the underlying presence of developmental arrest or aberrant development, principally in ego function and interpersonal relationships. Pine (1974) stated that these children's ego malfunction is characterized by a disturbance in distinguishing internal needs from external reality, as well as a related failure in the development of anxiety control so that unpleasant affect readily escalates to panic instead of triggering defensive operations. Interpersonal relationships are characterized by too great a dependence for internal structure on external mothering figures and by too great a tendency to regress to primary identification with and lack of differentiation from these figures.

Pine (1974) wrote about common developmental and structural features underlying the divergent surface features, and also about the divergent underlying features that appear in superficially similar presenting features. Pine argued against a unitary concept of *the* borderline child, which he saw as just as odd an idea as the concept of *the* neurotic. Instead of trying to combine all the theoretical and somewhat contradictory descriptions of these children into a single generalized concept, Pine instead proposed a set of phenomena to which he felt this term could be meaningfully applied. These phenomena are by no means mutually exclusive subtypes of the syndrome; instead, they are different aspects of the disorder all of which may exist to differing extents among the children. These phenomena also differ in severity, ranging from those with strong, perhaps genetic, pathology and with only a small reactive component, to those in which the major component is reactive, to those with firmer internal structure but with a gross developmental aberration or arrest in ego function and/or in interpersonal relations.

The phenomena involving strong integral pathology are those which he called "chronic ego deviance" and "shifting levels of ego organization." Central to the chronic ego deviance phenomenon is the failure among the children to make a complete transition from pleasure to reality principle, an unreliable use of signal anxiety, and fluctuating interpersonal relationships. Without mastery of the reliable use of others in order to grow and individuate, these children lack the basic stabilizers of functioning. This phenomenon is similar to that described by Weil (1953a,b) and to that by Rosenfeld and Sprince (1963).

The shifting levels of ego organization phenomenon entail the achievement of true ego organization but on at least two different levels. This is the "fluid ego organization" to which Ekstein and Wallerstein (1954) referred. This phenomenon involves a fluctuation back and forth from an external reality orientation, which is often painfully troubled, to a more psychotic-like way of dealing with

anxiety through a defensive retreat into a world of fantasy. Socially, these fluctuations range from true interpersonal relationships to near autistic unrelatedness or a symbiotic absence of differentiation of the other. This shift in state is related to the children's being in and out of contact with a mothering figure, such as a therapist or a teacher.

The phenomena involving a major reactive component are called "internal disorganization in response to external disorganization" and "incomplete internalization of psychosis." The first involves pathology being carried within. This pathology is the readiness to change seen in some of the children according to the structure or stress provided by the environment. These children often come from families full of abuse, neglect, psychosis, and violence, and their pathology is a function of their adaptation to this pathological environment. This overstimulation brings about imitative repetition and interferes with the process of repression; as a result, their primary process fantasy is often freely accessible. Often, these children are referred from deprived, impulse-ridden homes and neighborhoods because of their destructive behaviors, extreme uncontrollability, hallucinations, and shallow or inappropriate affect. Yet, in the stable consistently caring environment of the ward, they soon integrate well and begin to relate well, often appearing normal. These children seem to have been always capable of trust and realistic thought processes if their environment had permitted it.

The second in this group of phenomena, incomplete internalization of psychosis, involves an alternative reaction to family pathology. Instead of reacting with collapse, pain, repetition, or search for rescue, these children internalize the loved pathological parent along with their psychotic features. They react by enmeshing themselves in a symbiotic relationship that has grossly distorted features.

The phenomena involving firmer intrapsychic structure but with faulty development of ego functions and/or interpersonal relations are called "ego limited" and "schizoid personality of childhood." Ego-limited children show different combinations of peculiarities, but they all reveal a common dullness and meagerness, without judgment, self-care, and playfulness. These features seem primarily to be the result either of extreme environmental deprivation or of a biological disposition that leads to an early restriction of ego development. Further growth is stunted because thought is not elaborated, affect is not differentiated or modulated, and healthy relationships do not develop.

The schizoid personality of childhood involves an acutely constricted and underdeveloped affective life, with emotional distance in human relations, and preoccupation with one's own fantasy life. Bland interpersonal relations are replaced by an affectively charged fantasy life. Other aspects of these children's ego function are intact enough to permit these children to function passably, as in school. Their fantasy works so successfully as a defense against internal drives and external relationships that the threat of anxiety is not always experienced in disorganizing ways. Consequently, they do not endure the panic present in other phenomena. Nonetheless, even though they stabilize a character structure, they do so at the price of oddness and isolation.

Pine (1974) also offered another perspective on differentiating the borderline syndrome from neurosis and psychosis. He wrote that borderline states are clearly distinct from neuroses in that the states involve developmental arrest at an earlier stage and failures in the maturation of the ego and interpersonal relations; neuroses involve a longer period of normal development before pathology sets in (cf. Freud, 1969).

The distinction between borderline states and psychoses is a bit hazier. Pine wrote that borderline states are definitely distinct from infantile autism, symbiotic psychosis, and the type of childhood schizophrenia that is based on regression and with thought disorder. Borderline states are also distinct from psychoses in that they lack the fixity and structure of psychosis and are thus more highly influenced by the environment. Borderline children cannot deal with their anxiety by means of the rigidly structured defenses of the neurotic. Moreover, borderline children do not possess the gross reality distortion of psychotic children as a means to deal with their anxiety. Thus, borderline children's level and quality of functioning are highly dependent upon the structure and stress of the environment, are highly labile and reversible, and are constantly fluctuating.

In summary, Pine (1974) included in the borderline phenomena of childhood patterns of behavior based on developmental failure of interpersonal relations and of differentiating the boundary between self and others. Pine took the spatial metaphor of the borderline label seriously and tried to clarify the borders surrounding the domain—the clear border with neurosis on one side, and the hazier border with psychosis on the other. Finally, he tried to clarify the borders within the domain by defining various aspects of the syndrome.

Recently, Pine (1986) proposed a model of the development of the "borderline-child-to-be." He felt that three central themes contribute to the development of borderline disorders in children. (He was not certain that adult borderline disorders take the same developmental route). The three themes are: the experience of early trauma (which can be overt trauma or severe stress caused by poor or mismatched parenting), the evolution of developmental deficits, and the development of coping skills that end up becoming neurotic defense mechanisms. Even though this model is a fascinating integration of psychodynamic theory, object relations theory, and ethologically based attachment theory, it is primarily descriptive of the pathology of borderline children. Accordingly, because Pine's notions do not stem from empirical data, the model does not yet possess predictive power.

As the literature on borderline children has grown in sophistication and abundance, reviews and critiques have appeared. For example, Bemporad, Smith, and Hanson (1981) published a chapter on the subject in the *American Handbook of Psychiatry* (Arieti, 1981). Aarkrog (1981) compiled a review of the borderline literature in adults, adolescents, and children and included many detailed case studies. Robson (1983) has edited a book on the etiology, diagnosis, and treatment of borderline children.

Diagnostic Issues

Efforts have been made to develop and examine diagnostic criteria for borderline disorders in children. Currently, nothing exists for children that is as well developed as Gunderson's adult Diagnostic Interview for Borderline Patients (DIB) (Gunderson, Kolb, & Austin, 1981). The DIB was developed by compiling clusters of symptoms from clinical descriptions and diagnostic schemata that had come before it and then validating the interview. The DIB can be used as an interview tool or as a means of rating hospital records (DIB-R) (Armelius, Kullgren, & Renberg, 1985).

Following Pine's descriptive outline and based upon their clinical experience, Bemporad *et al.* (1982) attempted to further our understanding of the borderline syndromes in childhood by outlining what they believed to be the manifest symptoms of the disorder. Like Rosenfeld and Sprince (1963), these authors saw the need to view the entire clinical picture before making the diagnosis, since no single symptom is pathognomonic. Bemporad *et al.* (1982) felt that "only when the total compilation of dysfunction is evaluated does a coherent pattern emerge which may be differentiated from childhood neurosis and psychosis" (p. 234). They felt the manifest symptomatology or characteristic areas of general pathology were: (1) fluctuation of functioning, (2) nature and extent of anxiety, (3) thought content and processes, (4) relationships to others, and (5) lack of control.

The concept of fluctuation of functioning is the same ego fluctuation discussed by Ekstein and Wallerstein (1954). Bemporad *et al.* (1982) felt that these rapid fluctuations were one of the major differences between the borderline child and the neurotic or psychotic child. They discussed a "rapid shifting in levels of psychological functioning, from healthy or neurotic organization to psychotic-like states with intrusion of bizarre thinking, grossly inappropriate behavior and overwhelming anxiety" (p. 235). Borderline children will partly relieve their anxiety by resorting to such psychotic states during stressful situations; however, they do not remain in a psychotic state for as long as psychotic children would. In addition, assurance and support will terminate the episode.

Bemporad and colleagues stated that the nature and extent of anxiety is another key symptom in the diagnosis of borderline children. This characteristic is manifested by the constant presence of varying degrees of anxiety and by an inability to control the escalation of anxiety. Consequently, these children are always on the brink of decompensation that is due to this rising anxiety. Recall that borderline children possess an inability to use signal anxiety beneficially and treat such warnings as a threat, which causes a rapid escalation to panic and terror. Borderline children suffer more from this anxiety than do psychotic children because of their inadequate defense mechanisms. Although pathological in their gross distortion of reality, the psychotic's defenses offer some relief from anxiety. In contrast to neurotic anxiety, which may be caused by an urge to disobey while aware of the possible result in punishment or loss of esteem, the borderline child's anxiety is rooted in self-annihilation, body mutilation, and world catastrophe.

Similar to the fluctuation in ego state, Bemporad *et al.* (1982) viewed the thought content and processes in general as flowing with excessive fluidity between reality and fantasy. Some of the thought disturbances exhibited by borderline children are typical of schizophrenic thought, such as concretization, but are seen in much milder forms. The child's fantasy themes are based in everyday

reality, yet demonstrate the child's anxiety over self-annihilation, body mutilation, and world catastrophe. During periods of stress, the borderline child's thoughts are marked by the inability to control the progression between neutral thoughts and these anxiety-producing, more destructive, fantasy thoughts. Cognitive difficulties that are displayed on psychological testing and may indicate a neurological defect include difficulties in focusing attention, a variety of learning disabilities, difficulties in perceptual motor tasks, and poor abstract concept formulation.

Many clinicians believe that the fourth area, relationships to others, is the foundation of the borderline syndrome on which the other symptoms are based. These children demonstrate a constant need for support and assurance, but, at the same time, are not selective in choosing a supplier. Borderline children will substitute one person for another as long as they continue to receive the needed support. These relationships are characterized by the criteria outlined in Deutsch's "as-if" personality (1942), including the childlike mimicry, the overintense identification, the perception of completeness, and the willingness to mold their behavior to others' expectations. These children usually have poor peer relationships, for peers are not as tolerant as adults. The borderline may bully and torment younger children, yet simultaneously harbor paranoid fears of older children. In a group, these children tend to withdraw because they do not wish to share the affect they are given.

The final characteristic outlined by Bemporad *et al.* (1982) was lack of control. Borderline children show an inability to control anger, inhibit impulses, delay gratification, or suppress frightening fantasies. When confronted with frustration, they have a tendency to work themselves into a rage, ending in a temper tantrum. The tension seems to mount until they cannot control themselves any longer and become violently aggressive toward others or even themselves. During therapy, borderline children cannot control the escalation of frightening fantasies, and these fantasies move toward destruction and mutilation until the therapist intervenes, helping the child alter the fantasies' paths. Bemporad and his colleagues suggested that this deficit may account for some of the pseudoneurotic symptomatology. These children may form obsessions to protect themselves from some dreaded event. They may perceive rituals as having a magical quality, or they may develop phobias that allow them to avoid an object which elicits a chain of terrifying thoughts.

In addition to the five manifest areas of general pathology, Bemporad and colleagues (1982) included a category of associated symptoms. These symptoms included neurological problems, learning problems, and hyperactivity, among others. The authors used their criteria to identify a group of borderline children, aged 5 through 12, from psychiatric hospital records. This set of diagnostic criteria is an important first step in the validation of the category of childhood borderline disorders.

The Bemporad *et al.* (1982) criteria have also been utilized by other researchers. Bentivegna and colleagues (1985) used these exact criteria with hospital case records to identify a rigorously defined subgroup of borderline children. They were able to distinguish a group of 70 borderline children from two control groups of children, who were hospitalized for other psychiatric reasons.

Greenman *et al.* (1986) compared these criteria to the DIB-R (with minor modifications) on a group of children's medical records. They interpreted their findings to suggest that there are few important differences between borderlines and comparison groups of nonborderlines. They question the merit of the use of the term *borderline* with children. The DIB-R, however, is an adult measure and may be developmentally inappropriate for use with children in its current form. The use of the DIB-R in this way implies a belief in the phenotypic and etiologic continuity between adult and child borderline syndromes; but there is no consensus on such concordance. The comparison of the two sets of criteria, however, can be a useful step in the understanding of the diagnosis of borderline children. The authors, however, did not emphasize the amount of comparability between the two systems.

In a recent chapter, Vela *et al.* (1983) reported on a set of diagnostic criteria that they had developed by reviewing the literature on childhood borderlines. (They had presented these same criteria at a meeting of the American Academy of Child Psychiatry in 1980.) They found six criteria that are almost universally mentioned in diagnostic systems and a host of frequently and infrequently listed other symptoms. These primary criteria are: (1) disturbed relations with individuals, (2) disturbances in sense of reality, (3) excessive intense anxiety, (4) excessive and severe impulsive behavior, (5) neurotic-like symptoms, and (6) uneven or distorted development. These criteria are quite similar to those of Bemporad's group. Although Vela *et al.* (1983) claim that inconsistency characterizes the diagnostic literature and jeopardizes the utility of the term *borderline* with children, we disagree. We

see convergence of thinking reflected in the similarity of these two sets of diagnostic criteria. Furthermore, the DIB, though adult oriented, is also similar. It appears that researchers may be moving toward a consensus in their formulations of the basic symptomatology in borderline children.

Etiology

Poor quality early infant–mother interactions are often viewed as the primary cause of childhood borderline disorders even though there is little available empirical evidence to support this position. The majority of these theories suggest a developmental deviation or arrest during early interaction, with the most popular theory hypothesizing that the arrest occurs during the developmental stage of separation–individuation. Early theorists, such as Geleerd (1945, 1946, 1958) and Mahler, Ross, and DeFries (1949), and Mahler (1952) not only described the disorder but also noted the possible etiological significance of the early parental influence. They also used their hypotheses and theories to differentiate borderlines from psychotics and neurotics.

Soon after it was developed, the theory of arrest during the separation–individuation stage was being used to explain borderline children's behavior, ego development, and other levels of functioning. In 1956, Weil used this concept and applied it to reveal different observations she had made about these children. Noting that these children have poor reality testing, she used the separation–individuation concept to explain their need for symbiotic relationships. In addition, Weil extended the separation–individuation concept to the social sphere, hypothesizing that the reason these children fail to develop good quality peer relationships is that they have a strong need for symbiosis and narcissistic identifications. Weil stated that a truly symbiotic relationship, which would satisfy these children, is extremely rare outside the realm of a primary caregiver or therapist.

One of the most important contributors to the object relations theory of borderline disorder was O. Kernberg (1972) who argued that the fixation occurs after the period of self-object differentiation before the onset of object constancy. Unlike other object relations theorists, Kernberg (1966) emphasized the role of a constitutionally determined lack of anxiety tolerance which interferes with the process of integrating the "good" and "bad" self and object images. Kernberg (1966) argued that the presence of an excess of oral aggression and the inability to neutralize such aggression indicate a constitutional etiology. Because of their low degree of anxiety tolerance, these infants cannot hold both the bad and the good images simultaneously. In order to lessen the anxiety that such an effort provokes, there is a splitting of the "bad" and the "good" self-images.

More recently, Adler and Buie (1979) formulated an understanding of borderline disorder as resulting from an arrest in affective-cognitive development. According to Fraiberg (1969), the development of object permanency accompanies the shift from recognition to evocative memory, which occurs roughly around 18 months. With recognition memory, an image cannot be evoked unless aided by the presence of the object. With the acquisition of evocative memory, the object or person has permanency and can be evoked unaided by the object's presence. Adler and Buie (1979) believed that it was the failure of adequate mothering during the separation–individuation phase that leads to the inability to proceed from the stage of recognition memory to evocative memory in the area of affective object relations.

Fast and Chethik (1972) also conceptualized borderline disorder as resulting from a developmental arrest. They noted the inability of borderline children to fully integrate self and other-object images into a coherent whole. Rather, these children deal with others in terms of their own projections without taking the reality of others into account. Using the example of Gary, an 8-year-old borderline patient, Fast and Chethik (1972) argued that the characteristic of borderline disorder was the use of others as props for the borderline's own internal projections. In their view, borderline children are in a transitional stage between pleasure and reality ego. The advancement from the pleasure ego to the reality ego requires the ability to perceive external reality as being distinct from oneself as well as the ability to use the external world as the measure of reality against which images of the self and others are compared. Unlike more mature functioning individuals who can control regression to this primitive stage, borderline children are developmentally stuck in this transitional stage, and they lack the ego control needed to prevent excessive fantasy.

Masterson and Rinsley (1975) argued that maternal availability plays a key role in the etiology of borderline disorder. This conceptualization was based on family therapy casework with parents of borderline adolescents, the treatment of borderline mothers, observation and study of borderline moth-

ers in conjoint interviews with borderline adolescents, and long-term psychodynamic treatment of borderline adolescents. In their view, the typical borderline psychic structure results from a fixation at the separation–individuation phase that is due to the mother's withdrawal of her libidinal availability during the rapprochement subphase of the separation–individuation process. The fixation occurs then, because of the child's individuation efforts that threaten the mother's own defensive need to cling to her child. Defensively, she withdraws as her child separates. Although the mother is available only when the child clings and regresses, the child introjects both images of the mother (withdrawing and available) as part-object representations together with their associated affects and self-representations. This generates the split-object relations unit that is characteristic of the borderline patient.

Attempts have been made at more systematic studies that would give empirical support to these theoretical postulates. In one of the few systematic studies, Bradley (1979) examined the early separation experiences of borderline children and found that, during the first five years of life, borderline children experienced more frequent separations from mother figures than did neurotic, psychotic, or delinquent children. Although Bradley's study was an important step in the right direction toward a better understanding of the borderline syndrome, there were two basic flaws in this study. First, the children were diagnosed using criteria devised to diagnose adults. Second, the nature and extent of the separations that were experienced were not specified.

In addition to the theories that emphasized the role of mother–infant interaction in the etiology of childhood borderline disorders, several investigators have focused more broadly on the familial environment of these children. In 1981, Gunderson and Englund reviewed the empirical literature about the families of borderlines. Although these studies were plagued with poor diagnostic criteria and methodological problems, they felt that the data were good enough to suggest that disturbed family life may play an etiologic role in borderline disorders. This summary is not inconsistent with the major analytic and ego-psychology theories of etiology. For example, in a study of the families of borderline patients, Zinner and Shapiro (1975) found that the pattern of withdrawal related to separation efforts on the part of the borderline was not only characteristic of the mother, but of the entire family as well. In their analysis of family interactions, Shapiro, Zinner, Shapiro, and Berkowitz (1975) discovered that the parents themselves relied on projective identification and experienced difficulty in seeing their children as separate individuals.

Similarly, Bemporad *et al.* (1982) believed that family disturbances played a major etiological role in the development of childhood borderline pathology. For example, of the 24 children studied, 10 had a history of physical abuse. There was also a history of neglect with bizarre behavior and inconsistent care on the parents' part. Mothers were seen as unstable, easily frustrated, and unable to maintain many aspects of interpersonal relationships, and the fathers evidenced difficulty in self-control and instability in relationships. Virtually every family was characterized by chaos, a sense of turmoil, and a sense of impending violence combined with a lack of consistency in the parents' behavior. Bentivegna *et al.* (1985) corroborated these findings in a controlled study. Bemporad and his colleagues speculated that this type of environment might impair these children's development in several areas. First, it might impair their development of a secure sense of self, thereby making it difficult for them to regulate their anxieties. Second, it might impede the development of social competence that is required for the formation of satisfactory peer relations and necessary for adequate latency ego functioning. Finally, the stress involved might give rise to difficulties in reality testing and to preoccupations with self-annihilation and destruction.

Parent and family pathology seems to play an etiologic role in the development of borderline syndromes. However, questions still surround the diagnostic status of the parents of borderline children. Masterson (1975) found them to be borderline themselves. However, Singer (1979) found that 84% of borderline patients had either normal or neurotic parents based on psychological tests. In their work, Shapiro *et al.* (1979) found varying types of psychopathology among the parents of borderline patients.

Finally, several theoreticians have postulated that organic factors play an important role in the onset of childhood borderline disorders. Although O. Kernberg (1966) argued for a constitutional basis of the disorder, until very recently few neuropsychological tests were conducted with borderline populations. Bemporad *et al.* (1982) noted that compared to their siblings and to children with other types of psychopathology, many borderline children show evidence of organic impairment, including poor coordination, perceptual motor difficul-

ties, abnormal EEG readings, and hyperactivity. Others have also postulated a relationship between the neuropsychology of hyperactivity and borderline disorders (Cohen, Shaywitz, Young, & Shaywitz, 1983). Some borderline children also experience learning disabilities. Bemporad *et al.* (1982) postulated that the organic difficulty, although not causing the borderline disorder, may exacerbate the problems of anxiety regulation, reality testing, and poor motor control.

Follow-Up Studies

Most of the information available on the follow-up of children diagnosed as borderline comes from a few Scandinavian studies. Wergeland (1979) studied the outcomes of 29 children who were hospitalized for borderline psychosis across a range of ages from 2 to 13 years. The assessments were conducted between 5 to 20 years after the original diagnoses were made. There were no significant differences between a treated and a nontreated subgroup. Of the whole sample, 5 were still borderline, 4 were manifestly psychotic, 3 were moderately disturbed (some neurotic symptoms), 6 were severely neurotic, and 11 were symptom free. Wergeland concluded that little prognostic information was gained through this study, although the outcomes for borderline children seemed somewhat more promising than reported in the literature for other psychotic groups.

Aarkrog (1975, 1977), in a "follow-back" study, described the childhood histories of 50 borderline psychotic and 50 psychotic adolescents who had been admitted to the adolescent psychiatry department of a hospital in Copenhagen, Denmark. Of the 100 patients, 21 had been borderline in childhood and 22 had been healthy, although, oddly, these numbers were not broken down into the proportions of later psychotic and borderline disorders. Aarkrog (1975) reported that diagnoses may change for individual patients as they shift from childhood to adolescence but that symptomatology remained similar. Unfortunately, the data were not presented in a way that allows for systematic consideration of developmental or prognostic issues.

Dahl (1976) sampled 322 consecutive admissions to a clinic and found 6 borderline children. At a 20-year follow-up, 3 of the 6 had not had any further psychiatric hospitalizations (but were not sought out for assessment in the study). Three others were found on the "folkeregister" indicating later hospitalizations. Of these, 2 were borderline and 1 was labeled schizoid character disorder.

Taken as a whole, the data from these follow-up studies do not do much to advance the understanding of borderline disorders in childhood. Methodological shortcomings preclude drawing conclusions about the prognosis or course of borderline disorders. In these studies, diagnostic criteria were not specified. Only general theoretical and clinical descriptions of the prototypic patient were offered by Aarkrog (1975) and Wergeland (1979). The samples were either small or quite varied in age range, and no attempts were made to incorporate age-specific considerations in diagnosis. Further, there were no attempts made to insure blindness of the follow-up diagnosticians nor were there reliability checks on the diagnoses. Although there is some consensus among these researchers that borderline children have generally poor prognoses, there can be no certainty as to the diagnoses of the children described. Finally, it remains unknown how symptoms progress for children of varying ages with borderline disorders.

Treatment

Because of the severity and, perhaps more importantly, the changeability of the symptoms in borderline children, it is difficult to characterize an optimal treatment plan. Ideally, therapists and case managers should possess a large armamentarium of therapeutic techniques and should have contact with the parents, teachers, social workers, and other persons and professionals who are intimately involved in the child's world. Most of what is currently known about treating borderline children comes from clinical case reports and several good summaries provided by therapists who have worked closely with such children. The majority of the information about how to conduct individual psychotherapy with these children has been drawn from classic psychodynamic and ego-psychology perspectives. Recently, inpatient and day-treatment services (Hanson, Bemporad, & Smith, 1983), drug therapies (Petti, 1983), and the school milieu (Schimmer, 1983) also have been suggested as important treatment modalities.

Borderline children are prone to outbursts of anger and bouts of paralyzing anxiety, both of which happen seemingly without provocation. Additionally, the ambivalence and rapid changes in their interpersonal relations make them unusually

difficult patients for therapists to manage. Clinicians working with them need to be exceptionally tolerant of the children's outbursts. Moreover, they require special training or supervision in order to help them to understand and tolerate the special needs of such children. Finally, therapists must learn to handle their own inner frustrations that may be evoked by dealing with borderline children.

In general, the relationship between the child and the therapist is viewed as the primary vehicle for achieving advances in therapy. Rosenfeld and Sprince (1963) offered several techniques for the initial stages of individual psychotherapy with borderline children. They stressed the use of ego-supportive handling and the importance of the appropriate use and timing of interpretations. The use of ego-supportive handling refers to providing a physically safe and penalty free environment and the means for children to discharge and to contain tension—a kind of healthy sublimating of their acting out. Rosenfeld and Sprince (1963) also recommended that anxiety not be interpreted in initial sessions because such interpretations are often frightening and overwhelming to borderline children. They felt that the children could not confront their hostility until they had formed a relationship with the therapist. Furthermore, Rosenfeld and Sprince (1963) warned that defensive reactions on the part of therapists could be particularly dangerous with borderline children, primarily because these reactions could cause fear and exaggerated responses in these extremely sensitive children. In a similar vein, they cautioned therapists to avoid reactions of shock during these children's periods of abrupt psychotic-like behavior. Rather, Rosenfeld and Sprince (1963) urged treaters to work on themselves so that, for brief periods, they could genuinely meet borderline children's needs for symbiosis and follow their modes of communication, even on a primary-process level.

The idea of being supportive at the primary-process level has also been promoted by others. Ekstein and Wallerstein (1956) suggested the technique of "interpretation within the regression." Specifically, during such instances the therapist transiently enters into the borderline child's distorted perception of reality and renders an interpretation that can be understood and appreciated by the child at that moment on his or her own terms. Later in treatment, interpretations gradually become more mature and reality oriented. This technique allows the child to feel understood while negotiating a relatively stress-free transition into more intense therapeutic issues. Moreover, this mode of treatment can prevent further deterioration in borderline children.

P. Kernberg (1983) took a somewhat different approach, even though the major goal, the resolution of primitive defensive patterns with the formation of integrated and stable self- and object-representations, was similar to that espoused by Rosenfeld and Sprince (1963). She emphasized the here and now and suggested that splitting should be dealt with promptly in the session and that the therapist should keep in mind that the child does not possess the ability to test reality accurately. In her opinion, children experience less anxiety when their primary-process ideation is accepted and interpreted. It is felt that this therapeutic modality helps borderline children to develop the ability to sublimate such ideation into play, dreams, and daydreams.

The approach of Hanson *et al.* (1983) is also very reality oriented and stands in stark contrast to Ekstein and Wallerstein's (1956) notion of interpretation within the regression. Hanson and colleagues (1983) believe that borderline children can often frighten themselves with their bizarre fantasies. As regression occurs, they lose their ability to differentiate objective reality from their fantasy. It is precisely at this point that a reality-grounding intervention should be made. Such interventions recognize and label the children's emotions. The source of these emotions is identified as fantasy, and the children are reassured that reality is safe and psychically available to them. Hanson *et al* (1983) contended that part of the problem in the family of borderline children is a vicious cycle of parent and child regressive behavior feeding one upon the other until both are involved in bizarre, sometimes mutual, primary-process ideation. Ending such a negative feedback cycle is an important way of helping the child.

The families of borderlines have received surprisingly little attention from researchers. By and large, they appear to be quite disturbed, chaotic families (Bemporad *et al.*, 1982). Gunderson and Englund (1981) described family patterns of rigidity and intense expressions of affect, with mothers being either excessively dominant or passive and fathers who may openly express aggression. In a study of 12 families of borderlines, Gunderson, Kerr, and Englund (1980) found rigidity in the marital bond and an exclusion of the children in terms of attention, support, and protection to the point of being neglected and/or abused (see also

Bemporad *et al.*, 1982; Bentivegna *et al.*, 1985). Gunderson *et al.* (1980) recommended that family therapy come later, after an initial period during which the parents are seen without the child. This procedure allows the parents to be educated and allows their help to be enlisted, two things which might not occur if the unusual family dynamics were to be played out by the whole group before the therapist had the chance to assess the situation or intervene. Often blaming and accusation will mar family therapy with borderlines if it is begun too early.

Feldman and Guttman (1984) proposed that there were two constellations of family pathology in the families of borderlines. In one group, one parent is very literal minded and concrete without empathic abilities for the child. In the other family pattern, one parent has a borderline personality disorder, and the child is the target of the parent's regressive distortions of reality. In each family, the other parent is unavailable for protective or buffering functions, rendering the child especially vulnerable to the pathology of the former parent. The authors suggested that in each case the focus of family therapy should be to encourage the healthier, less influential parent to take a more active role in protecting and demonstrating positive affect and empathy toward the child.

The possibilities of working effectively with the families of borderline children are often slight. As suggested above, adult pathology may be a partial cause of child pathology, and it quite likely will be an obstacle to effective treatment. In many instances, it is difficult to engage borderline families in treatment. Balking or termination may occur at the suggestion that parent characteristics contribute to the child's problem. These parents are often looking for an explanation that absolves them of responsibility or guilt. They may, in fact, have a fairly elaborate explanation firmly in mind and may resist mightily the therapist who suggests otherwise. The borderline parent is also often looking for a magical cure and may resist treatments that involve a great deal of psychic work, such as intensive therapy. Borderline adults also tend to resist activities that involve a large amount of organizational effort, such as getting their child to a number of appointments and arranging treatment (Hanson *et al.*, 1983).

Because of the severity of problems in some borderline children's lives, and because of the frequency of poor support from their parents, Hanson *et al.* (1983) argued for inpatient or day-treatment services for some borderline children. Residential treatment allows for the negative relationship between child and parent to be eased. The parents of a borderline child are often the catalyst for the child's regressive behavior and vice versa. The sheer distance in time and place can be therapeutic for some families. In some extreme cases, arrangements have been made for foster care or long-term residential schooling for these children. Another valuable feature of residential treatment is that it allows for a unified and coherent case management. Hanson *et al.* (1983) called for a multidisciplinary team approach, coordinating trained caretakers in the milieu with therapists, special education teachers, and social workers who work with the family. The administration of medication can be closely monitored, too. Clearly this level of control is not possible on an outpatient basis. A final important facet of inpatient treatment is the degree to which the family is drawn into the treatment. On-site and home visits assure the therapist that parents feel as though they are part of the therapeutic team. Day-treatment facilities are an approximation of inpatient services, and all of the above advantages of inpatient treatment probably apply in a diluted form to day services as well.

A review of psychopharmacological treatments of borderline children currently can be no more specific than a review of several bodies of literature focusing upon the major constellations of symptoms associated with borderline disorder. A recent chapter by Petti (1983), summarizing the literature on psychopharmacological treatment of borderline children, illustrated the lack of drug-therapy experimentation targeting this group *per se*. Anxiety, depression, hyperactivity, aggression, mood swing, and "psychotic reactions" are all symptoms that have been said to be common in borderline children. Each of these symptoms has been studied with respect to psychotropic drug therapy. Petti's chapter summarized the literature as follows: Minor and major tranquilizers are frequently prescribed for anxiety in children. The major tranquilizers (neuroleptics) are also the preferred treatment for psychotic reactions. Imipramine is often useful in treating depression and methylphenidate and dextroamphetamine are often prescribed for attention deficit disorder with hyperactivity.

Although there is little experimental drug evidence that lends support to the notion of borderline as a syndrome or even as a unique constellation of symptoms, drug therapy is often very useful in the management of children so diagnosed. Bemporad *et al.* (1981) suggested:

> Generally, anything that will help increase his sense of mastery is very important to a borderline child, and if medication can help him decrease or tolerate severe anxiety and control his bodily functions, it should become a part of the overall treatment plan. (p. 250)

They did warn, however that "side effects, although limited and usually not serious, can be distressing to borderline children and their families, who often have rather extreme and magical fantasies about drugs and their effect on the body" (pg. 250). Thus, though medication can be a great asset in the treatment of borderline children, it, like all aspects of treatment, must be used with exceptional sensitivity for this group. There seems to be unanimity in the literature which suggests that drug therapy should be viewed as part of a therapeutic treatment plan and not as a singular treatment modality. Lewis (1976) suggested that milieu therapy, individual psychotherapy, and drug treatment comprise the best plan for some borderline children.

Recently, Schimmer (1983) illustrated how the predominant school culture in this country is directly at odds with the borderline child's particular patterns of behaviors and deficits. The focuses of school upon external objects, working to please others, delaying gratification, group cooperation, and reality contact all clash with the particular inabilities of borderline children. Schimmer argued valiantly why the borderline child will have great difficulty in school and then turned his attention toward the idea of changing the milieu to help borderline children. Schimmer suggested that teachers of borderline children be closely supervised by persons who are extremely knowledgeable about borderline syndromes and the case of the particular child in question. He also suggested that smaller classrooms would be better for borderline children. These changes would likely be for the better as far as the child was concerned. Unfortunately, it seems unlikely that many school districts have the resources to provide such services to "mainstreamed" children. In fact, the description given by Schimmer (1983) bears a resemblance to special education services. These are the type of classes in which many borderline children wind up when it is decided that they cannot handle regular classrooms.

Future Directions

Until recently, the vast majority of work on borderline children has been dominated by psychodynamically oriented developmental theories, most notably ego-psychology and object relations approaches. Furthermore, although very little empirical work has been conducted, that which has been done has not been influenced by the theories and research methods of mainstream developmental psychology or experimental psychopathology. Most of the studies have been characterized by poor methodologies (e.g., unblind ratings, no control groups, and small, unrepresentative samples) and have been primarily retrospective follow-back studies in nature. Although several follow-up investigations have been carried out, the field sorely needs more of these studies, as well as prospective follow-ups of children at "high-risk" for the development of a borderline disorder (e.g., the offspring of borderline parents, or severely maltreated children reared in especially chaotic homes). As a result of the conceptual and methodological biases inherent in the work to date, most of the etiological theories used to explain the clinical psychopathologies of these children have employed complex metapsychological constructs. Over time, these notions have been reified before there has been any chance to refute and/or to validate them.

In addition to the elusive nature of the clinical constructs proffered to explain the borderline disorders of childhood, the etiological models themselves have been overly simplistic. Even though they are implicitly interactional in nature, they read like linear, "main-effects" (Reese & Overton, 1970) models of etiology in which aberrant parental behavior, chiefly maternal, is viewed as producing borderline disorder.

Although there are some similarities in the causal models that have been put forth, there likewise exists an array of competing descriptions in the literature. It is not clear which theory (or whether any single theory) is best. Because the borderline disorders may be a heterogeneous group of disorders, it is plausible that there might not be a unitary etiology. If we agree with Pine (1974) that there are several subgroups of these children, then we ought to uncover different patterns of organization in these subgroups. That is, there may be multiple divergent pathways that lead to the development of childhood borderline disorders. Given the possible contributory roles of the family, the chaotic environment, parental psychopathology, and neuropsychological impairment, in addition to the deviant interactional patterns within the parent–child dyad, it is important that researchers and theoreticians increasingly adopt a transactional model of etiology (cf. Cicchetti & Schneider-Rosen, 1984;

Sameroff & Chandler, 1975). A good movement in this direction has been put forth by Bemporad and colleagues (1982) who hypothesized that borderline syndromes in childhood result from multifactorial etiologies, including organicity, deprivation of early socialization, and constant exposure to chaotic, traumatic environments. Sufficiently large populations of borderline children need to be studied in order to allow for competing and transactional theories to be tested.

In order to accomplish the tasks described above, we need to develop reliable and valid diagnostic criteria. Results here are encouraging because good agreement among researchers is developing. Moreover, preliminary findings have yielded good interrater reliability ratings using formal diagnostic criteria. The current belief is that diagnosis before latency age (below age 5) is not yet possible. However, because many of the etiological theories state that borderline disorders originate during the separation–individuation phase of development, it is important to ascertain whether the disorder can be reliably diagnosed during the prelatency/preschool years. Studies are needed which follow very young high-risk or preborderline children prospectively.

Although many theorists (cf. O. Kernberg, 1967) argue that there is an underlying borderline personality organization, it may be a wise strategy in predictive outcome studies to look for continuity at the level of underlying organization rather than to search for symptomatic isomorphism over time. Instead of focusing on discrete symptoms in making the diagnosis of borderline disorder in childhood, it may be more fruitful to assess concurrently the child's resolution of the stage-salient issues of development for his or her age period (e.g., attachment, self-development, autonomy, affect regulation, and peer relations). Thus, preschool borderline children may not look identical to school-aged borderlines. Analogously, child and adult borderline disorder may also be phenotypically dissimilar. The idea of an underlying continuity in the organization of psychopathologies across developmental levels has been suggested for other disorders (Cicchetti & Schneider-Rosen, 1986; Kohlberg, Snarey, & Ricks, 1984). Again prospective follow-up studies of children at "high risk" for the development of the borderline syndrome may shed light on this more organizational approach to childhood borderline syndromes.

Finally, we believe that the study of borderline children from an organizational, developmental psychopathology perspective, guided by a transactional model, can make significant contributions to our understanding of many issues extant in normal developmental theory (Cicchetti, 1984). For example, careful studies of the interactional precursors to attachment outcomes in borderline children should broaden our knowledge of parent–child relationships. It is very likely that these children would form insecure disorganized/disoriented, or Type "D", attachments, given the chaotic environments they experience as well as the severity of accompanying maltreatment (see Carlson, Barnett, Braunwald, & Cicchetti, 1989, and Main, Kaplan, & Cassidy, 1985, for descriptions of the "D" category). Furthermore, an inspection of their manifest symptomatology suggests that the study of borderline children could contribute to our knowledge base in several other important areas, including the relation between affect and cognition, the development of "self-system" processes, affect regulation/dysregulation, peer relations, and the ability to integrate multiple conflicting emotions (the opposite of "splitting").

We have called for prospective longitudinal research with large populations, early identified (or high-risk) subjects, competing and more transactional theories of etiology, and ever-improving diagnostic criteria. Even though this is a tall order, the research literature on borderline disorders of childhood is young and growing quickly. Greater knowledge of the etiologies and specific dysfunctions of these disorders will guide enhanced treatment (and possible preventive) efforts for these puzzling and troubled children.

ACKNOWLEDGMENTS

We would like to acknowledge the help we have received from our discussions with Jules Bemporad, Sheree Toth, Lew Ward, and Jennifer White. In addition, we would like to express our sincere thanks to Victoria Gill for typing this manuscript. The writing of this chapter was supported, in part, by a grant from the National Institute of Mental Health (R01-MH37960) to Dante Cicchetti.

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CHAPTER 28

Taxonomy of Major Disorders in Childhood

Patricia Howlin and William Yule

Pervasive developmental disorders, or major disorders in childhood, used to be classified as childhood psychoses. The term *psychosis* has no precise meaning but was used to describe severe conditions in which the child had a severely impaired sense of reality with little insight into his or her own behavior. That behavior is frequently different from normal behavior.

In recent years, there has been unease at using the term *psychosis* to describe the behavior of such children, partly because the conditions are less well understood than adult psychoses and partly because it has been realized that a distinction must be made between those disorders arising in infancy and those that arise in later childhood or adulthood. The former reflect a serious abnormality in development itself, and hence, the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) (American Psychiatric Association, 1980) refers to them as "pervasive developmental disorders." The latter conditions involve an actual loss of reality sense previously developed (Rutter, 1985).

Over the years, there has been a plethora of labels used to describe severe mental disorders in childhood, including *dementia precocissma, dementia infantilis, childhood schizophrenia, infantile autism, autistic psychopathy, symbiotic psychosis,* and, simply, *atypical child.* Following the view that disorders arising in infancy should be separated from those arising later, one then has to differentiate *early childhood autism* from other conditions. Indeed, it is the commonest of the severe disturbances of childhood and so will be discussed in detail in this chapter.

Of the later arising disorders, it is helpful to distinguish Asperger's syndrome from disintegrative psychosis, both of which will be considered briefly. Schizophrenia, strictly diagnosed, arises in late adolescence and follows the same pattern as the adult disorder. Other than to clarify the distinction between autism and schizophrenia, it will not be considered further in this chapter. Likewise, although the existence of childhood depression remains controversial (Rutter, Izard, & Read, 1986), it is clear that cases of manic-depressive psychoses rarely arise before late adolescence and even then are few in number (Steinberg, 1985).

Early Childhood Autism

In 1943, Kanner first described a group of children in whom the outstanding, "pathognomonic," fundamental disorder is the children's *inability to relate themselves* in the ordinary way to people and situations from the beginning

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> of life. . . . There is from the start an *extreme autistic aloneness* that, whenever possible, disregards, ignores, shuts out anything that comes to the child from the outside. (p. 43)

Because of these apparently "inborn autistic disturbances of affective contact" Kanner named the syndrome "early infantile autism."

In addition to the social abnormality, Kanner (1943) described a number of other distinctive features. One was the failure to develop normal communicative language and another was described as "an anxiously obsessive desire for the maintenance of sameness, resulting in marked limitation in the variety of spontaneous activity" (p. 37). Play, too, was characterized by a preoccupation with stereotyped repetitive activities lacking any creative social function. In addition, children showed a variety of nonspecific pervasive problems, including temper tantrums, fears and phobias, and sleeping and behavioral disturbances. Kanner considered all the children to be of basically normal IQ (a finding that has not been subsequently substantiated) although he did note the existence of particular patterns of cognitive functioning. Manual dexterity was generally good, but although the children were very skilled at assembling items, such as complex jigsaw puzzles, they showed no functional use of even simple toys. Their rote and long-term memory was frequently excellent, although they often showed little understanding of the facts that they repeated back. Their musical and computational skills were often striking, although they were quite incapable of dealing with even the simplest abstract tasks.

In the forty years since the condition of autism was first delineated, many hundreds of children with this condition have been diagnosed, and Kanner's descriptions ring as true today as they did then (Howlin & Rutter, 1987).

Characteristics of Autism

Impaired Social Relationships

The deviant social development of autistic children is apparent even in the early months of life. They do not develop strong specific attachments to their parents nor do they show the usual delight and anticipation if familiar adults approach. As toddlers, they tend not to follow their parents around the house nor do they become upset if separated from them. They rarely seek out their parents for comfort, and there is a general failure to use smiling, gesture, or physical contact either to respond to or signal social intent. Eye contact, too, is abnormal, and many children fail to discriminate between adults, so that they will approach a stranger almost as readily as their own parents. They do not necessarily withdraw physically from people. They may respond happily to gross physical contact, such as rough-and-tumble games or being tickled, but they rarely initiate contacts of this or any other kind. They may attempt to gain adults' attention in order to meet their immediate needs, but they will rarely attempt to share their own experiences with others.

As they grow older, some of these grosser abnormalities disappear, and most children develop strong attachments toward their parents; nevertheless, serious social difficulties persist. In particular, there is a lack of reciprocity and social responsiveness in peer interactions. Autistic children rarely engage in cooperative group play nor do they develop specific friendships. There is a curious lack of empathy in their response to other people, and frequently they seem insensitive to other people's feelings, even if these are expressed quite strongly. There is very little reciprocity in social interactions and very little responsiveness to the needs and concerns of others. In addition, the inability to interpret or appreciate nonverbal social cues tends to persist well into adulthood and leads to a failure in appreciating the demands of social situations, often resulting in embarrassing or unacceptable behaviors in public (Rutter, 1985).

Language Deficits

Abnormalities of language are frequently reported by parents as being the first problem to give concern. Early babbling tends to be affected so that the sounds made by autistic infants are restricted both in range and frequency, and the speech cadences that usually develop by the age of 9 to 12 months do not appear. The child fails to take part in the reciprocal "prelinguistic conversations" that characterize normal mother–infant interaction, and there is a failure either to respond to or initiate verbal contact. Understanding of spoken language is usually markedly delayed, and a lack of symbolic gesture or mime is equally characteristic.

Although comprehension skills tend to improve with time, older children may still fail to respond to simple instructions if given in an unfamiliar context. Complex instructions continue to present many difficulties, and problems in dealing with abstract concepts generally persist throughout adult life.

Not only are autistic children unusually de-

layed in their acquisition of speech, but their *pattern* of language development is also highly abnormal, with probably the most characteristic feature being their failure to use speech for social communication. There is none of the normal chatter or "to-and-fro" quality of conversations that characterize the speech of young normal children. The development of generative language is delayed, and repetitive and stereotyped utterances take the place of novel and creative ones. Delayed and inappropriate echoing is common, with a variety of characteristic abnormalities, such as pronoun reversal and abnormal, egocentric use of language.

Many autistic children, especially if they are severely mentally retarded, never develop speech. Some children, particularly those of normal intelligence, do achieve relatively good levels of language competence, at least as measured by syntactic skills. Nevertheless, many abnormalities in language usage remain. Speech continues to be repetitive and stereotyped with conversations often being dominated by obsessive questioning or their particular preoccupations. Difficulties with complex and abstract language concepts are also evident. There are abnormalities, too, in the delivery of speech, which is often monotonous in tone and lacking in cadence and inflection. Sentence structures tend to be overformal and pedantic, and the understanding of nonverbal cues, such as the gestures and facial expressions of other people, also remains severely affected (Rutter, 1985).

Abnormal Play Patterns

Just as language processes are stereotyped and impoverished so, too, play is ritualistic and lacking in imagination. Play patterns are rigid and limited, with little variety or creativity. There is little of the normal enjoyment in dolls or trains or cars nor is there any evidence of the inventiveness shown by normal children in making playthings out of virtually any object. Instead, involvement with toys or household objects is limited to stereotyped activities, such as spinning wheels, dismantling objects, or arranging them in complex patterns or endless straight lines.

This lack of imaginative functioning not only affects the autistic child's ability to take part in group games but it also inhibits the development of abstract symbolic functioning. Without the development of imagination or fantasy, the day-to-day life of autistic children remains concrete and steeped in routine.

Obsessional and Ritualistic Behaviors

In place of normal play patterns, autistic children tend to exhibit highly stereotyped and ritualistic responses to their environment. A resistance to change and a marked dislike of alterations in daily routine are common. This dislike of change frequently extends to the physical environment so that household utensils must always be left in a particular position, doors left open at a particular angle, or curtains hung in a particular way. Not only do the child's own activities become highly routinized, but he or she may also insist that other people in the house behave in very rigid ways.

As autistic children grow older, these rituals often become even more pervasive and can cause considerable disruption to family life. They frequently become more insistent on other people taking part in their routines, and verbal rituals become particularly pronounced. The child may refuse to let members of his family use certain words or may insist that they take part in frequent and often prolonged question-and-answer routines, with exactly the same questions asked and exactly the same answers given on every occasion.

Obsessional Interests and Attachments

A fascination with unusual objects or with unusual aspects of objects is common with autistic children. For example, they may become obsessed with touching every lamppost, garden gate, or even door handle that they see while out walking. They may be fascinated by the feel of people's hair or clothing, although showing little interest in them as people. Even if they show an interest in more normal objects, such as teddy bears or cars, their use of such objects is highly abnormal. Collecting, spinning, or lining up objects are common, and they will become extremely distressed if these activities are disturbed in any way. In later childhood, many autistic children also develop unusual preoccupations that are followed to the exclusion of other activities. Frequently, these involve such topics as bus routes, plane or train timetables, directions, numbers, or colors and patterns. Marked attachments to objects are also common, but these are not the cuddly teddys or blankets of normal children. Instead the attachment tends to be to much more bizarre objects, such as bits of glass, a piece of leather belt, or torn-up picture postcards. These objects may be carried everywhere by the child, and their loss or removal results in extreme distress. The way in which objects are handled may be extremely

stereotyped. They may be carried in a very precise manner or held at a particular angle at the side of the child's face. Stereotyped and repetitive movements are common in the absence of attachment objects. These include hand-and-finger mannerisms, such as flicking or flapping, but whole body movements, such as spinning and rocking, also occur (Rutter, 1985).

Cognitive Abnormalities

Deviant patterns of development, rather than simple delays, are also characteristic of the cognitive functioning of autistic children. Their intellectual abilities also vary widely. A small proportion of autistic children have IQs in the normal to above average range, but over half are moderately to severely retarded. Kanner (1943) believed that this poor cognitive performance was a reflection of the child's social withdrawal, but subsequent research suggests that this is not the case. Early home movies of autistic children (Rosenthal, Massie, & Wulff, 1980) show that autistic children's sensorimotor intelligence is impaired even during the early infancy period. Clark and Rutter (1977) and Volkmar, Hoder and Cohen (1985) also confirm that low IQ cannot be explained by lack of motivation or social withdrawal. The *patterning* of IQ scores, however, tends to be different from both normal children and those suffering from mental retardation. Even in children whose memory and visual and spatial abilities are relatively intact there tend to be marked deficits in tasks involving symbolization, abstraction, and conceptual processing. There are particular difficulties in tasks that require the processing of complex temporal sequences; in tasks that require the use of meaning in recall; and, in particular, on any tasks involving the interpretation of social or emotional cues (Weeks & Hobson, 1987). Functioning on a whole range of cognitive tests is also characterized by highly rigid and stereotyped response patterns (see Frith, 1971, 1972; Hermelin, 1976; Hermelin & Frith, 1971).

Age of Onset

It is now recognized (Rutter, 1985) that early onset before 2½ to 3 years is one of the diagnostic criteria for early infantile autism. This fact makes it possible to distinguish autistic children from a smaller group who initially develop normally and then, after the age of 3 to 4 years, show a marked regression in their development with a loss of speech, bowel control, and motor skills, and the progressive development of severe behavioral, emotional, and social disorders. These disintegrative psychoses are usually associated with organic and progressive brain damage.

Prevalence of Early Infantile Autism

A number of epidemiological studies in Great Britain (Lotter, 1978; Wing & Gould, 1979), Denmark (Brask, 1967), Sweden (Gillberg & Schaumann, 1982), and the United States (Treffert, 1970) are in agreement in showing that autism occurs in about 2 to 4 children in every 10,000. However, if severe mental retardation with some autistic features is included, the rate rises to as high as 20 per 10,000 (Wing & Gould, 1979). In common with most conditions involving language delays, autism is more frequent in boys than in girls, with a sex ratio of approximately 3 : 1. Recent research (Lord, Schopler, & Revick, 1982; Tsai, Stewart, & August, 1981) suggests that although autism is less common in girls, if girls are affected, they tend to be much more globally handicapped and also are more likely to have a family history of cognitive problems.

The Causes of Autism

In his earliest writings, Kanner (1943) considered autism to be a constitutionally determined developmental disorder and noted

> We must . . . assume that these children have come into the world with an innate inability to form the usual biologically provided affective contact with people, just as other children come into the world with innate physical or mental handicaps. (p. 43).

At the same time, however, he also commented on the lack of warmth shown by many parents and their tendency toward a "mechanization of human contacts."

As time went on, Kanner's emphasis on the primary biological nature of the disorder took second place to theories stressing psychogenic origins. In 1954, Kanner wrote:

> It should not be forgotten that the emotional refrigeration which children experience from . . . parents cannot but be a highly pathogenic element in the patient's early personality development. (p. 74)

Subsequently, a host of environmental influences and psychogenic factors have been suggested as possible contributory factors in the causation of infantile autism. Such authors as Bettelheim (1967),

Boatman and Szurek (1960), and O'Gorman (1970) and many others have suggested variously that autism is due to a lack of stimulation, parental rejection, lack of parental warmth, or intrapsychic conflict resulting from deviant family interactions. For a time, such theories were widely accepted throughout much of Europe and America and had a direct influence on the types of treatment offered to the families of autistic children.

Subsequent research has failed to support arguments that autism is due to abnormal patterns of family functioning. These early theories of psychogenic causation were derived either from anecdotal clinical observations or very poorly controlled studies involving inadequately diagnosed groups of children. Cantwell, Rutter, and Baker (1978) found no evidence that the parents of autistic children suffered from any greater emotional or personality problems than did the parents of other groups of children. Environmental stresses were no more or less common than in other families; no general abnormalities in patterns of family interaction were found, nor was there any specific psychopathology related to the autistic child. Moreover, although it had been claimed that autism was due to psychosocial factors, the characteristic behaviors of the autistic child proved very different from those shown by children who *had* suffered from physical or emotional abuse or neglect (Rutter & Lord, 1987; Skuse, 1984). Studies, such as that by Massie (1978), indicate that mothers of autistic children may interact less successfully with their infants, but these findings fail to take into account the reciprocal nature of the parent–child interaction. Rosenthal *et al.* (1980) showed that many of the infants in the Massie study were already showing cognitive abnormalities, and the likelihood that the apparent deviance in parental behavior may have resulted from the pathology of the child, rather than vice versa, could not be ignored.

Psychogenic theories, too, have failed to explain the highly specific nature of the deficits shown in autism, particularly the extreme unresponsiveness to social contact that is apparent in the early weeks of life. The association with organic factors, including the high rate of perinatal problems, the development of epilepsy in early adolescence, and other evidence of neurological or neurochemical disturbance, conflicts with theories that stress the parents' role in etiology.

Admittedly, evidence from neurological studies of various kinds has failed to produce any consistent or conclusive evidence about the specific causes of autism. Neurophysiological investigations and studies of organic pathology or biochemical abnormalities have given rise to a whole host of theories about the possible causes of the disorder. However, as Rutter (1985) pointed out, such studies are fraught with problems. Many findings have been inconsistent or not replicated. In many, control groups are lacking or are inadequate (so that it is unclear whether the results reflect low mental age or brain abnormalities associated with mental retardation rather than with autism *per se*). There are also serious problems in interpreting the ways in which the abnormalities found may exert their effects.

Although neurophysiological and biochemical studies have failed to find any direct links with autism, in recent years positive evidence of hereditary influences has emerged. First, it is evident that the rate of autism in siblings of autistic children is 50 times that of the general population (Rutter, 1985). Second, although a family history of autism is rare, a family history of speech delay is much more common, and the siblings of autistic children are far more likely to have language disorders, learning disabilities, or mental retardation than the siblings of other handicapped children (August, Stewart, & Tsai, 1981; Bartak, Rutter, & Cox, 1975). In addition, Minton, Cambell, Green, Jennings, and Samit (1982) found that the siblings of autistic children had lower IQs than expected on the basis of demographic variables and that the deficits were more evident on verbal IQ scores than on performance tests. Third, the study of autistic twins by Folstein and Rutter (1977) showed an extremely high concordance rate for cognitive abnormalities in monozygotic twins. All these findings indicate the presence of important genetic influences, although they suggest that it is not autism as such that is inherited but rather some broader predisposition to language and cognitive abnormalities of which autism constitutes but one part. Recently, too, a number of studies (Blomquist *et al.*, 1985; Brown, *et al.*, 1982; Levitas, Hagerman, Braden, Rimland, McBogg, & Matus, 1983) have indicated that some cases of autism are associated with the fragile-X syndrome. However, the results have often been inconsistent and the studies so poorly controlled that it remains unclear whether this anomaly has any specific association with autism or is predominantly related to general mental retardation.

Later Outcome

Follow-up studies of autistic children indicate that although improvements in language and personal relationships occur as they grow older, many

abnormalities remain. About one half of autistic children eventually gain useful speech, but there are usually persistent abnormalities in language usage and speech delivery. In particular, there are continuing and often widespread difficulties in dealing with concepts involving abstract, emotional, or interpersonal features.

Social awareness tends to improve considerably in late adolescence, and this is accompanied by an increasing awareness of the individual's own problems. However, although many older, autistic people express a desire to socialize and to make friends, their ability to do this is severely limited by their lack of empathy with, and sympathy for, other people's feelings and emotions.

Total social independence is gained by only a very small minority of autistic individuals. Prognosis is strongly influenced by the extent of the initial handicap, with IQ and language skills being the most powerful predictors of later outcome. Only those children who develop useful verbal skills *and* have IQs in the normal range achieve relatively acceptable levels of social adjustment in adult life. Among this group, approximately 50% will fail to achieve a satisfactory social outcome and, even among those who do manage to find jobs and live independently, the level of occupation attained is generally much lower than their IQ or educational training would predict. The factors that are important in distinguishing between those autistic children who do and do not make satisfactory progress as they grow older have still to be identified.

A further finding from follow-up studies is the relative frequency with which epileptic seizures occur for the first time during adolescence. Approximately one quarter of autistic children develop seizures by 18 years (Deykin & MacMahon, 1979; Rutter, 1970). Not only is the rate of seizures much greater than that found in the general population, but the age of onset of seizures is also very unusual. The onset of seizures in nonautistic children usually occurs in the early years of life, with the risk *decreasing* as they reach adolescence. In contrast, in autism, the peak period for onset is between 11 and 14 years. Deykin and MacMahon (1979) suggested that the high incidence of seizures at puberty may be specific to autistic children and may represent a distinct pathological process. However, it should be noted that the risk of seizures is several times higher in mentally retarded autistic children than in those children of normal nonverbal intelligence. Again, any specific relationships between autism and epilepsy still have to be demonstrated.

The Classification of Autism

In the forty years since Kanner's classic description of autism, there have been many attempts to improve the diagnosis. The British Working Party on Schizophrenia Syndrome in Childhood (Creak, 1961) described a cluster of nine points basic to "childhood psychoses." These included gross impairment of emotional relationships, absence of speech, and preoccupation with objects. In the United States, Rimland (1968) produced a checklist of over 300 items of behavior. In 1970, Rutter pointed out that the various checklists of symptoms could be reduced to three main groups. This cluster of symptoms would be characteristic of all autistic children but would not be found in other psychiatric disorders of childhood. These diagnostic criteria, which are now central to the DSM-III classification, are:

1. Impaired social development that is out of keeping with the child's chronological and intellectual level and which shows a number of specific characteristics, including a relative lack of selective attachments, poor use of eye-to-eye gaze to regulate social interactions, a lack of social reciprocity, and a failure to form selective peer relationships
2. Delayed and deviant language that is out of keeping with the child's mental level and that involves deficits in language comprehension, abnormal use of language for social communication purposes, together with serious impairments in nonverbal communication and creative imagination
3. Stereotyped and repetitive behaviors as shown by rigid play patterns, abnormal preoccupations, unusual routines or rituals, abnormal attachments to objects, and protests over environmental change

A fourth criterion is that the condition should be apparent by 30 months of age. There is some dissent over the inclusion of this condition, and many would argue that it should not be used as an exclusionary diagnostic criterion (Cohen, Paul, & Volkmar, 1986). However, the purpose is to distinguish autism from those degenerative disorders in childhood in which a period of obvious normal development is followed by a sudden and marked loss of previously well-established skills.

The Differential Diagnosis between Autism and Other Childhood Disorders

Given the early confusions in terminology and diagnosis and the very different theoretical backgrounds of child psychiatrists, it is perhaps not surprising that difficulties have arisen in distinguishing autism from other childhood disorders. However, autism clearly differs from other conditions, not only in the constellation of core symptoms but also with regard to family history, possible causative factors, patterns of cognitive impairment, responsiveness to treatment, and general prognosis.

Autism or Childhood Schizophrenia?

Confusion between autism and schizophrenia in later childhood has arisen not only because of the problems in terminology. Kanner (1954) noted the close resemblance between schizophrenic withdrawal and that found in autism and believed that "there is no likelihood that early infantile autism will at any future time have to be separated from the schizophrenias" (p. 55). Theories of psychogenic causation also strengthened the view that autism was an early form of schizophrenia. Such authors as Singer and Wynne (1963) and Mishler and Waxler (1963) had attributed the emergence of schizophrenia in adolescence to abnormal patterns of interaction. As noted above, deviant patterns of family functioning were also implicated in the causation of autism. However, it is now apparent, on the basis of many different findings, that autism cannot be considered as a form of early onset schizophrenia.

First, the symptomatology is quite different. Autism comprises a failure of development, whereas schizophrenia involves the emergence of abnormal thought processes after a period of apparently normal development. Moreover, the characteristic delusions and hallucinations found in schizophrenia are extremely rare in autism, even in adult life. It is true that some autistic individuals may hold strange beliefs bordering on delusions (Volkmar & Cohen, 1985). However, as Wing (1981) points out, careful assessment of these beliefs usually reveals that they are associated with the individual's obsessional interests or caused by a misunderstanding of social cues rather than being truly delusional in nature. Furthermore, although many autistic children would satisfy at least some of the criteria for schizophrenia, on the basis of their illogical thinking, poverty of speech, inappropriate affect, and social isolation, the quality of these problems is very different in the two groups. Moreover, such symptoms are apparent very early on in the development of an autistic child. In schizophrenia, they do not emerge until much later, usually the mid- to late-teens or early adulthood. This *absence* of the key symptoms of schizophrenia holds true for autistic adults.

The reverse also holds true: adult schizophrenic patients do not have histories of infantile autism. As far as causation is concerned, investigations into schizophrenia tend to have been dogged by the same problems as those seeking the causes of autism. A variety of biochemical and neurological abnormalities have been described, but many studies have been poorly controlled. Few causal relationships have been demonstrated between the abnormalities that were identified and the emergence of schizophrenia so that the interpretation of many of these findings is still in doubt. Of course, genetic factors are implicated in schizophrenia as they are in autism, but, as noted below, the family history is very different in the two conditions. Moreover, environmental factors seem to play an important role in the emergence of schizophrenia, but they have no impact on the development of autism.

Second, family characteristics are markedly different in the two conditions. Schizophrenia is rare in the parents or siblings of autistic children, whereas it occurs in about 10% of the first-degree relatives of schizophrenic individuals. Although there is an increased risk of autism or other learning disabilities in families with an autistic child, this is not the case in the families of schizophrenic children. Also, there seems to be some tendency for the parents of autistic children to be of higher than average social class, whereas the parents of schizophrenics show more or less normal social class distribution.

Third, the sex distribution is very different in the two conditions. Overall, schizophrenia occurs as frequently in males as in females, but autism occurs at least three times as frequently in males as in females. Organic factors, too, differentiate the two conditions. Perinatal complications and epilepsy are far more common in autism (Deykin & MacMahon, 1979), and if epilepsy does occur in schizophrenia, it is usually of a different type (typically of temporal lobe origin) and has a different age of onset than in autism.

Fourth, there are striking intellectual differences between the two conditions. Mental retardation, which is commonly associated with autism, is much less frequently associated with schizo-

phrenia. Nor do schizophrenics show the typical patterns of cognitive dysfunctioning that are found in autism.

Fifth, the response to treatment is very different. Although many of the drugs and physical treatments (such as electroconvulsive therapy) that have been used in the treatment of schizophrenia have also been employed in the treatment of autism, their effectiveness tends to be very different. Campbell (1978) suggested that drugs which may be beneficial in the treatment of autism can result in deterioration in schizophrenia and vice versa. In the treatment of schizophrenia, medication tends to have relatively specific antipsychotic effects, and there is good evidence that drug maintenance is crucial in preventing relapses. There is no such effect in autism. Medication can be useful in reducing some of the nonspecific symptoms associated with autism, such as overactivity and sleeping problems, but, in general, drug treatments are far from being essential and do very little to affect the basic disorder.

Finally, the course and prognosis of the two conditions differ greatly. Autism is apparent in the first or second year of life and although the early social and communication difficulties may show some improvements, major abnormalities in these areas tend to persist. Occasionally, there may be deterioration in some individuals around adolescence, but otherwise autism tends to follow a fairly steady course. In contrast, schizophrenia begins much later in life, and remissions and relapses are common. If medication is effective, many schizophrenics are able to function relatively successfully for the greater part of their lives. However, this is not the case for autistic individuals for whom the prognosis is much less positive.

Autism as a Form of Mental Retardation

Kanner's initial claim that autistic individuals were of normal or above average intelligence has not been substantiated by later research. As already noted, at least one half of all autistic children have IQs in the mildly to severely retarded range and only a relatively small number are of normal intelligence. The fact that so many children are retarded has led practitioners to suggest that autism is simply a "fancy" title for retardation and that it is used particularly by parents in the upper social class brackets who do not wish their children to be stigmatized in this way. However, it is clear that such views are totally at variance with the available evidence. Although autistic and mentally retarded children share a variety of nonspecific problem behaviors, such as behavioral disturbances, stereotyped and manneristic behaviors and problems in self-help skills, nonautistic but mentally retarded children do not show the core symptoms of deviant social and language development. Instead, although their language and social development may be delayed, development in these areas is generally in keeping with their nonverbal mental age. Also, they fail to show the characteristic patterns of obsessional and ritualistic behavior that typify autism.

Family patterns, too, differ. Mild mental retardation tends to have a strong family history, with many family members also showing below average IQ. In contrast, the IQ of families with a severely mentally retarded child tends to be similar to the normal population. Neither of these two patterns is characteristic of the families of autistic children.

In terms of causation, it is more difficult to differentiate between the two conditions. In a minority of mentally retarded children, the cause can be attributed to specific biochemical, organic, or genetic abnormalities. In the majority of cases, as with autism, the cause remains unknown. Neither autism nor mental retardation is a unitary syndrome with a single biological cause. Instead, a variety of pathological mechanisms, acting in very different ways, may produce the same end results.

Cognitively, too, there are many differences between autistic and nonautistic children even if they are matched for overall mental age. In young autistic children, there is almost always a marked discrepancy between verbal and nonverbal skills, with verbal abilities being relatively much more delayed. This difference does not necessarily hold true for older autistic children but, nevertheless, very specific patterns of cognitive dysfunction can still be found. There are marked deficits in skills involving sequencing, abstraction, semantic meaning, and particularly those skills involving the use of socioemotional cues. Response patterns on a wide range of cognitive tests also tend to be very rigid and stereotyped. Mentally retarded children of similar developmental levels do not show this characteristic pattern of cognitive deficit. They are, by definition, globally retarded in all aspects of their development. They do not show the very uneven pattern of skills and disabilities that is apparent in autism.

Finally, the outcome tends to be rather different in the two groups. The prognosis for mentally retarded children depends very much on the level of their handicap. Children with mild retardation are

very likely to live more or less independent lives, to form strong emotional attachments to other people, and to earn their own living. Outcome in autism is also dependent on IQ; but even among the most mildly cognitively handicapped, prognosis is generally much less positive than in nonautistic individuals of similar IQ levels.

Autism and Severe Developmental Language Disorders

Apart from language delay, children with severe developmental language disorders, especially those affecting receptive skills, share a number of problems with autistic children, thus making differential diagnoses difficult. For example, both groups show social and communication difficulties; however, the nature and quality of the social and language impairments differ.

Although unable to speak, dysphasic children make successful attempts to communicate nonverbally and respond to nonverbal cues and gestures. Their verbal comprehension is generally much higher than that of autistic children of comparable levels of intelligence. Dysphasic children tend to show better imaginative play, and their early attachment behavior is relatively normal. At later ages, dysphasic children may show some difficulties in peer relationships and some rituals, but these difficulties are generally much milder than those seen in younger autistic children.

The family history shows some similarities, with a higher than normal rate of occurrence of language delays in families of both groups. Causation is generally unclear, although, as with autism, a variety of abnormalities has been associated with the condition. Chromosomal abnormalities have been found in some individuals, and Lewis and Mezey (1985) found lesions in the cavum-septum pellucidum in a series of older dysphasic individuals. However, again, the way in which these abnormalities may exert their effects is unknown.

Although cognitive deficits differ between autistic and dysphasic children, there tend to be a number of specific deficits found in this group. The condition is not associated with global mental retardation, but there are particular difficulties in such areas as hierarchical planning abilities (Cromer, 1981), sequencing (Poppen, Stack, Eisenson, Forrest, & Wertheim, 1969), representation (Kahmi, Catts, Koenig, & Lewis, 1884), associative imagery, and short-term memory (Eisenson, 1968; Graham, 1970). In terms of outcome, the picture is unclear. It has generally been accepted that the problems shown by dysphasic children tend to show much greater improvement as they grow older. Bartak, *et al.* (1975), for example, found that even when matched for language level and nonverbal IQ, autistic children did comparatively badly on a wide range of skills, particularly those involving social communication. A follow-up of the same children in middle childhood indicated that in some ways the two groups were *less* dissimilar than they had been originally. Many differences continued to exist, with the dysphasic group being generally less handicapped than the autistic children, but in a number of ways they showed fewer improvements than had been expected (Cantwell, Baker, Rutter, & Mawhood, 1989). Subsequently, these results have been confirmed by a number of other studies that indicate that the prognosis for dysphasic children, and even children with very mild language handicaps, is much less positive than had been originally thought (Paul, Cohen, & Caparulo, 1983). Even if the initial language deficits improve, many children show persistent learning difficulties, and their social relationships, particularly with peers, are often impaired. Clinical observations of children in the original autistic/dysphasic comparison, who are now in early adulthood, indicate the persistence of often gross social abnormalities in many of the dysphasic individuals. A number of cases (including those with a septum pellucidum lesion) have also been diagnosed as having some form of adult psychosis (Cantwell *et al.*, 1989).

The nature of the relationship between autism and developmental dysphasia is therefore somewhat unclear. Although up to now they have been viewed as very distinct conditions, it seems that, in some ways, the similarities between the groups may increase rather than decrease as individuals grow older. A further detailed study of these individuals is currently underway, but the possibility that the two conditions may be less distinct, both in terms of outcome and genetic background than was originally thought, cannot be dismissed.

Autism and Asperger's Syndrome

Shortly after Kanner's description of autistic children in 1943, Asperger (1944) outlined a similar syndrome that he termed *autistic psychopathy*. The children that he described were all boys, and although they were not necessarily as delayed in their language development as autistic children, all had shown consistent social and communication ab-

normalities from the second year of life. Abnormalities of gaze, poverty of nonverbal expression, a lack of empathy with others, solitariness, lack of humor, extreme egocentrism, unusual and restricted intellectual interests, and bizarre attachments to objects were among the main phenomena described.

A rather similar group of children was described by Wolff and her colleagues (Chick, Waterhouse, & Wolff, 1979; Wolff & Barlow, 1979; Wolff & Chick, 1980), but they preferred to refer to the condition as "schizoid personality." Again, they showed a very similar pattern of problems in their group to the Asperger children, although the authors stressed the links with schizophrenia of an adult kind rather than with childhood autism. Abnormalities of personality in childhood may be precursors to adult schizophrenia, but only one "schizoid" individual in their follow-up study of 92 boys actually developed a schizophrenic-type psychosis. Moreover, schizophrenia does not show the huge male preponderance that was reported for Asperger's syndrome or the schizoid group studied by Wolff *et al.*, and it is not more common in those from middle-class backgrounds (as was the case in Wolff's studies).

At present, there is considerable controversy as to whether the conditions described as Asperger's syndrome or schizoid personality should be considered clinical entities in their own right or whether they are simply synonymous with mild cases of autism. Certainly, if one compares Kanner's early accounts with Asperger's original paper, there is very little reason to distinguish between the two groups (Howlin, 1987). The parallels with autism are obvious in terms of the behavioral characteristics, sex ratio, and adult outcome. Although language is not necessarily delayed in either Asperger's syndrome or in the schizoid group, the use of language for communicative purposes is highly abnormal. Deviant use of language, in the absence of any marked delays in acquiring first words, has been reported in a number cases of autistic children of normal or above levels of intelligence.

The relationship between Asperger's syndrome or schizoid personality and schizophrenia is also unclear. Although there are many differences between these conditions, it is known that there are abnormalities of personality that are genetically linked with schizophrenia and that these have certain similarities with some features of Asperger's syndrome. Developmental delays of various kinds (although not necessarily related to language) are also known to be possible precursors to schizophrenia.

Although there is no doubt that children with the features said to characterize Asperger's syndrome or schizoid personality exist, the nosological validity of the hypothesized syndromes is unclear. The possible links between an abnormality of personality and a severe pervasive disorder of development (or less probably with schizophrenia) are worthy of much greater research investigation. The genetic bases to these conditions also require further study. At present, however, much more detailed retrospective and prospective studies of such children are required before any firm conclusions can be drawn about the fundamental nature of the disorder.

Disintegrative Disorders

The criteria for the diagnosis of autism stress the fact that the condition should be apparent from the age of around 2 to 3 years. This is to distinguish autism from a number of other although rarer conditions in early childhood in which a period of normal development is followed by regression and behavioral deterioration, which often gives rise to problems similar to those found in autism. Heller (1930) described a condition that he named "dementia infantilis." In this condition, development usually appears normal or almost normal to the age of 3 or 4 years, followed by a marked deterioration in many areas. Often there is a preliminary period of vague illness during which the child may become restive, irritable, anxious, and overactive. Subsequently, over the course of the next few months, speech and language tend to deteriorate. Comprehension decreases and intelligence skills often show a decline, although a normally intelligent facial expression is retained. There is a loss of social skills, withdrawal from interpersonal contact, and a general loss of interest in objects. Stereotypies and mannerisms also develop. Sometimes this condition is linked with an attack of measles-encephalitis, or some other organic illness affecting the brain. In most cases, however, there are no clinical signs of neurological damage. Some postmortem studies have revealed cortical degeneration, but in others there is no evidence of brain disease or damage. The general assumption is that the disorder is caused by some form of organic brain disease whose exact etiology remains unknown.

Although the onset in disintegrative disorders and autism is very different, the clinical picture in the two groups of conditions shows many similarities. Rutter (1985) suggested that some cases may represent atypical forms of autism, whereas

others stem from acquired brain disease. It is likely that the group of disintegrative disorders will prove to be heterogeneous with some being related to autism and others unrelated. Rutter suggested that, for the moment, it seems highly desirable to retain the separate entities because of the lack of knowledge about etiology and also because the nature and extent of the overlap between autism and degenerative disorders has yet to be determined.

Generally, the prognosis for children in this group of conditions is poor, with most remaining severely mentally handicapped and without speech. Often a plateau of development is reached after which there is no further deterioration. However, in the case of many lipoidoses and leukodystrophies, there may be progressive deterioration leading to death.

Another, and also rare, syndrome, which may occasionally be difficult to differentiate from autism or disintegrative psychoses, is the condition of acquired aphasia with convulsions (also known as the Landau Kleffner syndrome) (Landau & Kleffner, 1957). In this condition, social and language development are normal in the early years but then, over a period of weeks or months, both receptive and expressive language skills are lost. In most cases, there are a few seizures and generalized EEG abnormalities at the time of onset, but usually these do not persist. Work by Bishop and Rosenbloom (1987) and Bishop (1979) has shown that the children show very deviant patterns of language comprehension as well as impairment in their spoken language. In some cases, language skills are recovered, but usually they remain substantially impaired. Nevertheless, the children do not show general intellectual deterioration nor are there any related problems of social development. However, the very frightening experience of suddenly being unable to talk or understand may lead to social withdrawal and behavioral disturbances that, initially, may be rather similar to the problems found in autism.

A third condition giving rise to many of the behaviors typically associated with autism is that of Rett's syndrome, first described in Vienna in 1966. It was not until 1983 that it emerged that the syndrome had also been reported previously in several European countries, although it had not been recognized as a specific entity. Hegberg, Aicurdi, Dias, and Ramos (1983) described a series of 35 cases, all female, in which neurological and mental development during the first 12 to 18 months of life was quite normal. Subsequently, there was a period of "stagnation" when no new skills were acquired, and then a rapid deterioration of behavior and mental abilities. Interpersonal contact with people and objects became either absent or very limited. Fears of unfamiliar situations were pronounced, and motor activity became stereotyped and repetitive. In particular, there were characteristic "handwashing" movements as well as other facial mannerisms, teeth grinding, and grimacing. Many motor skills were lost so that children were unable either to walk or sit unaided. There was also marked slowing of head circumference growth, resulting in an acquired microcephaly. A jerky ataxia was also frequently apparent.

After the initial period of rapid deterioration, which usually took place within 18 months of the first signs of the disorder, there was a slowing down in progressive neurological abnormalities. However, most subjects remained severely motorically handicapped, epileptic seizures were common, and the abnormal social and autistic features remained, although they tended to become less prominent as the subjects grew older. Again, although it is presumed that the syndrome is caused by a progressive degenerative disorder of the brain, the nature and cause of the progressive deterioration remain unknown. The confinement of the syndrome to girls in all known cases also adds to the puzzle about causation, and although some genetic explanations have been put forward (Hegberg *et al.*, 1983), these are not entirely satisfactory.

Concluding Comments

There are two main peak ages when major disorders of childhood first manifest themselves: before 2½ years and after puberty. The former set of disorders is largely accounted for by early childhood autism, whereas the latter set consists mainly of adult-type schizophrenia. In between, there is the small, but extremely distressing, group of disintegrative psychoses.

It is now widely accepted that early childhood autism is a pervasive developmental disorder that differs radically from adult schizophrenia. The earlier held psychogenic theories of etiology are now largely discredited. There is increasing evidence that the wide variety of cognitive deficits is related to underlying biologically and physiological disturbances, but the exact etiology has yet to be established. Educational treatment along structured, behavioral lines is the most promising approach in therapy, but the overall prognosis for autistic children remains poor, with fewer than one in six eventually being able to enjoy independent lives as adults.

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CHAPTER 29

Autism and Pervasive Developmental Disorders

Margaret E. Hertzig and Theodore Shapiro

Origins and Classification

In 1943, Kanner published a report on 11 children who displayed such symptoms as extreme social withdrawal, idiosyncratic and delayed use of language, restriction of interests, and insistence on sameness which he believed constituted a specific syndrome to which he gave the name "autistic disturbances of affective contact." Although Kanner was not the first researcher to describe severe distortions of developmental processes occurring in very young children (DeSanctis, 1906; Heller, 1930; Maudsley, 1867; Potter, 1933), his particularly clear and incisive clinical report has shaped the direction of clinical investigative activity for close to half a century.

Although Kanner recognized that early infantile autism (EIA) (Kanner, 1944) was similar to schizophrenia, as it was then thought to occur during childhood, he considered his cases unique because the extreme aloneness of such children was evident from the very beginning of life. He also suggested that these children were of normal intelligence but acknowledged that previously many had been considered to be feebleminded. He also thought that their families were characterized by coldness and formality. Nevertheless, he asserted that "these children have come into the world with innate inability to form the usual, biologically provided affective contact with people, just as other children come into the world with innate physical or intellectual handicaps" (p. 43). The major symptoms of impaired communication, rigidities, stereotypies, and insistence on sameness developed in the service of "[an] all-powerful need for being left undisturbed" (p. 36). Thus, Kanner set the stage for controversy about the syndrome, its relation to other disorders of early childhood, and its etiology, pathogenesis, and longitudinal course.

During the next quarter of a century, as confirmatory reports appeared with increasing frequency, early infantile autism came to be thought of as the earliest form of schizophrenia (Bender, 1947; Eisenberg & Kanner, 1956). Efforts to distinguish further among severely disordered young children led to the proliferation of additional syndromes: the atypical child (Rank, 1955), symbiotic psychosis (Mahler & Gosliner, 1955), childhood psychosis (Creak, 1963), and autistic psychopathology (Asperger, 1944). However, neither of the two earlier editions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-I, American Psychiatric Association [APA], 1952) (DSM-II, APA, 1968) provided guidelines for distinguishing among them. The designation "childhood schizophrenia" in DSM-II included all conditions "manifested by autistic atypical and withdrawn behavior; failure to develop identity separate from the mother's; and

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general unevenness, gross immaturity and inadequacy in development" (APA, 1968, p. 35). This poor specificity led Rutter (1972) to conclude that the term *childhood schizophrenia* had outlived its usefulness.

The publication of the third edition of the manual (DSM-III, APA, 1980) provided a new sanction for autism. A growing body of evidence (Kolvin, 1971; Kolvin, 1971; Rutter, 1972, 1978; Rutter & Lockyer, 1967) indicating clear differences between "psychoses" beginning before the age of 5 years and those beginning later in childhood provided the basis for the establishment of diagnostic distinctions between schizophrenia and Pervasive Developmental Disorder (PDD), which includes autism. DSM-III criteria for the diagnosis of schizophrenia were the same for adults, adolescents, and those rarely occurring cases that begin before the onset of puberty. *Pervasive Developmental Disorder* was the term that was used to describe conditions characterized by distortions in the development of multiple, basic psychological functions in prepubertal children.

In DSM-III, PDD, despite its appellation as a developmental disorder, was listed on Axis I—that section of the multiaxial classification reserved for clinical syndromes—and only later in the most recent edition of the manual (DSM-III-R) was it moved to Axis II as a developmental disorder. The two disorders described under PDD in DSM-III, Infantile Autism (IA) and Childhood Onset Pervasive Developmental Disorder (COPDD), differed principally with respect to age of onset and severity of symptomatology (Cohen, Volkmar, & Paul, 1986). The criteria for IA were very similar to those originally specified by Kanner: onset before 30 months of age, pervasive lack of responsiveness to other people, gross deficits in language development, and bizarre responses to various aspects of the environment occurring in the absence of delusions, hallucinations, loosening of associations, and incoherence as in schizophrenia. COPDD included children in whom the onset of gross and sustained impairment in social relationships was after 30 months of age and who needed to display only three of the following seven symptoms: excessive anxiety, constricted or inappropriate affect, resistance to change, oddities of motor movements, abnormalities of speech, hyper- or hyposensitivity to sensory stimuli or self-mutilation, also occurring in the absence of delusions and hallucinations, and, lastly, incoherence or marked loosening of associations. Atypical PDD was to be applied to children whose multiple developmental distortions could not be classified as either IA or COPDD.

In 1987, the nomenclature in the diagnostic manual underwent further revision. DSM-III-R (APA, 1987) criteria are summarized in Table 1. In this latest edition, PDD is more properly listed on Axis II, along with other disorders of development and personality. The criteria derive from the findings of the epidemiologically based studies of Wing and her colleagues (Wing & Gould, 1979; Wing, Gould, Yeates, & Brierley, 1977) who concluded that "Kanner's syndrome [DSM-III] is not a unique specific condition, but a small segment of a spectrum of disorders" (Wing & Attwood, 1987, p. 8). Although the concept of Pervasive Developmental Disorder is retained, the characteristics of this group of disorders now include

> qualitative impairment in the development of reciprocal social interaction, in the development of verbal and nonverbal communication skills, and in imaginative activity, often accompanied by a markedly restricted repertoire of activities and interests which frequently are stereotyped and repetitive. (APA, 1987, p. 33)

The manual recognizes only one subgroup of the general PDD category: Autistic Disorder (AD), noting that this disorder is the most severe and prototypical form of PDD. Recognizing that the severity and expression of impairment can vary greatly from child to child, the criteria for AD requires the presence of at least 8 of 16 symptoms, including a minimum of 2 in the area of impaired social interaction, and 1 symptom each in the areas of communicative impairment and restricted repertoire of activities and interests. Pervasive Developmental Disorder Not Otherwise Specified (PDD NOS) is to be used when there is qualitative impairment of social interaction and communicative skills, but the criteria for AD are not met. Unlike the requirements in the DSM-III, neither onset before 30 months or the absence of hallucinations or delusions are required for the diagnosis.

Preliminary investigations contrasting DSM-III and DSM-III-R criteria indicate that the sensitivity of DSM-III-R criteria for DSM-III cases of PDD is high. However, DSM-III-R criteria are less specific. The concept of autism has been broadened to include children previously diagnosed as Atypical PDD, who, although they may display qualitative impairments of social interaction, communication, and a markedly restricted repertoire of activities and interests, are *not* pervasively unresponsive to other people (Hertzig, Snow, New, & Shapiro, 1988).

Table 1. DSM-III-R Criteria for Pervasive Developmental Disorders[a]

Autistic Disorder

At least eight of the following sixteen items are present: this must include at least two items from A, one from B and one from C.

Note: Consider a criterion to be met *only* if the behavior is abnormal for the individual's developmental level.

A. Qualitative impairment in reciprocal social interaction as manifested by the following:
   (1) marked lack of awareness of the existence or feelings of others
   (2) absent or abnormal seeking of comfort at times of distress
   (3) absent or impaired imitation
   (4) absent or abnormal social play
   (5) gross impairment in ability to make peer friendships

B. Qualitative impairment in verbal and nonverbal communication, and in imaginative activity as manifested by the following:
   (1) no mode of communication, such as communicative babbling, facial expression, gesture, mime, or spoken language
   (2) markedly abnormal nonverbal communication, as in the use of eye-to-eye gaze, facial expression, body posture, or gestures to initiate or modulate social interaction
   (3) absence of imaginative activity, such as play-acting of adult roles, fantasy characters, or animals; lack of interest in stores of imaginary events
   (4) marked abnormalities in the production of speech, including volume, pitch, stress, rate, rhythm, and intonation
   (5) marked abnormalities in the form or content of speech, including stereotyped and repetitive use of speech
   (6) marked impairment in the ability to initiate or sustain a conversation with others, despite adequate speech

C. Markedly restricted repertoire of activities and interests, as manifested by the following:
   (1) stereotyped body movements
   (2) persistent preoccupation with parts of objects
   (3) marked distress over changes in trivial aspects of environment
   (4) unreasonable insistence on following routines in precise detail
   (5) markedly restricted range of interests and a preoccupation with one narrow interest

D. Onset during infancy or childhood.

   Specify if childhood onset (after 36 months of age).

Pervasive Developmental Disorder not Otherwise Specified

This category should be used when there is a qualitative impairment in the development of reciprocal social interaction and of verbal and nonverbal communication skills, but the criteria are not met for Autistic Disorder, Schizophrenia, or Schizotypal or Schizoid Personality Disorder. Some people with this diagnosis will exhibit a markedly restricted repertoire of activities and interests, but others will not.

[a]From *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev., pp. 38–39) by the American Psychiatric Association, 1987, Washington, DC: American Psychiatric Association. Copyright 1987 by the American Psychiatric Association. Adapted by permission.

Epidemiology

Epidemiologic studies (Brask, 1967; Lotter, 1966; Treffert, 1970; Wing & Gould, 1979) conducted before the publication of DSM-III have estimated the prevalence rate of Kanner's autism to be between 2 and 5 per 10,000. Studies utilizing DSM-III criteria for Infantile Autism have essentially confirmed these estimates (Burd, Fisher, & Kerbeshian, 1987; Steffenburg & Gillberg, 1986). Although no estimates of the prevalence of Autistic Disorder as defined by DSM-III-R criteria are available, the combined prevalence of Infantile Autism and "autistic-like" conditions (apparently similar to PDD as defined in both DSM-III and DSM-III-R) have ranged from 21 per 10,000 (Wing & Gould, 1979) to 11 per 10,000 (Burd *et al.*, 1987).

Autism occurs more frequently in boys, with the estimated male to female ratio for all PDD (DSM-III criteria) being 2.7 : 1 (Burd *et al.*, 1987). Some investigations have confirmed Kanner's (1943) original observation that parents of autistic

children come from higher socioeconomic groups than the general population (Allen, DeMyer, Norton, Pontius, & Yang, 1971; Cox, Rutter, Newman, & Bartak, 1975; Kolvin *et al.*, 1971; O'Moore, 1972; Treffert, 1970). However, these findings appear to reflect patterns of referral rather than a genuine difference. More recent epidemiologically based studies (Gillberg & Schaumann, 1982; Wing, 1980) lend no support for the view that autistic children come from higher social classes.

Although autism can be associated with any level of intelligence, about 40% of children with the disorder have an IQ below 50 and only 30% score above 70 on standardized tests (Brask, 1970; DeMyer, 1976; Kolvin, Humphrey, & McNay, 1971; Lotter, 1966; Rutter, 1970; Wing, 1981; Wing & Gould, 1979).

Clinical Characteristics from Infancy to Adulthood

The longitudinal course of this developmental disorder and the range of symptomatic expression at each age stage of development are now well studied. In the great majority of cases, onset is reported before 3 years of age, although milder impairments may not be detected until school entrance (Paul, 1987; Wing & Attwood, 1987). Three onset patterns have been described: (1) "always different," (2) a period of normal development followed by a definite developmental turning point or "setback," and (3) a gradual failure to progress normally (DeMyer, 1979). The reliability of retrospective histories are difficult to assess, as accuracy is determined by parental ability to recognize subtle symptoms during infancy as well as by possible distortions of recall. No systematic relation between mode of onset and clinical picture have been observed (DeMyer, 1979; Ornitz & Ritvo, 1976).

Wing and Gould (1979) used variations in social impairment to develop a descriptive subclassification of children with pervasive developmental disorder, distinguishing among aloof, passive, and active-but-odd groups at each age stage of development. Wing (Wing & Attwood, 1987) suggested that individual socialization patterns are consistent over time. However, Paul (1987) indicated that for autistic individuals with higher levels of cognitive functioning, the progression from aloofness to passive acceptance to active-but-deviant ways of seeking interaction appears to be a more typical course. For those whose IQs are in the retarded range, the shift from aloofness to passivity is common, but active-but-odd social interactions are generally not seen.

During the preschool period, those in the aloof group are the most cut off from social contact. Unlike normal toddlers, aloof autistic children tend not to follow parents around the house, or to run to greet them if they have been away, or to seek comfort when in pain or distress. However, almost all pervasively developmentally disordered children have begun to exhibit awareness of the presence or absence of primary caretakers by 3 to 5 years of age (Shapiro, Sherman, Calamari, & Koch, 1987). Children in the passive group do not make spontaneous social approaches but do accept the approaches of others and can be led to join in games or other social activities. Those who are active but odd during the preschool period may initiate social interactions in peculiar and deviant ways, by clinging, touching, or sniffing others (Wing & Attwood, 1987; Wing & Gould, 1979).

Although all autistic children have marked difficulties in both understanding and using verbal and nonverbal communications, these impairments are most severe among the aloof group (Wing & Attwood, 1987). Understanding spoken language is significantly impaired. Lack of response to speech may lead to a suspicion of deafness, but the children often display an ability to react to sounds that seem to have meaning for them. Poor eye contact, active visual avoidance, passive facial expression, lack of social gestures, such as waving or nodding, are also noticeable. About one half of all autistic people, especially those who are also mentally retarded, remain mute throughout their lives. When speech does develop, it is usually preceded by an increased interest in attending to speech and improved comprehension of language. Typical abnormalities include delayed and inappropriate echoing, reversal of pronouns, repetitiveness, literalness of meaning, idiosyncratic use of words and phrases, as well as intonation which is monotonous, mechanical, or singsong in quality (Baltaxe & Simmons, 1975). Although speech tends to be better developed in the passive and active-but-odd-groups during the preschool period, similar abnormalities are observed.

Most aloof children do not engage in symbolic play. Although they can manipulate objects, they do not seem to be able to pretend that toys represent real things. Passive children may participate in imaginative games that are organized by others, typically taking the role of "baby" in a game of house, whereas active-but-odd individuals may build and rebuild the same imaginary system of

roads or pretend to be an animal or superhero over and over again (Wing & Attwood, 1987). During the preschool period, repetitive stereotyped activities are prominent among all three social groups. Those whose IQs are low tend to engage in simple actions, such as finger flicking, arm flapping, or body rocking (Rutter & Lockyer, 1967), whereas the more able may display more complex repetitive behavior, such as organizing objects into lines or patterns, following a lengthy bedtime ritual, or insisting on taking exactly the same route each day (Kanner, 1943; Wing, 1980). Difficult, socially embarrassing behavior can occur in response to interference with repetitive routines and a lack of understanding of instructions and the rules of social behavior.

During the school years, many of the more disturbing behaviors lessen (Gillberg, 1984). The child learns to respond more or less appropriately to routine demands and expectations (Lotter, 1974), although ritualistic behaviors and peculiar circumscribed interests may persist into adulthood (Rutter, 1970). Some socially passive or aloof children whose IQs are in the normal to mildly impaired range can make academic progress in regular classroom settings, but most autistic children require placement in special classes.

Adolescence is relatively uneventful for many autistic people. Although menstruation and sexual drive are often tolerated quite calmly (Wing, 1980), exhibitionism or public masturbation may cause problems (Corbett, 1976). Some individuals not only show marked improvement during this period that is seemingly associated with growing self-awareness (Kanner, Rodriquez, & Ashenden, 1972) but also gains that continue into the adult years (Gillberg, 1984). However, somewhere between 10% and 35% undergo serious regression, with no obvious precipitating factor other than puberty itself (Gillberg & Schaumann, 1981; Rutter, 1970). If such deterioration does occur, it appears to level off and not to progress further during the adult years (Rutter, 1985). Approximately one fifth of autistic children develop seizures by age 18, with the peak onset being between 11 and 14 years (Deykin & MacMahon, 1979; Rutter, 1970). Seizures are more likely in autistic children with severe retardation than in those of normal nonverbal intelligence (Rutter, 1983).

Long-term follow-up studies of autistic children (DeMyer, Barton, DeMyer, Norton, Allen, & Steele, 1973; Eisenberg, 1956, 1957; Eisenberg & Kanner, 1956; Kanner *et al.*, 1972; Lotter, 1974; Rumsey, Rapoport, & Sceery, 1985; Rutter, 1970; Rutter & Lockyer, 1967) have tended to show that the natural course of the disorder is one of gradual symptomatic improvement with persistent social impairments. However, in adult life, some two thirds of autistic individuals continue to be completely unable to care for themselves, whereas only 5% to 17% develop sufficiently to be able to work independently and lead some kind of social life. IQ and language skills are the best predictors of adult functioning. If a child's nonverbal IQ is below 50 or 60, it is virtually certain that the individual will remain severely handicapped. If IQ is above that level but gross language impairments persist beyond 5 years of age, the child may make a fair social adjustment and be able to live and work in sheltered supervised settings. Children whose intelligence is in the normal range, and who have developed communicative language by 5 years of age, have a fifty-fifty chance of achieving a good social outcome in adulthood. Even those who have progressed well may lack social skills and an ability to empathize with others (Bemporad, 1979; Kanner 1971; Kanner *et al.*, 1972; Rumsey, Rapoport, & Sceery, 1985; Rutter, 1970). The language characteristics of high-functioning adults include evidence of concrete, perseverative, impoverished, circumstantial, and obsessional thinking (Rumsey *et al.*, 1985), delivered in a flat monotonic, mechanical style that can sometimes appear to be quite similar to the language characteristics of schizophrenics (Paul, 1987). Although DSM-III criteria clearly discriminate between autistic and schizophrenic children (Green, Campbell, Hardesty, Grega, Padron, Gayol, Shell, & Erlenmeyer-Kimling, 1984) DSM-III schizophrenia has been described, albeit rarely, in older individuals with histories of autism (Petty, Ornitz, Michelman, & Zimmerman, 1984).

Theories of Autism

Biological Studies

Kanner's (1943) original suggestion that autism is inborn stimulated investigation of biological correlates. The role of genetic factors has been examined by Folstein and Rutter (1977), who described a sample of 21 same-sexed twin pairs that included at least one autistic child. Ten of these pairs were monozygotic (MZ), and four of the MZ co-twins were diagnosed as having autism, whereas none of the dizygotic (DZ) co-twins met criteria. However, 9 of the 10 MZ twins were concordant for milder cognitive impairment and language delay,

suggesting that autism could be the severest form of a continuum of inherited language and cognitive problems. More recently, Ritvo and his colleagues (Ritvo, Freeman, Mason-Brothers, Mo, & Ritvo, 1985) collected 61 families with autistic twins who volunteered for study. Forty-seven families provided sufficient records to make a diagnosis. Forty met research diagnostic criteria for autism with 95.7% concordance in the MZ twins (22 of 23) and 23.5% of the DZ group (4 of 17). Sample selection may have contributed to the very high MZ concordance in this study. However, cognitive-processing difficulties were found in 10.3% of siblings, a finding consistent with the view that there may be a subgroup of autistic children with a genetic etiology and variable penetrance.

Autism has been described in association with a variety of chromosomal abnormalities, including Down's syndrome (Knobloch & Pasamanick, 1975; Wakabayashi, 1979) and Klinefelter's syndrome, (Nielsen, Christensen, Friedrich, Zeuthen, & Stergaard, 1973). More recently, Brown (Brown, Jenkins, Friedman, Brooks, Wisniewski, Raguthu, & French, 1982) reported on 6 autistic children with fragile-X syndrome. This syndrome is said to be second only to Down's syndrome in the etiology of retardation. Cells harvested from a thymidine-deficient culture medium show a fragile long arm of the X chromosome. Affected males show retardation, macro-orchidism, square prognathous jaw, high arched narrow palate, and large ears. Composite estimates suggest that approximately 12% of autistic persons will have associated fragile-X syndrome (Fisch, Cohen, Wolf, Brown, Jenkins, & Gross, 1986).

Autism has also been reported to occur in association with a wide array of other known disorders of the central nervous system, including congenital rubella (Chess, 1971), phenylketonuria (Knobloch & Pasamanick, 1975), infantile spasms (Taft & Cohen, 1971), Rett's syndrome (Hegberg, Aicurdi, Dias & Ramos, 1983), and acute encephalopathic illness (DeLong, Bean, & Brown, 1981).

Neuroradiologic techniques have been used in an effort to define structural abnormalities in the brains of autistic children. Although CT scans have revealed ventricular enlargement in some autistic children (Caparulo, Cohen, Young, Roth, Shaywitz, Shaywitz, & Rothman, 1981; Campbell, Rosenbloom, Perry, George, Dricheff, Anderson, Small, & Jennings, 1982; Gillberg & Svendsen, 1983), the scans of those who are higher functioning and without concomitant central nervous system disease appear normal (Prior *et al.*, 1984). Moreover, brain measurements vary widely within the autistic population (Rosenbloom, Campbell, George, Kricheff, Taeporos, Anderson, Reuben, & Korein, 1984). Recent magnetic resonance imaging (MRI) studies (Courchesne, Yeung-Courchesne, Press, 1988) indicate some hypoplasia of the vermis of the cerebellum. The Purkinje layer of the cerebellar hemisphere and vermis showed significantly lower counts in four autistic brains at autopsy (Ritvo, Freeman, Scheibel, Duong, Robinson, Guthrie, & Ritvo, 1986).

Studies directed toward the exploration of aspects of brain metabolism have also led to inconsistent findings. It appears as though whatever parameter is studied, there is a higher degree of variability and abnormality among autistic subjects as compared with controls, but the abnormalities are rarely consistent. PET scan studies, using radioactive labeled glucose (Rumsey, Duara, Grady, Rapaport, Margolin, Rapoport, & Cutler, 1985), showed elevated glucose utilization in autism, but no distinctive differences from controls. Similarly, radioactive xenon inhalation studies reveal diminished bilateral cerebral blood flow and an abnormal resting landscape, as well as the standard decrement in bihemispheric blood flow from one resting study to another (Sherman, Nass, & Shapiro, 1984). There is some agreement between these results and Damasio and Maurer's (1978) suggestion, elaborated in the course of studies of the gait and immobile faces of autistic persons (Damasio & Maurer, 1978; Maurer & Damasio, 1982), that the mesolimbic dopaminergic system may not be as active in autism as in controls. However, no consistent defect in dopamine metabolism has as yet been found.

Although studies of other neurotransmitters, including norepinephrine and acetylcholine, have also been inconclusive, hyperserotonemia has been a consistent finding in about one third of autistic children (Hanley, Stahl, & Freedman, 1977; Ritvo *et al.*, 1970; Schain & Freedman, 1961; Takahashi, Kanai, & Miyamoto, 1976). More detailed studies of serotonin receptors and serotonin levels show persistent diminution of receptor sites (Anderson, Freedman, Cohen, Volkman, Hoder, McPhedran, Minderaa, Hansen, & Young, 1987). Recently, a challenge test based upon the fenfluramine–serotonin relationship indicates that central serotonergic responsivity is reduced in autistic adults (McBride, Anderson, Hertzig, Sweeney, Kream, Cohen, & Mann, 1988).

Although there is little doubt that autism is associated with abnormal function—and perhaps structure—of the central nervous system, the multiplicity of possible etiological factors makes the search for relationships between identified abnor-

malities and the linguistic, cognitive, and interpersonal disabilities of autism difficult indeed. On the level of the organization of the central nervous system, several model systems, including dysfunction of central vestibular connections (Ornitz, 1983, 1985), temporal lobe dysfunction (DeLong, 1978; Hauser, DeLong, & Rosman, 1975), and left hemispheric dysfunction (Prior, 1979), have been advanced as possible mechanisms underlying the brain–behavior relationships in autism. Although clearly preliminary, these proposals do provide directions for further hypothesis-bound research (Golden, 1987), in the course of which biologic investigators may be better able to link the unique behavioral features of autism to the findings generated by the application of new techniques for defining the architecture and function of the central nervous system.

Psychological Studies

The detailed examination of the organization of the psychological functions in autism has also been dominated by the search for a single defining mechanism. The cognitive and linguistic attributes of autistic children have been the most extensively investigated, perhaps because of the early recognition of their importance as predictors of later functional level (Eisenberg & Kanner, 1956; Lotter, 1974). For the past two decades, autism has been viewed primarily as a cognitively based disorder in which socially deviant behavior emerges secondarily (Hermelin & O'Conner, 1970; Reichler & Schopler, 1971; Rutter, 1978, 1983). Autistic children vary greatly in their intellectual performance. As they do in normals, their scores predict later scholastic achievement and occupational and social status. They are moderately consistent over time (Snow, Hertzig, & Shapiro, 1987) and do not fluctuate markedly according to changes in psychiatric state. Cognitive performance is not fully explainable in terms of motivational factor, but rather varies systematically in accordance with task difficulty (Clark & Rutter, 1979) and the nature of task demands (Volkmar & Cohen, 1982). The IQ scores of autistic children are found to differ from mental age matched controls in showing a distinctive pattern of deficits in verbal sequencing and abstraction skills as opposed to rather good performance in visuospatial or rote-memory skills, a pattern suggestive of the presence of specific deficits in language and language-related functions (DeMyer, 1975; DeMyer, Barton, & Norton, 1972; Hermelin & O'Connor, 1970; Lockyer & Rutter, 1970; Rutter, 1983; Tymchuk, Simmons, & Neafsey, 1977). Curiously, their rote memory is excellent, but they do not take advantage of the usual structuring clues that normals use, remembering disconnected loose as well as asyntactically organized sentences.

Studies of language organization in speaking autistic children has revealed that they do not function as simply arrested children, but that their language capacity shows a pattern disturbance with special difficulties in the communicative sector. Autistic children echo far more than did normally developing 2- and 3-year-olds, but their echoing is not productive or generative as in normal language acquisition. Autistic children are adequate designators, but the development of grammatical complexity and flexibility is both delayed and deviant. Echoing sometimes gives way to poorly contextualized remarks, irrelevancies, and pat repetition of phrases that are called *delayed echolalia* by Kanner (1943), accounting for their being taken as odd. Moreover, reaction time for echoes is significantly less than for other responses, suggesting a use of the echo as a social closure device and a pragmatic technique deriving from a perceived need to take turns in communicating (Shapiro, 1977; Shapiro, Chiarandini, & Fish, 1974; Shapiro, Roberts, & Fish, 1970). More recent studies that take off from these earlier findings show that autistic children's language problems do not stem primarily from a syntactic deficit (Howlin, 1984; Tager-Flusberg, 1981) but involve the use of language in dialogues (Cromer, 1985; Prizant, 1982).

More recently, the affective and social components of autistic behavior, which were long neglected, have once again become a focus of investigation (Fein, Pennington, Markowitz, Braverman, & Waterhouse, 1986). The natural spontaneous expression of positive affect is diminished in autistic children (Snow *et al.*, 1987) as is the frequency with which they use emotionally expressive gestures (Hermelin, 1982). Moreover, their ability to imitate and analyze affective cues in controlled task situations is also deficient (Hertzig & Sherman, 1989; Hobson, 1986; Jennings, 1973; Langdell, 1978). It is becoming increasingly clear that "the stimuli that pose difficulties for autistic children are those that carry emotional or social meaning" (Rutter, 1983, p. 528).

Treatment and Education

There is hardly a treatment approach that has not been offered to autistic children. Psychoanalysis, behavior modification, therapeutic nursery school, dyadic and triadic treatments, special

education, speech and language therapy, holding therapy, sign language, language-based approaches to learning, neurodevelopmentally directed therapies, including perceptual retraining as well as pharmacotherapies, have all been applied to autistic patients (Shapiro, 1978). The overwhelming finding has been that humane management leads to better outcome than when the child is isolated and permitted to regress. Nevertheless, systematic comparison of the progress made by autistic children in three different educational settings found that the best results were obtained by the unit that used extensive, specific teaching in a well-controlled classroom that provided an organized and structured program and emphasized the use of techniques appropriate to the level and pattern of handicap. Least progress was made in a permissive environment in which regressive techniques dominated (Bartak & Rutter, 1973; Rutter & Bartak, 1973).

The goals of treatment for autistic children include (1) the fostering of normal development, (2) the promotion of learning, (3) the reduction of rigidities and stereotypies and other maladaptive behaviors, and (4) the reduction of family distress (Rutter, 1985). Autistic children require a multimodal approach that includes a focus on the development of socialization and communication skills. Improvement is consistently related to IQ, with most impaired children making the least progress. Stereotypies tend to increase in unstimulating environments (Baumeister, 1978), so that it is desirable that autistic persons be kept actively engaged in play or work with structured opportunities for personal interactions (Clark & Rutter, 1981). A system of graded change (Marchant, Howlin, Yule, & Rutter, 1974), that is, the introduction of a series of very small steps, each of which is not noticed or reacted to by the child, to lead to a major change, may be helpful in the reduction of rigidity and stereotypy. Behavioral approaches are often useful in the reduction or elimination of nonspecific maladaptive behaviors, such as tantrums, aggression, and bedwetting (Hemsley, Howlin, Berger, Herzov, Holbrook, Rutter, & Yule, 1978; Howlin, Marchant, Rutter, Berger, Herzov, & Yule, 1973), as well as in the facilitation of communicative skills (Lovass, 1987).

There are no drugs that specifically affect autism. Early reports of the efficacy of fenfluramine (Ritvo *et al.*, 1983) have not been sustained (Campbell, Adams, Small, Curren, Overall, Anderson, Lynch, & Perry, 1988; Leventhal, 1985). Nevertheless, drugs may be useful for the control of specific behaviors. Major tranquilizers have been used successfully in the reduction of agitation, tension, and overactivity (Campbell, 1978; Corbett 1976), although need must be balanced against the possibility of adverse effects on learning as well as the long-term risks of tardive dyskinesia.

Education and treatment of the autistic child cannot occur in a vacuum. Attention to the changing needs of parents is a critical part of any intervention program. Autism imposes a life-long handicap, and the families of affected individuals require a long series of guiding experiences as they cope with the ongoing stresses of caring for an impaired child, adolescent, and adult.

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CHAPTER 30

The Nature and Types of Mental Illness in the Mentally Retarded

Frank J. Menolascino

Introduction

The various degrees of mental retardation bring unique challenges relative to the risk of development of clusters of maladaptive behaviors. In general, persons with mental retardation are nearly twice as likely to have severe behavioral problems or mental illness as are the nonmentally retarded (Balthazar & Stevens, 1975; Beier, 1964; Eaton & Menolascino, 1982; May & May, 1979; Menolascino, 1975; Penrose, 1966; Pollock, 1958). This risk may result in part from the person's difficulty in processing information, allied medical, physical, or sensorial difficulties; actual organic problems; cultural-familial factors; and society's nonacceptance of retarded persons and elements of raw prejudice toward them. Other factors involve the inability of some retarded persons to communicate their feelings; this impairment is particularly important in the clinical setting wherein mentally retarded-mentally ill citizens have great difficulty in realizing the subjective and historical perspective of their behavioral status. Even those who appear to have superficially adequate language have extreme difficulty in processing abstract feelings and emotions. Many severe behavioral problems are caused by the way we teach or do not teach mentally retarded persons to become integrated into society. Globally, all of these problems are affected by society's role in the acceptance and support of people with special needs.

Undeniably, maladaptive behaviors are frequently reinforced and increase unnecessarily because of such reinforcement. For example, Johnny might throw objects on the floor in the classroom because he is frustrated. His frustration may be a condition of his inability to express himself. But if the teacher demands retribution, behavior could likely increase because he is getting the attention he might not otherwise receive. Paradoxically, the teacher's punishment becomes the child's reward; his maladaptive behavior is a means of avoiding the particular task, and the punishment allows him to avoid it. Over time this child can develop a cluster of "primitive" behaviors that enable him to gain control over an unresponsive world. If constantly punished, he would likely submit to his teacher, but he would not bond with the teacher. He would not learn the value inherent in human presence and participation, in equitable interpersonal transactions.

Examining the various levels of mental retardation shows its behavioral at-risk nature. Persons with *severe* and *profound* mental retardation often have major central nervous system disorders, sensory handicaps, such as deafness and blindness, and

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severe motor and physical disabilities, such as cerebral palsy and allied seizure disorders. These conditions all make the person more likely to have difficulty with interpersonal transactions, dealing with stress, and even with what we might regard as minor changes in daily routine (Chess, Korn, & Fernandez, 1971). Many persons with severe-profound mental retardation cannot express themselves without intensive teaching and ongoing support. They can easily learn to withdraw from the world through autistic-like behaviors (e.g., rocking, finger flicking, arm waving, and other persevered behaviors) as an alternative to seeking others out and living independently. The lack of expressive language in many can easily lead to "primitive" communication in the form of aggressive, self-injurious, or self-stimulatory behaviors. These are often attempts to interact with, respond to, or master an otherwise overly complex or unresponsive world. *Moderately* retarded persons, besides their obvious developmental delays, have difficulty primarily with processing abstract information (Webster, 1970). Most such persons possess some degree of receptive and expressive language and are able to adapt well and functionally in the world. In general, they can easily be taught the basic parameters of language, a wide repertoire of self-care and daily living skills, and vocational skills. Yet they are also highly at-risk to develop behavioral problems. Often their functionally adaptive and communicative abilities are a facade. The caregiver, too, often assumes that a moderately retarded person is adequately processing and dealing with complex situations, such as the death of a loved one, the loss of a job, or loneliness, when actually such events can be overwhelming—in terms of both the person's comprehension and expression. Such complex and abstract human events may lead to severe "behavioral" problems. If the in-crises retarded persons are not fully understood, are not enabled to express themselves, or are not actively supported in times of stress, they may display their emotions through what we would term *maladaptive* behaviors, such as excessive sadness, withdrawal, or avoidance, beyond what is considered normal. In reality, these may be "normal" responses, given their ability to understand, communicate, and emotionally express themselves. Without adequate and sensitive support, a person with moderate mental retardation might rather quickly succumb to structured mental illness, such as an adjustment disorder, as a stepping stone to more serious illness (e.g., depression).

Diagnostic and Treatment Challenges

The diagnostic and treatment challenges of providing services for mentally retarded citizens who have allied emotional or behavioral needs are topics that have recently taken on increased importance in view of significant changes in society's care and treatment of its retarded citizens. Deinstitutionalization has not only changed the physical site of service delivery, bringing persons with mental retardation into the mainstream of community life, but has also dramatically highlighted the need for teaching and treatment strategies for persons with mental retardation and allied behavioral challenges. No longer are these persons automatically hidden away in state institutions, and no longer are punishment and restraint practices endured away from public scrutiny. Increasingly, many persons with both mental retardation and mental illness now live in their respective communities in a societal climate that critically questions the demeaning and "put-them-away" practices of the past and present (United States Senate, 1984).

The recent movement of retarded persons from institutional back wards into the community has literally changed the definitions of normal and abnormal behavior. Behaviors that were traditionally "expected" or "tolerated" in institutionalized retarded citizens are viewed as abnormal within the community mainstreams of our society. For example, the clinical phenomena of rocking, rumination, and headbanging are frequent behaviors in institutionalized persons; within the institution they are expected behaviors and their abnormality is tolerated at best and severely restrained at worst. It should be pause for reflection that such behaviors are rarely seen in retarded citizens who have been raised at home and who have matriculated through their home community system of educational-vocational-residential services.

As mentally retarded-mentally ill persons move from institutions into the mainstream of community life, they present caregivers with new diagnostic and treatment challenges. Diagnosis becomes important so that caregivers can gain therapeutically helpful insights into the underlying causes of "behavioral problems" and "noncompliant behaviors" and thereby apply similar treatment approaches to persons with similar behavioral needs.

A major issue in the care of retarded persons with severe behavioral problems is the widespread use of high levels and chronic usage of psychoac-

tive medications for chemical restraint (Donaldson, 1984; Lipman, 1970; Lipman, DiMascio, Reating, & Kirkson, 1978; Wilson, 1983) rather than as entrées to learning. Another major issue is the relative lack of a sufficient number of community-based support systems for persons with mental retardation and allied mental illness (Menolascino & McCann, 1983).

A number of major concepts, issues, and trends are relevant to the relationships between mental illness and mental retardation. The definition of mental retardation as found in the diagnostic system of the American Association on Mental Deficiency (Grossman, 1983) posits the presence of subaverage intellectual functioning and associated deficits in social-adaptive behavior. The majority of the disorders listed as capable of producing the symptom of mental retardation, however, are more descriptive of syndromes than of specifically understood diagnostic entities (especially in AAMD categories VII and VIII; see Table 1).

Table 1. Classification of the Causes of Mental Retardation[a]

O.	Infections and intoxications (e.g., prenatal and postnatal infections)
I.	Trauma or physical agent (e.g., inborn errors of metabolism)
II.	Metabolism or nutrition (e.g., inborn errors of metabolism)
III.	Gross brain disease (postnatal) (e.g., neurocutaneous dysplasia)
IV.	Unknown prenatal influence (e.g., malformations of the brain)
V.	Chromosomal anomalies (e.g., Down's syndrome)
VI.	Gestational disorders (e.g., prematurity)
VII.	Following psychiatric disorder (e.g., childhood schizophrenia)
VIII.	Environmental influences (e.g., psychosocial disadvantages)
IX.	Other conditions (e.g., defects of special senses)

[a]Adapted from *Manual of Terminology and Classification in Mental Retardation* by H. Grossman, 1983, Washington, DC: American Association on Mental Deficiency. Copyright 1983 by the American Asociation on Mental Deficiency. Reprinted by permission.

It should be noted that defining or describing the known/unknown causes of the symptom of mental retardation may say much (i.e., psychosis as part of the PKU syndrome and likewise for Lesch-Nyhan; tuberous sclerosis, and the like) or very little about expected behaviors beyond rough guidelines for social-adaptive accomplishments at different levels of retardation. Similar perplexity may exist in the delineation of the causes and manifestations of mental illness in a given individual. We define mental illness as occurring at that time when a cluster of behavioral signs and symptoms come together and become overly disruptive of the person's ability to function smoothly within the mainstreams of his or her family and community settings (Menolascino & Stark, 1984).

Mentally retarded persons have been noted to display the vast majority of the major psychiatric disorders, that is, classically described instances of mental illness coexist with mental retardation. The one does not exclude the other, even in severe-profound mental retardation. Some mental illnesses can cause pervasive developmental delays that may be separated only with great difficulty from strikingly similar developmental manifestations of mental retardation (e.g., infantile autism). When the uncertainty of primary causes of mental retardation are combined with the problems of clinical description in mental illness, an area of possible clinical confusion arises: symptomatic behaviors (although produced by different causes) can have the *same* final symptom(s) pathway: abnormal behavior. For example, the origins of an eight-year-old retarded child's hyperactivity may range from motor expression of anxiety to manifestations of cerebral dysfunction, or it may arise from both. Similarly, a shortened attention span in a mentally retarded four-year-old child may be the end product of factors ranging from inadequate parenting relationships in infancy (suggesting that the parents were unable to operate as a selective stimulation barrier for the child) to actually impaired midbrain neurologic screening of incoming stimuli. The adult with moderate mental retardation can surely have a seizure disorder and also display schizophrenia along with his or her "old" developmental manifestations of mental retardation. Combined diagnoses are clearly indicated in these frequent instances of mixed disorders. Failure to describe and delineate multiple disorders in the same individual sharply limits both professional understanding and effective treatment.

The symptomatic nature of mental retardation,

however, coupled with multiple causes of an associated behavioral disorder, not only increases the possible number of causes but also presents challenging opportunities for alert professionals to initiate the necessarily wide array of specific treatment interventions that are so often needed. The *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) (American Psychiatric Association, 1980) and its 1987 revised edition (DSM-III-R) embrace most of the symptomatic and developmental parameters that are so important here. These considerations suggest that a description of mental illness in the mentally retarded should include behavioral patterns that produce serious conflicts within retarded individuals, their families, and the greater circle of their community transactions. A constellation of persistent personality traits, coupled with pervasive anxiety at a time of personal-social crisis, will significantly reduce the effectiveness of the mentally retarded in their intellectual and social-adaptive spheres.

Nature of Mental Illness in the Retarded

One reason frequently given for institutionalizing persons with mental retardation has been concern about their unmanageable behavior as they approach mid-adolescence or adulthood. Although such persons show a higher incidence of epilepsy and motor, special sensory, and allied handicapping conditions (Menolascino & Egger, 1978), the tremendous improvements in medical treatment of these problems during the last 20 years has made possible revolutionary changes in our approach. Similarly, improved mental health treatment approaches have permitted a dramatic decrease in overly restrictive procedures and practices: isolation rooms, restraints, locked units, and excessive pharmacologic regimes have become associated with the professional practices of the past and, where still present, they are increasingly questioned by professionals and parents alike.

Improvements in the treatment of mental illness in the mentally retarded have flowed from an increased understanding of the nature (i.e., especially the etiological and developmental factors) and manifestations of mental illness in persons who are mentally retarded. Further exploration has made it abundantly clear that any condition that renders one less capable of handling reality-based demands makes one more susceptible to mental illness. Table 2 enumerates some of the factors that make persons with mental retardation vulnerable to mental illness.

Influences of the Level of Retardation

Severe Mental Retardation

The factors noted in Table 2 are further affected by the level of mental retardation present. For example, in the severely retarded, the presence of major central nervous system impairments tends to impair greatly their ability to access or participate in social transactions. Without gentle and stable interpersonal support systems, the severely retarded tend to respond adversely to instances of gross interpersonal stress with stereotypical and out-of-contact behaviors that mimic autism. Similarly, their severe language delay (which is usually present) seriously restricts the development of complex personality development, thus enhancing vulnerability to emotional under- or overreactivity. A study by Chess *et al.* (1971) clearly noted that if severely retarded infants (in this study they were also blind and deaf secondary to rubella) were provided with early and ongoing interpersonal and developmental stimuli, they tended primarily to show delayed (i.e., primitive) behaviors that were consistent with their level of retardation. They also noted, however, that in those instances in which these early sets of interventive experiences were *not* provided, the infants consistently displayed "blindisms," prolonged rocking, marked irritability, persistent obstinancy, and "organic autism." Such learned responses often led to gross forms of harsh parental punishment and restraint. Thus, the lack of early preventive intervention (including parental supports) so often, in reality, creates the very "problems" it later seeks to eradicate. If the handicaps were energetically treated, the impact on the person would be developmental delay, but behavioral intactness; without these intervening developmental-treatment experiences, a wide array of very disturbed and out-of-contact behaviors would result.

Moderate Mental Retardation

In the moderately retarded, a set of personality characteristics is noted that can either lead to maximal developmental growth or the focal point for mental illness. Specifically, during an extensive study of young moderately retarded children, Webster (1970) noted a consistent set of personality

Table 2. At-Risk Factors for Mental Illness in the Retarded

1. Relative inability to understand the demands of culture secondary to the presence of intellectual and social-adaptive limitations.

2. Major handicaps secondary to central nervous system impairments. These often lead to secondary behavior manifestations (e.g., self-stimulation as an external replacement for impaired inner resources).

3. Constricted emotional and personality growth secondary to delayed self-concept (i.e., differentiating the self from the nonself). Allied components are sensory and motor handicaps, and delayed integrative functions. These include poor reality perceptions and retained primitive thinking (e.g., magical thinking, confusion of reality/fantasy; problems in handling aggression; difficulty in pinpointing sources of frustration).

4. Delayed language development, with particular reference to delayed ability to express appropriate needs and decreased exploration of the world. Lack of ability to understand the interpersonal world around them leads to a very concrete cause-and-effect understanding of that world.

5. Impaired memory and transfer of learning. This leads to lowered inner control and decreased ability to delay responses and plan alternative methods of action. Thinking tends to be concrete and rigid, leading to poor responses to external and internal stress.

6. Low self-esteem fostered by early attendance at special schools. This leads to a sense of "differentness" and, eventually, a self-image of deviancy. The client perceives he or she failed the parents, leading to an increased need for praise and approval. Repeated nonapproval from peers leads to an increasing sense of incompetence and ineffectiveness. Peer rejection and lowered family and group expectations are reflected in lowered individual motivation.

7. Atypical personality defenses include increased obsessional behavior. This helps to control new situations and control and/or cope with the environment. Fears, however, are retained longer and defenses are more fixed, leading to rigidity of lifestyle and increased utilization of nonadaptive defenses (e.g., denial, compulsive rigidity, withdrawal, regression, projection, and the like).

8. Vulnerability in adulthood. Continuation of the previously noted factors in childhood and adolescence directly leads to longer dependence, social and vocational limitations, and interference with heterosexual experiences. Failure to master key adaptational tasks at developmentally appropriate milestones in childhood and adolescence, when complied with the lowered global intelligence, culminates in poor judgment.

characteristics that he termed the primary psychopathology of moderately retarded youngsters: (1) benign autism, (2) repetitiousness, (3) relative inflexibility (rapid external changes → heightened propensity for personality disorganization), (4) passivity (a protective posture secondary to perceived failure), (5) simplicity of emotional life (secondary to delayed personality development and allied symptoms, such as motor and special sensory handicaps), and (6) marked language delay.

The benign autism, nonobsessive repetitiousness, relative inflexibility, passivity, and simplicity of emotional life are all fertile ground for both the initiation of behavioral disorders (if highly supportive family or necessary program services are not provided) and misdiagnosis. Regarding the latter, one must be careful not to view the personality characteristics of persons with moderate mental retardation as signs or symptoms of mental illness. The few available autobiographies of moderately retarded individuals are helpful (e.g., Hunt, 1967), because they illustrate the portrayal of the basic personality features without symptoms or signs of allied mental illness.

Mild Mental Retardation

Mildly retarded persons have a unique set of stresses. Often, their nearly normal appearance tends to preclude their identification by others as disabled. This state of affairs can lead them and their loved ones to unrealistic expectations and major interpersonal failures. At the same time, these individuals are capable of developing some insight into their limitations.

Mental illness in the mildly retarded often is the result of the individual being labeled as deviant, while being concomitantly enmeshed in the dynam-

ic interplay of disturbed family transactions. The frequent delay in establishing that these youngsters have a distinct disability (usually not confirmed until 6 to 8 years of age) is a common source of anxiety. Low self-esteem is very frequent in persons with mild degrees of mental retardation; the stigma of attending segregated classes and adverse encounters with the social-interpersonal environment tend to make them feel even more ineffective and different. This life status is compounded by their inability to integrate the normal developmental sequences at the appropriate times. For example, during the late childhood period of personality integration, mildly retarded persons have considerable difficulty understanding the symbolic abstractions of schoolwork and the ongoing complexities of social-adaptive expectations from both family and peer group. At this stage, they often gain some understanding of their limitations. Unfortunately, by early adolescence, they have all too often established an identity that incorporates both mental retardation and behavioral deviancy. So often, they are not buffered (or redirected) by loved ones into new interpersonal coping styles that could help them to correct earlier misconceptions about their self-identity. Without a source of ongoing family or community support and direction, they are at high risk for developing patterns of severe behavioral problems or marginal identities. In our experience with persons with mild mental retardation, it is not uncommon to deal with severe psychosexual disorders, suicide attempts, and transgressions with the legal system. These personal-social developmental problems are due primarily to the lack of ongoing family and community support systems.

Persons with *mild* mental retardation primarily have difficulty with the societal acceptance of their "differentness" (Menolascino & McCann, 1983). Society's posture of devaluation of retarded persons often leads to segregation and a superimposed subculture of deviancy. Raw prejudice can lead to more deviancy, which, in turn, can lead to more socially unacceptable behaviors. Most often, the mild mentally retarded are all too cognizant of their cognitive limitations and understand the consequences of their actions. Yet, through naivete, misdirection, exploitation, or the pressures of social conforming, their behavior can develop outside their direct control and lead to severe behavioral problems or mental illness more easily than in the population at large.

Lastly, a major complicating factor in the diagnosis and treatment of mental illness in the mentally retarded is the possible presence of any of a number of allied medical or developmental disabilities. The most common ones are cerebral palsy, the various types of epilepsy, auditory disorders, visual disorders, diabetes mellitus, and hypothyroidism (Ruedrich & Menolascino, 1984).

Frequency and Types of Mental Illness in the Retarded

A few introductory comments on clinical diagnostic assessment are in order before the frequencies and types of mental illness in the mentally retarded are discussed.

The 1970s brought mental health and mental retardation professionals to a realization that mental illness can frequently hinder the intellectual and social adaptive growth of mentally retarded individuals. Nevertheless, there has been only small change in the management of the mentally ill-mentally retarded child or adult; his or her clinical diagnostic assessment is too often accomplished only with much difficulty and too little understanding of the manifestations of mental illness in persons with mental retardation.

It should be stressed at the outset that the mental status examination of a mentally retarded individual—whether to confirm the primary diagnosis of mental retardation, to assess the possibility of "pseudoretardation," or to confirm the coexistence of mental illness with mental retardation—cannot be accomplished in isolation from general diagnostic procedures. In short, a psychiatrist ideally should carry out his or her own physical and neurological examinations and should be able to interpret pediatric, internal medicine, and neurologic consultations, as well as such studies as psychological evaluations and electroencephalograms. Direct caregivers play a key role in providing concrete objective data in these consultations. With these types of information, the mental health professional is in a much better position to see the whole person and to piece together the diagnostic puzzle that mental illness can present in the mentally retarded person. To cast the mental health professional in the role of a "complete professional" may appear to be asking too much, but failure to assess clearly all parameters of behavior can result in many of the major diagnostic errors made with persons with mental retardation. It seems that exclusive focus on the mental status examination has perpetuated the classic split between the *psyche* and the *soma* in evaluating and treating persons with these special needs.

Studies concerning the frequency and types of mental illness in the mentally retarded have some major methodological problems. Before 1960, studies were carried out primarily in institutions. Frequency rates reported range from 16% (Penrose, 1966) to 28% (Beier, 1964) and as high as 40% (Pollock, 1958). These studies, as well as more recent investigations of institutionalized persons (Balthazar & Stevens, 1975; May & May, 1979; Menolascino, 1975) are questionable guides to the true frequency of mental illness in the mentally retarded because institutionalization traditionally has been used as society's mechanism for "managing" persons with social-behavioral difficulties. The more recent studies may also reflect the increasing referral of "community rejects" to institutions (Menolascino, 1970). In other words, are these individuals disturbed because of personality deficits secondary to their underlying mental retardation, or are they disturbed because of the way they are treated by society?

Many reports on mentally retarded individuals living with their natural families or in their primary communities have appeared (Berman, 1977; Chess, 1962, 1970; Dewan, 1948; Eaton & Menolascino, 1966, 1967, 1982; Menolascino, 1967, 1969; Webster, 1970). These studies report that persons with mental retardation fall prey to the same types of mental illness that befall people with normal intellectual abilities. Recent professional literature repeats the theme that persons with mental retardation have the same range of psychoses, neuroses, personality disorders, behavior disorders, and adjustment reactions as the general population. Other investigators report a higher incidence and a different spectrum of mental illness among retarded persons than in the general population (Beier, 1964; Garfield, 1963; Phillips & Williams, 1966). Some suggest that there are qualitative differences as well, so that some psychiatric conditions seen in retarded persons may represent unique syndromes (Bender, 1970; Menolascino & Egger, 1978; Webster, 1970). Yet there have been few studies that have looked specifically at the frequency and types of mental illness in this population.

The data in Table 3 summarize the types of mental illness found in a review of 543 admissions of mentally retarded persons with mental illness to an acute psychiatric care setting over a 5-year span of time. (This review was conducted by the authors at the Nebraska Psychiatric Institute, Omaha, Nebraska.)

Table 3. Mental Illness in the Mentally Retarded[a]

	Ages								
Psychiatric diagnosis	1–5	6–10	11–15	16–20	21–25	26–30	31–35	36+	Totals
Schizophrenic disorders									
Catatonic type			1	4	2	1		2	10
Paranoid type				8	5	3	4	10	30
Undifferentiated type				15	8	10	5	15	53
Residual type					4	13	13	15	45
									138
Organic brain disorders									
Behavioral reaction		4	11	7	13	6	8	6	55
Psychotic reaction	1	1	2	2	10	14	8	5	43
Presentile dementia								7	7
									105
Adjustment disorders									
Childhood and adolescent	5	4	10	25	15				44
Disturbance of conduct		3	5	10					18
Adulthood						12	9	5	41
									103

(*continued*)

Table 3. Mental Illness in the Mentally Retarded[a] (*Continued*)

Psychiatric diagnosis	Ages 1–5	6–10	11–15	16–20	21–25	26–30	31–35	36+	Totals
Personality disorders									
Schizoid				2	4	2	1		6
Paranoid						2	4		3
Narcissistic						1	1	1	7
Avoidant				1	2	2	5	2	7
Passive Aggressive				5	4	7	2	6	22
Antisocial				1	8			5	23
									68
Affective disorders									
Unipolar manic disorder				4	2	1			7
Bipolar affective					2			3	5
Major depression, recurrent			5	4	4	9	5	5	32
Cyclothymic disorder					1				1
									45
Psychosexual disorders									
Fetishism				1					1
Transvestism				2	1			1	4
Zoophilia			1						1
Pedophilia			1	1	4	2	1	3	12
Exhibitionism			1	1	2	1		1	6
Ego-dystonic homosexuality			1	1					2
Disorder not elsewhere classified				3	2	1	1	1	8
									34
Anxiety disorders									
Generalized anxiety disorder				2	1	2	2	2	9
Posttraumatic stress disorder				2	2	1	2	3	10
Obsessive compulsive disorder								1	1
									20
Other mental disorders									
Pervasive development disorder	2	5	7	2					16
Anorexia nervosa			3	2	1				6
Oppositional disorder			2						2
Substance use disorder					3		1	2	6
									30
Total									543

[a]From a review of mentally retarded-mentally ill patients at the Nebraska Psychiatric Institute, Omaha, Nebraska (unduplicated count) July 1, 1979 through June 30, 1985.

A similar spectrum of types of mental illness was noted in a study of 115 mentally retarded citizens, all of whom lived in their primary or a community-based program (Table 4). They were seen for diagnosis and treatment in an outpatient setting.

Review of Tables 3 and 4 shows that the mentally retarded can, and do, fall prey to many of the major psychiatric syndromes.

Even when persons with mental retardation are emotionally well adjusted, they experience some difficulty functioning independently or semi-independently in their community. If their lives are complicated by mental illness, their adjustment difficulties are obviously compounded. Recent studies have reported rather consistently a 25% to 30% frequency rate of emotional disturbances and sug-

Table 4. Psychiatric Disorders in a Community-Based Sample of 115 Retarded Persons, by Level of Retardation

Diagnosis	Mild (N = 50)	Moderate (N = 41)	Severe (N = 24)
Schizophrenia	13	8	4
Personality disorder	22	7	2
Anxiety disorders	1	0	0
Adjustment disorders	9	14	1
Organic brain syndrome			
With transient behavioral reaction	2	8	11
With transient psychotic reaction	3	4	6

gested strongly that clinical focus must be placed on the diagnosis and subsequent treatment of the combined findings of mental illness and mental retardation.

Childhood Schizophrenia and Mental Retardation

Psychotic reactions of childhood have presented a major challenge to caregivers because the psychotic child frequently functions at a mentally retarded level. Early observers believed that all psychotic children "deteriorated." In 1943, "early infantile autism" was described (Kanner, 1943); yet labeling a child "autistic" presents some formidable diagnostic and treatment problems. A number of follow-up studies (Bender, 1959, 1970; Menolascino, 1970; Rimland, 1964; Rutter & Schopler, 1978; Schain & Yannet, 1960), along with the literal rediscovery of the wide variety of primitive behavioral repertoires in retarded persons, have tended to mute the earlier clinical enthusiasm concerning the interrelationship between functional psychoses and early infantile autism. It is striking to observe bizarre behavior, persistent withdrawal, echolalic speech, and the affective unavailability of some persons in early adolescence who had clearly regressed from an earlier higher level of functioning.

Because treatment–prognosis guidelines may differ for youngsters with autistic reactions and the combined mental retardation–childhood schizophrenia syndrome noted above, the differential diagnosis is therapeutically significant beyond mere academic interest. For example, the vast majority of autistic youngsters seem to profit greatly from mental retardation services and secondary mental health consultations (Rutter & Schopler, 1978). Conversely, the retarded youngster who develops schizophrenia has a primary need for mental health services, with mental retardation services filling a secondary role until the functional psychosis subsides.

Adult Schizophrenia and Mental Retardation

Instances of paranoid schizophrenia are noted in both verbal and nonverbal persons. Examples in our recent clinical work include three nonverbal adults who drew on paper their attackers, complete with nonverbal gestures. One young man labeled his separate fingers as the "source" of his common delusions, which he portrayed symbolically in crude drawings. In the entire group of people with combined diagnoses of mental retardation and schizophrenia, the schizophrenia was clearly marked by altered affective responses, bizarre rituals, and interpersonal distancing.

Personality Disorders

Personality disorders are characterized by chronically maladaptive patterns of behavior (e.g., antisocial personality, passive-aggressive personality, and the like), which are qualitatively different from psychotic or anxiety disorders (American Psychiatric Association, 1980). It is interesting to note that the schizoid personality has been reported only rarely in retarded persons. In our recent clinical work, we diagnosed and treated a number of such persons. The personality disorders occurred in individuals whose behavior was based primarily on extrinsic factors; none had causal relationships to the symptom of mental retardation. The presence of personality disorders can be an allied disability for mentally retarded persons.

Anxiety Disorders

Early reviews (Beier, 1964; Garfield, 1963) of the occurrence of psychoneurotic disorders in retarded persons suggested that their frequency was quite low. The type of psychoneuroses reported was limited to such disorders as anxiety and depressive reactions. Recent reviews (Balthazar & Stevens, 1975; May & May, 1979) however, dispute the concept of the incompatibility between neuroses and retardation. They are quite explicit regarding the diagnostic criteria and attribute the neurotic phenomena to factors associated with atypical developmental patterns and disturbed family functioning. For example, anxiety disorders in retarded children clearly link symptoms of anxiety (e.g., fear of failure and insecurity) to external factors, such as chronic frustration, unrealistic family expectations, and persistent interpersonal deprivations. Interestingly, these reports suggest that psychoneurotic disorders are more common in individuals in the high-moderate and mild levels of mental retardation. This finding has prompted speculation that the complexity of psychoneurotic transactions is beyond the adaptive limits of more severely retarded persons.

Adjustment Disorders of Childhood, Adolescence, or Adulthood

Although the category of adjustment disorders is perhaps overutilized in the assessment of nonretarded persons, it is only infrequently employed during the clinical assessment of emotional disturbances in the retarded population. Because mentally retarded individuals have a predisposition to overreact to stimuli and a limited understanding of social-interpersonal expectations, they are more likely to exhibit personality disorganization after minimal interpersonal stress. The adjustment disorders may be caused by continuing inappropriate social-adaptive expectations or unexpected and frequent changes in externally imposed life patterns. Clinically, these disorders respond rapidly to environmental adjustment (when coupled with the realignment of parental, residential, and educational personnel's expectations and goals), brief utilization of psychopharmacological adjuncts, supportive counseling, or a combination of these treatments.

Depressive Disorders

An area that has attracted little attention until recently is the appearance and recognition of depression in persons with mental retardation. Yet, if we consider the language and communication problems of retarded persons as well as their limited social skills and lack of social networks, it make sense that the retarded person is highly at-risk to develop depressive reactions (Berman, 1977; Matson, 1982a,b; Schloss, 1982). In our experiences, persons often will develop severe behavioral problems that are due to the loss of loved ones or to transitions in family life. Caregivers may misunderstand these behavioral problems unless they are sensitized to the full sentient nature of persons with mental retardation and to the difficulty that many of them have in dealing with abstract phenomena. Indeed, the retarded persons may display signs of depression different from the nonretarded population.

In summary, persons with mental retardation, because of their high incidence of central nervous system impairment and their diminished interpersonal coping abilities, present a greater than average risk for developing associated mental illness. The reported frequency of mental illness in mentally retarded individuals suffers from the myopia of the reporters. As May and May (1979) noted, well-recognized forms of major personality dysfunctions (i.e., the symptom clusters noted in alcoholism or suicide) are only rarely reported in the current literature on the dual diagnosis of mental retardation and mental illness.

Conclusion

Caregivers need to understand the at-risk nature of persons with mental retardation that is related to a variety of factors, both intrinsic and extrinsic. Many of the predisposing conditions can be eliminated through ongoing supports that prevent mental illness. Others may be "given" predispositions that have to be worked around and dealt with in a compassionate, caring, and respectful way. Just as important, caregivers must understand that any clustering of behaviors, which equates with a major mental illness, requires active intervention; that such intervention requires the teaching or reteaching of new behaviors; and that persons with these needs require more integration and support, not less, if they are to live in the confluence of family life. They require sensitive caregivers and often a restructuring of their personal and social reality to help them pass through times of acute stress.

The caregiver is essentially responsible for in-

tervening at points of risk in a person's life to help that person make the transition from at-risk points to emotional stability. At these moments, the caregiver becomes the person's emotional mediator.

Understanding the nature and underlying causes of mental illness in the mentally retarded is only the beginning. It provides the caregiver with insight into the person's problems and a roadmap for ongoing care and treatment. Regardless of the types or intensity of behaviors, the caregiver's fundamental goal should be to break through whatever avoidant, self-injurious, disruptive, or destructive behaviors might exist, and to move that person respectfully and gently from these states to human interdependence. Clearly, persons with the dual diagnosis of mental retardation and mental illness present a persistent challenge to the clinician and require specialized service models if their needs are to be appropriately met. These persons can be served appropriately if a range of psychiatric, medical, educational, vocational, and residential services and supports are available. On the surface, the continuum of care for this population appears to be no different from that utilized for the mentally retarded population in general. However, the services differ in their treatment intensity relative to staffing competency, staffing patterns, and the availability of psychiatric support services. Professionals need to develop specific acute care, subacute care, and ongoing long-term management approaches that utilize a balanced range of treatment techniques to ensure the optimal social reintegration of these complex retarded persons.

Mental retardation is a given. Some of the physiological causes of mental illness may be givens. The question is: How can caregivers bring persons with mental retardation from a disengaged state—a state in which their behaviors block them from meaningful human interactions—to a state in which they can participate fully in family and community life? The entire need for change does not rest with the person with mental retardation. Caregivers need to discover personal and social change strategies to help these persons with special needs to live fully in family and community life.

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CHAPTER 31

A Developmental Perspective on Eating Disorders and Eating Problems

Ilana Attie, Jeanne Brooks-Gunn, and Anne C. Petersen

A great deal of research and theory has elucidated the psychological, biological, and social correlates of eating problems and eating disorders that emerge during adolescence. However, with few exceptions (Strober & Yager, 1985), investigators have not brought a developmental perspective to the study of dieting, bingeing, or the more severe clinical syndromes of anorexia nervosa and bulimia. The absence of developmentally oriented research in this area is unfortunate, given that these eating problems tend to have their onset during early to middle adolescence, are strongly gender- and class-related, and are closely tied to the biological and psychosocial changes that occur during the adolescent period.

A developmental understanding of eating problems and eating disorders would be greatly enhanced by the application of concepts and methods from the emerging field of developmental psychopathology, an investigative approach concerned with the reciprocal relations between development and psychopathology (Cicchetti, 1984), or the connections and lack of connections between normal development and patterns of maladaptation as these emerge and change throughout the life span (Rutter, 1986). A developmental perspective on the study of psychopathology takes into account the continuities and discontinuities between normal growth and psychological disorder, age-related changes in modes of adaptation and symptom expression, behavioral reorganizations that occur around salient developmental challenges, internal and external sources of competence and vulnerability, and the effects of development on pathology and of pathology on development (Carlson & Garber, 1986; Cicchetti & Schneider-Rosen, 1986; Ebata, Petersen, & Conger, in press). Integral to this approach is the use of prospective research designs in which individuals at risk are followed as they negotiate developmental tasks and major life transitions.

The emergence of eating disorders may be considered in light of several adolescent developmental issues. Continuities and discontinuities in development may be particularly highlighted during early adolescence (Hamburg, 1980). For the psychologically and perhaps biologically vulnerable female adolescent, the pubertal transition may be a discontinuous experience with regard to phys-

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ical transformations of the body, the changing experience of the self, and the unanticipated responses and demands of the social environment. Multiple new challenges come together during these years, possibly creating susceptibilities to stress on several fronts, including the school, peer relationships, and the adolescent's role within the family and the larger society (Brooks-Gunn, 1987; Brooks-Gunn, Warren, & Rosso, 1987; Hamburg, 1974, 1980; Petersen, 1987; Simmons, Blyth, Van Cleave, & Bush, 1979). Late adolescent challenges include the establishment of intimacy, pursuit of education and an occupation, and the development of an identity apart from the family. For the adolescent girl, there also are gender-related sources of vulnerability, such as a culturally mediated focus on body shape and weight, conflicts between social role demands and interpersonal focus of female identity formation, and an increased risk of depression (Attie & Brooks-Gunn, 1987; Hill & Lynch, 1983).

It is our premise that eating problems are most likely to emerge in adolescence because of the convergence of physical changes and psychosocial challenges with which the individual must cope. Adolescents girls who are vulnerable, either because of predisposing characteristics (biological and intrapsychic) or the particular context in which they find themselves (e.g., enmeshing and/or distancing family systems; social and vocational environments in which a great deal of emphasis is placed upon maintaining a low weight), may respond to developmental challenges with efforts to control their weight, or with an eating disorder. We can only speculate as to why eating disorders are a peculiarly female condition; even depression, which affects women twice as often as men across cultures and age groups, is not comparable to the syndromes of anorexia nervosa and bulimia, which affect women almost exclusively (Pyle, Mitchell, Eckert, Halvorson, Neuman, & Goff, 1983; Strober, 1986). Also, why a particular individual or group of individuals is vulnerable to eating disorders rather than to other psychosomatic syndromes or to depression is not well understood, since direct comparisons among such groups are rarely made.

In this chapter, eating disorders and problems are considered within a developmental perspective. Beginning with a selective review of the literature on anorexia nervosa and bulimia, a broad, biopsychosocial framework is presented for understanding eating disorders. Then, eating problems that fall within the "normal" range, that is, dieting and binge-eating patterns that are widespread among adolescent and young adult women, are discussed. Finally, the controversy surrounding the question of continuity between presumably normative concerns with weight and dieting and the clinical eating disorders is addressed: Although the majority of young women have attempted to diet to control their weight, why do only a fraction of these develop a severe eating disorder?

Eating Disorders

Diagnosis and Prevalence

The two major eating disorders are anorexia nervosa and bulimia. *Anorexia nervosa* is an eating disorder characterized by behavior directed toward weight loss, intense fear of gaining weight, body image disturbance, amenorrhea, and an implacable refusal to maintain body weight (American Psychiatric Association, 1987). Occurring predominantly in females (90% to 95%), it is one of the few psychiatric disorders that can have an unremitting course resulting in death (Halmi, 1980). *Bulimia* is an eating disturbance characterized by intense concern about weight, recurrent episodes of excessive overeating accompanied by a subjective sense of loss of control, and the use of vomiting, exercise, and/or purgative abuse to counteract the effects of binge eating (Fairburn & Garner, 1986). Accumulated evidence from clinical studies suggests that some form of binge eating occurs in varying proportions of normal weight, underweight, and obese populations (Wardle & Beinart, 1981). Certain characteristic features of an eating "binge," such as an alternating pattern of dietary restriction and overindulgence in food, are similar across all weight groups. Other features, namely, vomiting and purging, are more common among psychiatric patients with anorexia nervosa and bulimia; they are less frequently mentioned in the literature on obesity. Accordingly, "binge eating," which refers to episodes of uncontrollable and excessive eating, is now being distinguished from the syndrome of "bulimia," which, in addition to current eating binges, also includes vomiting as well as a "fear of fatness" (Cooper & Fairburn, 1983). The relatively recent discovery of bulimic eating patterns among high proportions of anorectic patients (ranging from 30% to 50%) has raised the issue of heterogeneity within this diagnostic category (Casper, Eckert, Halmi, Goldberg, & Davis, 1980; Garfinkel, Moldofsky, & Garner, 1980). In an effort to resolve the current controversy over the role of bulimia in this presumed "self-starvation" syndrome, a growing body of research has focused on the documentation

of bulimic and restricter subtypes of anorexia nervosa, and the possible distinct premorbid, clinical, and prognostic features of each.

Although voluntary emaciation or gorging are not new phenomena, social and cultural pressures have come together to define anorexia nervosa and bulimia as diseases of modernity (Boskind-White & White, 1986; Brumberg, 1985). Good epidemiological data are rare, and the possibility of bias exists from referral or exposure factors; however, available evidence suggests that both of these disorders have been on the rise over recent decades (Lucas, Beard, Kranz, & Kurland, 1983; Strober, 1986). In one survey of nine girls' schools in England, Crisp, Palmer, and Kalucy (1976) estimated the prevalence of anorexia nervosa to be 1 in every 200 girls under the age of 16, and 1 in every 100 girls over the age of 16. Recent studies suggest that higher socioeconomic status confers increased vulnerability to the development of eating disorders, independent of race or ethnic background (Andersen & Hay, 1985). Among groups under vocational pressure to control body weight, such as ballet dancers, the prevalence increases several-fold, with estimates ranging from 5% to 7% of adolescent and 30% of adult dancers (Garner & Garfinkel, 1980; Hamilton, Brooks-Gunn, & Warren, 1985). Prevalence estimates for bulimia, independent of anorexia nervosa, range from around 2% of women in a community sample (Cooper & Fairburn, 1983), to about 4% or 5% in white high school and university samples (Crowther, Post, & Zaynor, 1985; Katzman, Wolchik, & Braver, 1984; Pyle *et al.*, 1983). In contrast, about .04% to 1% of male college students are bulimic (Halmi, Falk, & Schwartz, 1981; Pyle *et al.*, 1983). Although the modal age of onset occurs during adolescence, the risk for developing an eating disorder extends well into adulthood (Pope, Hudson, Yurgelun-Todd, & Hudson, 1984). Anorexia nervosa occurs modally at times of developmental transition: at the threshold of sexual maturity, and during late adolescence, prior to and during the adolescent's separation from the family (Halmi, Casper, Eckert, Goldberg, & Davis, 1979). Bulimia more commonly occurs during the passage from late adolescence to young adulthood (Wooley & Kearney-Cooke, 1986).

The Heterogeneity of Anorexia Nervosa

In clinical studies of anorexia nervosa, distinct personality characteristics have been found to correspond to contrasting patterns of eating behavior, differentiating two subgroups of anorectics: (1) dieters, or restricters, who continually restrict their food intake, and (2) binge eaters, or bulimics, who alternate between binge eating and either vomiting, food restriction, and/or purgative abuse to maintain weight loss (Casper *et al.*, 1980; Garfinkel *et al.*, 1980). In one study, when compared with restricters, bulimics scored higher on measures of anorectic symptomatology, premorbid obesity, depression, and alcohol abuse (Strober, 1981). They also were distinguished by the following characteristics: greater premorbid distress manifested in anxiety, affective instability, and interpersonal difficulties; higher levels of conflictual family interaction; greater alienation from fathers than mothers; higher levels of marital discord; a higher incidence of disorganizing life stress preceding onset of illness; and, finally, greater psychiatric morbidity and health problems in parents. Parent psychiatric diagnosis, life stress, family cohesion, and premorbid unhappiness constituted the best predictors of group membership. Greater disturbance of body image and more pronounced external control expectancies have been observed in anorectics who binge and vomit (Strober, 1982, 1983).

In one study, Garner, Garfinkel, and O'Shaughnessy (1985) compared restricter and bulimic anorectics with normal weight bulimic women who had never met the criteria for anorexia nervosa. The normal weight bulimic group closely resembled the bulimic anorectic group, and the two bulimic samples could be distinguished from the restricter patients on the basis of demographic, clinical, and psychometric variables. The bulimic groups were more likely than the restricter group to have a history of stealing, alcohol and drug use, self-mutilation, suicide attempts, and labile mood. They also reported greater family conflict and dyscontrol, and a higher incidence of paternal obesity. These authors conclude that a history of bulimia may have greater diagnostic significance than a history of emaciation.

Theories of Etiology

The genesis of anorexia nervosa and bulimia nervosa has been explained from diverse clinical perspectives, including psychodynamic, cognitive-behavioral, family systems, and neuroendocrine approaches. Even though each of these theoretical models has contributed significantly to our understanding of these disorders, no single model satisfactorily explains their etiology, onset, and maintenance. The more developmentally oriented theories

have emerged from family systems and psychodynamic perspectives. Several risk factors, including individual personality development, specific family contexts, biogenetic predispositions, and sociocultural influences may contribute, to greater and lesser degrees, to the development of a child vulnerable to eating disorders (Garfinkel & Garner, 1982; Johnson & Maddi, 1986; Katz, 1985).

Psychodynamic Perspectives

The personality development of the child vulnerable to eating disorders has been discussed from several psychodynamic perspectives, including ego psychology, object relations, and self psychology (Bruch, 1973, 1978; Geist, 1985; Goodsitt, 1985; Rizzuto, Peterson, & Reed, 1981; Sugarman & Kurash, 1982; Sugarman, Quinlan, & Devenis, 1981; Swift & Letven, 1984). Each of these theories is essentially developmental in nature, attributing etiological significance to developmental arrests occurring in early infancy and childhood. Researchers typically have not integrated psychodynamic constructs into the scientific study of normal development and psychopathology.

Bruch (1973, 1978) has been credited with providing a major impetus for the contemporary psychodynamic theorizing on anorexia nervosa. Working out of an ego psychological perspective, she was the first to recognize the relentless pursuit of thinness as a central organizing feature of this disorder, altering the theoretical understanding of anorectic symptoms by stressing the female patient's equation of thinness with her sense of identity and of ownership of her body. Bruch (1973) identified three core clinical features of the anorectic syndrome: (1) a disturbance of body image and of body concept of possibly delusional proportions; (2) a distortion of perceptual and cognitive interpretations of internal feeling states related to bodily and affective needs; and (3) a paralyzing sense of ineffectiveness, related to the belief that one is acting only in response to demands from others. According to Bruch, these ego deficiencies result from chronically disturbed mother–daughter interactions that leave the adolescent with a pervasive feeling of limited autonomous control and an underdeveloped sense of self. The anorectic's characteristic strivings for perfection and superior achievement are understood as one manifestation of her overcompliant or "false self" adaptation (Winnicott, 1965), a defense against parental overcontrol and nonrecognition of the child's uniqueness and emotional needs. Bruch believes that these ego vulnerabilities have been present throughout the childhood of the anorectic individual and become manifest during adolescence because of its developmental demands for greater psychological autonomy.

Some psychoanalytic authors view anorexia nervosa as a major form of narcissistic or self-pathology (Kohut, 1971), arising from a chronic disturbance of parental empathic responsiveness—a disturbance which precludes the development of the child's sense of wholeness, vitality, and self-esteem (Geist, 1985). The absence of internal self-regulating structures for self-soothing, for the modulation of mood and anxiety, and for the maintenance of a sense of well-being and security are believed to be the pathological outcome of persistent failures of parental empathy, which ordinarily fosters the internalization of those parental functions in the formation of psychic structure. Research in this area is lacking (Geist, 1985).

Family Theories

Family theorists have conceptualized anorexia nervosa as a disorder that maintains the illusion of harmony and perfection and avoids conflict in "psychosomatic families" (Minuchin, Rosman, & Baker, 1978). Two observational studies have focused on the role of anorectic psychopathology in maintaining family homeostasis and stability. From a structural family perspective, Minuchin and his colleagues identified five predominant characteristics of interaction in families with an anorectic member: enmeshment, overprotectiveness, rigidity, lack of conflict resolution, and unresolved marital conflict (Minuchin *et al.*, 1978; Sargent, Liebman, & Silver, 1985). Selvini-Palazzoli (1974) described similar patterns in Italian families of anorectics. Overall, families of bulimics have been thought to resemble families of anorectics with regard to the enmeshment, overprotectiveness, rigidity, and triangulating of their daughters. However, preliminary studies of family interaction suggest that overt conflict, miscommunication of affect, and indirect expressions of anger and hostility are more typical than closeness and overinvolvement in bulimic families (Humphrey, 1986). Moreover, sources of marital distress frequently include lack of emotional intimacy, alcoholism, and/or depression. These early studies suggest that families of bulimic as compared with nonbulimic eating disor-

dered adolescents tend to be more disengaged, chaotic, conflict-ridden, stressed, deficient in problem-solving, and unable to openly communicate both positive and negative feelings (Johnson & Maddi, 1986). However, in the absence of controlled prospective and comparative studies of family process, it is difficult to identify predisposing factors and to distinguish these from those that ensue following the onset of these disorders.

Biological Theories

The contribution of biological factors to the onset and development of eating disorders focuses on four areas: (1) genetic studies; (2) the relationship between eating disorders and affective disorders thought to have a biological cause; (3) the physiological consequences of self-imposed restriction of food and caloric intake, and of the alternation of food restriction with binge-eating, vomiting, purging, diuretic and laxative abuse; and (4) alterations of hypothalamic-pituitary-thyroid mechanisms, catecholamine metabolism, and endogenous opioid activity secondary to starvation and weight loss. Because a discussion of the pathophysiology of eating disorders is beyond the scope of this chapter (cf. Weiner, 1985), we focus on the first three areas.

Genetic Studies

Scott (1986) proposed that a genetic diathesis–stress paradigm may be a valuable model for understanding eating disorders. This paradigm considers the interaction between a biological predisposition toward an illness (the diathesis) and adverse environmental factors affecting the individual (termed "stress"). Depending on the strictness of diagnostic criteria and of methods used for establishing zygosity, between 44% and 55% of monozygotic (MZ) twins have been found to be concordant for anorexia nervosa. Regrettably, comparable data for dizygotic (DZ) twins were not available from these studies (Garfinkel & Garner, 1982; Nowlin, 1983; Vandereycken & Pierloot, 1981). Eating disorders may be overrepresented in "anorectic families." The prevalence of anorexia nervosa among sisters ranges from 3% to 10%, greatly exceeding the 1% prevalence rate among young females (Scott, 1986). In one study of 56 families, Kalucy, Crisp, and Harding (1977) found a history of significantly low adolescent weight, anorexia nervosa, or weight phobias in 27% of mothers and 16% of fathers. Increased rates of anorexia nervosa, bulimia nervosa, subclinical anorexia nervosa, affective disorders, and feeding disorders have been reported in the relatives of anorectic patients as compared with nonanorectic psychiatrically ill controls (Gershon *et al.*, 1983; Strober, Morrell, Burroughs, Salkin, & Jacobs, 1985).

Taken together, available evidence suggests that a predisposition to eating disorders may be familial in origin, although the nature of such a vulnerability, and the specific environmental influences that promote or inhibit its expression, are not well understood. However, in a disorder as disruptive to family life as anorexia nervosa, one is faced with the ubiquitous problem of disentangling cause from effect in the interpretation of retrospective findings.

Biological Predisposition for Affective Disorders

Signs and symptoms of depression, including sleep disturbance, irritability, inability to concentrate, low and highly variable mood states, and suicidal ideation are frequently noted in patients with anorexia nervosa and bulimia. Because some depressive symptomatology is negatively correlated with weight gain and decreases over time in treatment (Halmi, 1985), several reviews of the subject have addressed the question of whether depression is consequent to the gross physiological and psychological distortions that characterize these disorders, whether it should be regarded as a separate (and sometimes coexisting) entity, or whether anorexia nervosa and bulimia may be variant expressions of a primary mood disturbance (Altshuler, & Weiner, 1985; Halmi, 1985; Levy & Dixon, 1985; Swift, Andrews, & Barklage, 1986). The potential relationship between eating disorders and affective disorders remains a topic of continued interest and debate. Although affective illness and eating disorders may be related phenomena, the weight of evidence does not support the view of anorexia nervosa as an adolescent variant of depression.

Physiological Consequences of Eating Disorders

The importance of starvation in the perpetuation of the disorder cannot be overlooked. Starvation induces biological changes, both centrally and peripherally; these physiological abnormalities interact with psychological reinforcers to sustain the

disorder (Garfinkel & Kaplan, 1985; Katz, 1985). In the vulnerable adolescent, what begins as a weight-loss diet to enhance feelings of control and self-worth eventuates in a chronic course of self-starvation. The self-perpetuating role of starvation in anorexia nervosa is evident in its pervasive impact on behavior, mood, cognition, and perception (Garfinkel & Kaplan, 1985; Katz, 1985). For the anorectic, stringent dieting itself may become self-sustaining, fueled by a loss of control over eating and the satisfaction of seeing one's weight go down to ever lower numbers. Progressive weight loss is maintained by a phobic avoidance of body fat and of any weight increase. With progressive starvation, the stomach shrinks and gastric emptying is delayed, producing feelings of fullness, early satiety, and protrusion of the stomach upon eating. These factors, along with physiologically induced fluid retention and bloating, may reinforce the anorectic's fears and fantasies of being fat (Katz, 1985).

Changes associated with starvation include impaired concentration, loss of general interests, depressive symptomatology, social withdrawal, and a focus on food-related concerns (Garner, 1986). Starvation-induced physiologic changes include sleep disturbance, amenorrhea, hypotension, bradycardia, reduced core temperature, insensitivity to pain, loss of scalp hair, and lunago hair (Warren, 1986). There are also perpetuating factors in the psychosocial realm, such as changes in the family, loss of social skills and vocational interests, loss of friendships, and unresolved emotional conflict (Garfinkel & Kaplan, 1985). The effects of starvation are not specific to the eating disorders; they are seen in all starving individuals (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950). The consequences of overeating and undereating are thought to be related to the body's attempt to defend its preferred body weight in the face of powerful efforts to override this weight through starvation. According to the "setpoint" theory (Nisbett, 1972), each person has a biologically programmed optimum weight that is regulated by that person's individual setpoint or level of fat storage. Although a person's setpoint is complexly determined by multiple factors, genetic endowment and early feeding experiences are thought to be significant (Coates & Thoresen, 1978). Importantly, prolonged caloric restriction may produce starvation effects in individuals who are not emaciated, but who nevertheless are starving themselves by suppressing their weights below their bodies' physiological demands (cf., Garner, 1986).

Given the powerful physiological and psychological factors that perpetuate eating disordered behavior, the question arises: Can dieting and weight loss, in themselves, serve as precipitants for anorexia or bulimia in a nonspecifically vulnerable adolescent (Katz, 1985)? Or, alternatively, How may we distinguish the "normal" adolescent, who is preoccupied with weight and dieting and/or is struggling with developmental issues of autonomy and identity, from one who will eventually develop an eating disorder? The question of continuity between "normal" dieting behavior and eating disorders is far from resolved (Garner, Olmsted, & Garfinkel, 1983; Swift and Stern, 1982; Wooley & Wooley, 1985). The answer to these and other questions requires a developmental perspective.

The Developmental Emergence of Eating Problems

One of the implications of a developmental perspective for understanding eating disorders and eating problems concerns the importance of considering the emergence of these problems in the context of adolescent developmental challenges as well as the patterning and sequencing of life events confronting most individuals during this life phase (Brooks-Gunn, 1987). In the discussion that follows, we consider these issues with respect to the emergence of weight preoccupation, dieting, and binge-eating patterns among adolescent girls. Dieting is endemic in white adolescent girls, as from 50% to 80% of middle and upper school girls have been on a diet (Attie, Brooks-Gunn, & Warren, 1985; Huenemann, & Shapiro, Hampton, Mitchell, 1966; Nylander, 1977).

Cultural Influences

Evidence of an inverse relationship between social class and obesity (Stunkard, D'Aquili, Fox, & Filion, 1972) together with gender-related pressures to attain a thin body ideal point to the importance of sociocultural influences in mediating concerns about shape and weight (Garfinkel & Garner, 1982). Viewed from a historical perspective, the preoccupation with weight and thinness, particularly among middle to upper middle-class women in Western cultures reflects a relatively recent, yet growing, cultural ideological trend. In this country, the cultural standards for feminine attractiveness

have shifted away from the voluptuous, curvaceous figure of earlier eras (Bennett & Gurin, 1982). Discussing images of attractiveness portrayed in the media, Faust (1983) writes: "The lean, lithe look is the benchmark for feminine beauty today and it has been so for several decades. This 'prepubertal look' is presumably to be emulated by all women, regardless of age" (p. 115).

The correlations of weight problems with gender and affluence suggest that sociocultural attitudes about thinness and fatness may be intensified within certain social strata or subcultures (Striegel-Moore, Silberstein, & Rodin, 1986). Controlling for differences in actual levels of fatness, girls in the higher social classes in a national sample of youths aged 12 to 17 years showed a much greater desire to be thinner than did girls from lower social class backgrounds (Dornbusch *et al.*, 1984). Other studies suggest that adolescent girls in competitive social environments that emphasize weight and appearance may experience intensified social pressures to meet the thin ideal (Brooks-Gunn and Warren, 1985; Striegel-Moore *et al.*, 1986).

Proximate Influences

Several proximate influences upon the emergence of dieting and bingeing have been identified. These include the development of counterregulatory behavior, pubertal factors, body image changes, and family factors (Attie & Brooks-Gunn, 1987).

Counterregulatory Eating

Observations of clinical populations and self-reports by normal dieters reveal that stringent dieting is frequently followed by bouts of overeating or bingeing. Laboratory studies of normal "restrained eaters" have demonstrated that chronic dieting creates a vulnerability to "counterregulatory" eating behavior, that is, the disinhibition of dietary restraint in the face of high-calorie food. This counterregulatory behavior appears to be mediated by cognitions; experimental studies in normal weight and obese individuals have shown that the mere belief that one has overeaten is sufficient to trigger counterregulatory eating in the highly restrained eater (Polivy, 1976; Spencer & Fremouw, 1979; Woody, Costanzo, Liefer, & Conger, 1981). Likening the dieter's seemingly uncontrolled eating to a naturally occurring eating binge, Polivy (1976) suggested that a binge episode may be elicited by potent attributions, such as the dieter's perception that she has "blown" her diet.

Just as adherence to a diet regimen is cognitively controlled (often in terms of daily "quotas" imposed on food intake), so too the mere belief that one has transgressed one's diet is enough to break the dieter's resolve and trigger counterregulatory consumption. Other factors, such as anxiety, depression, and the belief that alcohol has been consumed, have been shown to disinhibit the restrained eater, leading to greater food consumption (Herman & Polivy, 1975; Polivy & Herman, 1976a,b). The nondieter, unconstrained by any diet boundary (and therefore more responsive to physiologic pressures), responds to affective discomfort or to stress as to other sympathomimetic experiences by decreasing food consumption (Herman & Polivy, 1984).

Of relevance to counterregulatory eating patterns is the notion that body weight is homeostatically regulated around a "set point" (Nisbett, 1972). Attempts to lose weight through dieting are notoriously ineffective, perhaps because many dieters are "fighting" their natural weight or set point. Indeed, dieting is positively, not negatively, associated with weight in middle to late adolescent girls (Brooks-Gunn & Warren, 1985). A person's set point may be important in determining who will diet and who will be successful. Individuals who are naturally thin may diet less and, if they gain weight, may find it relatively easy to lose. Persistent dieting and counterregulatory behavior may be more prevalent in women who are not naturally thin or in women who maintain very low weights for professional reasons (i.e., ballet dancers). Indeed, dancers report higher levels of dieting and bingeing in comparison with girls who are uninvolved with athletics; they also are much more likely to develop an eating disorder (Brooks-Gunn & Warren, 1985; Brooks-Gunn, Warren, & Hamilton, 1987; Hamilton *et al.*, 1985). Even among dancers, heterogeneity in eating problems may be linked to whether individuals are naturally suited to the profession; that is, whether they have a genetic predisposition toward slimness (Hamilton *et al.*, 1985).

Pubertal Factors

As girls mature sexually, they accumulate large quantities of fat in subcutaneous tissue, as indicated by increased skinfold thickness (Young,

Sipin, & Roe, 1968). For the adolescent girl, this "fat spurt" is one of the most dramatic physical changes associated with puberty, adding an average of 11 kilograms (24 pounds) of weight in the form of body fat. Increases in body fat during the pubertal years are associated with efforts to diet (Attie *et al.*, 1985).

Other pubertal changes may be associated with increased concerns about weight and efforts to control it. Menarche and breast development are two highly salient events of female puberty (Brooks-Gunn, 1984). We examined the relative contribution of different pubertal influences to the intensification of dieting in a sample of 7th- to 10th-grade girls, finding that dieting increased in relation to both menarche and breast development, independent of chronological age (Attie *et al.*, 1985).

From a psychological standpoint, the process of integrating changes in physical appearance, bodily feelings, and reproductive status may require a fundamental reorganization of the adolescent's body image and other self-representations (Blos, 1962). With their developing capacity for self-reflection, young adolescents may be uniquely preoccupied with their changing body selves and with the responses of others to these changes (Hamburg, 1980). For girls, body image is intimately bound up with subjective perceptions of weight; pubescent girls who perceive themselves as underweight are most satisfied, followed by those who think they are simply average (Tobin-Richards, Boxer, & Petersen, 1983). In our study of 7th- through 10th-grade girls, body image was more predictive of dieting than both maturational status and weight, suggesting that it may mediate the maturation–diet relationship (Attie *et al.*, 1985). Taken together, the findings suggest that dieting emerges as the body develops and is in part a function of body-image transformations occurring at puberty.

The Adolescent within the Family

The mother–daughter relationship is considered central to the development of a girl's body image and eating attitudes. A mother's comfort with her own body and sexuality, as well as her satisfaction with her role in the family and society, may be important for the daughter's development of a stable and cohesive body image (Orbach, 1986). Mothers, like their daughters, experience social pressures to meet the thin, fashionable ideal. Indeed, given their ages, mothers' bodies are less likely to fit current cultural aesthetics. Mothers' insecurities about their bodies, as well as conflict around eating and feeding, may be communicated to their daughters and be internalized by them. Interestingly, many young women believe that their mothers dislike their own bodies. In one survey of readers of *Glamour* magazine, admittedly a sample which may be more likely to adhere to media-inspired ideals than more nationally representative samples, only 13% of 33,000 respondents thought their mothers were satisfied with their bodies (Wooley & Wooley, 1985). To our knowledge, only one study has directly assessed the influence of maternal bodily and eating attitudes on daughters' problem eating in a nonclinical sample. Among 9th- through 12th-grade private school girls, mothers' self-reported preoccupation with thinness and disturbed eating behavior predicted similar eating and weight concerns in their daughters (Attie, 1987).

Not only may mothers' feelings about their own bodies affect their daughters, so may their feelings about their daughters' physical development. Mothers may transmit personal and cultural ideals and contradictions about motions of gender and femininity (Orbach, 1986). In a survey of bulimic behavior in college women, the belief that the mother was critical of the daughter's body was the second strongest predictor of body dissatisfaction and bulimic symptom scores (Debs, Wooley, Harkness-King, & Wooley, 1983, cited in Wooley & Wooley, 1985).

Few studies have addressed the role of paternal attitudes about weight or the father–daughter relationship in the development of eating problems in adolescent girls. However, we suspect that fathers' affirmations of their daughters' femininity and autonomy play an important yet different role in the development of the adolescent girl's body image and sense of self.

Eating Disorders and Developmental Psychopathology

In this chapter, the emergence of eating disorders and eating problems from a developmental perspective has been considered. Generally, we subscribe to the view that both find expression in the course of adaptation to stage-salient developmental tasks. For a variety of reasons, biological, social, and psychological, a child may be vulnera-

ble, lacking the competencies and supportive contexts to cope with the normative challenges of growing up. Different factors, or combinations of factors, may predispose the adolescent to develop anorexia nervosa, bulimia, or major depression. Subclinical eating problems may have yet a different etiology, especially since they are almost normative in some segments of the population.

Eating disorders and eating problems to a lesser extent are most likely to emerge at two different points: around the time of the pubertal transition and during the passage out of adolescence toward young adulthood. Their emergence during these two transitions may be understood from a developmental perspective. First, the onset of puberty may herald overwhelming developmental challenges for the vulnerable adolescent, who may have difficulty coping with all the simultaneous changes of this age period (Brooks-Gunn, 1987; Brooks-Gunn *et al.*, 1987). Second, the older adolescent vulnerable to an eating disorder may be more likely to develop bulimia than a younger counterpart. Most cases of bulimia emerge during the late teens and early twenties, in contrast to anorexia nervosa, which has a stable prevalence rate throughout adolescence. Whether leaving home and the issues surrounding this event are causal in the increased prevalence of bulimia is not known at this time. It is clear that bingeing and purging generally are influenced by social contagion; young women who live in dormatories or apartments may be more likely to witness such behavior than when they were living at home. However, little is known about the factors that predispose a young woman to develop bulimia or binging/purging behavior.

Despite the recent enthusiasm surrounding the emergence of developmental psychopathology as a unique model of inquiry (Cicchetti, 1984), the study of eating disorders has been virtually overlooked by researchers in this area (Rutter & Garmezy, 1983). We can only speculate about the reasons for this omission. One of the defining features of developmental psychopathology research is the attempt to delineate patterns of continuity and change as these manifest themselves in normal development, maladaptive behavior, and socialization across time (Sroufe & Rutter, 1984). In this context, we must consider an important theoretical and empirical question: Which developmental processes, psychosocial tasks, and/or specific stressors are pertinent to the developmental study of eating disorders? In the case of childhood depression, for example, researchers have examined the emergence of normal and abnormal mood states, psychobiological markers, age-dependent responses to such specific stressors as separation and loss, the development of a "depressive" attributional style, continuities between childhood and adult depression, and, in some children, the interaction with a depressed parent (Brooks-Gunn & Petersen, 1987; Rutter, Izard, & Read, 1986). An analogous set of developmental dimensions in eating disorders has yet to emerge in investigative efforts. However, recent work suggests at least two avenues for further inquiry.

The first is the application of a high-risk approach to the study of eating disorders. Sroufe and Rutter (1984) have stated that "risk research is in many ways paradigmatic developmental psychopathology" (p. 19). In anorexia nervosa, the low base rate (estimated at 1 per 100 adolescents) and the lack of clearly defined risk factors are some of the dimensions that may pose particular problems for prospective research (Bell, 1982). However, notwithstanding the potential limitations of the high-risk method (cf. Weintraub, Winters, & Neale, 1986), such an approach to the study of anorexia nervosa deserves serious consideration. Importantly, anorexia nervosa has a relatively short latency period (occurring almost universally in adolescence), making it accessible to longitudinal and risk designs. Furthermore, the current state of knowledge suggests both biological and psychosocial bases of risk, including female gender, high socioeconomic status, high intellectual achievement, and a family or personal history of eating disorders, obesity, affective disorder, and/or excessive focus on weight and bodily concerns.

Second, severe forms of disordered eating, and their relationship with normal development and with clinical eating disorders, need to be studied. There are similarities as well as differences between "weight-preoccupied" chronic dieters and patients with anorexia nervosa (Garner *et al.*, 1983; Thompson & Schwartz, 1982). The study of dieting and bingeing is especially relevant to the developmental psychopathologist, because these behaviors are prevalent among large proportions of adolescent and young adult women; they also are precursors to eating-related psychopathology. However, compulsive eaters have yet to be followed prospectively, in order to determine the continuities and discontinuities between eating problems and eating disorders. Further developmentally oriented research must also distinguish those adolescents who adapt to developmental challenges through control of their bodily experience, from those who develop other forms of developmental psychopathology.

ACKNOWLEDGMENTS

We wish to thank R. Deibler for help in manuscript preparation. The writing of this chapter was supported in part by the National Institutes of Health and the W. T. Grant Foundation.

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CHAPTER 32

The Elimination Disorders

Enuresis and Encopresis

Robert M. Liebert and Janet E. Fischel

Every one of us began life wetting the bed and making a "mess" of diapers on a daily basis. Our parents and others then imposed some form of toilet training on us. By various means, they taught us when to eliminate and when to withhold urine and feces—and they insisted that we learn to comply with the rules. We were not alone. In a classic study of 22 cultures, Whiting and Child (1953) concluded that toilet training is the most basic and universal target of socialization everywhere. Moreover, bladder and bowel training are closely linked. That is, parents almost always begin training for urination and defecation concurrently and generally report that children stop soiling and become dry at about the same time, regardless of whether it is "early" or "late."

It is also interesting that virtually every culture appears to succeed in toilet training 80% to 90% of its new members within the expected time limit. The few who remain untrained, or who become trained but then relapse, are said to have a *disorder of elimination*. Elimination disorders, particularly simple bedwetting (the most common form), were among the first behavioral problems to be recognized by human societies; early cave drawings depicted the problem, and ancient physicians were discussing it and concocting various treatments more than 3,500 years ago (Glicklich, 1951). Bedwetting is still of concern today in most societies, and there are recent discussions about etiology and treatment in cultures as diverse as the Ghanaian village and the Russian army (Danquah, 1975).

Our purpose in this chapter is to summarize what is known about the origins, diagnosis, and treatment of the elimination disorders. We have drawn on formal studies in the published literature and on our clinical experience and impressions. In addition, we have tried to point out, wherever possible, unresolved theoretical issues.

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Basic Physiological Mechanisms

In order to understand the elimination disorders and the methods used to treat them, it is necessary to begin with a general sense of the physiological mechanisms underlying urination and defecation.

Enuresis and the Physiology of Urination

The human bladder is actually a stretchy, hollow muscle (the *detrusor vesicae*) in which urine is collected continuously. As urine accumulates, the bladder stretches, producing an initial urge to urinate. The urge can be voluntarily suppressed for considerable periods of time by the age of 4 or 5.

Urine is evacuated through a canal-like structure (the *urethra*), which has valvelike muscles—the internal and external sphincters. Bladder emptying is a self-intensifying process, meaning that once an individual begins to pass urine, central inhibitory processes, which would otherwise work to maintain continence, are blocked. Thus, bladder control depends upon early detection of the impulse to urinate (i.e., before the voiding reflex has begun), so that the individual can either find an appropriate place to urinate or voluntarily prevent the internal sphincter from opening. Thus, bladder control can be thought of as synonymous with sphincter control, and can be seen to depend upon the early detection of cues from the bladder signaling that a significant amount of urine has accumulated. Successful treatment of enuresis is presumed to involve increasing the individual's sensitivity to these signals and/or increasing voluntary control over the relevant muscles.

Encopresis and the Physiology of Defecation

The digestive tract is a long hollow tube with the colon and the rectum at the distal end. After food is digested in the stomach and small intestine, the remaining waste products move through the colon and gradually shift from a liquid state to a semisolid one. When sufficient waste accumulates, muscle contractions move it down the colon and into the rectum. The resultant stretching of the walls of the rectum leads to the urge to defecate. Unless voluntarily controlled, this urge will lead to a relaxation of the external and internal sphincters and subsequent evacuation. It is easier to inhibit defecation than it is to inhibit urination, because the former requires voluntarily producing intra-abdominal pressure ("pushing") as well as muscle contractions in the colon.

Nomenclature and Prevalence

Inappropriate urination is referred to as *enuresis* and inappropriate defecation is called *encopresis*. Neither term is applicable until children have clearly passed the age at which members of their culture are expected to be fully continent. Most children in the United States are completely toilet trained by their fourth birthdays, so children over this age who are still wetting or soiling are said to have a disorder of elimination. In other cultures, toilet training may be much later or much earlier. For example, the Bena of Africa do not begin toilet training until their children are almost 5 years old, and often the children are not completely trained until they are 6 or 7; the Tanala of Madagascar, on the other hand, begin toilet training when their infants are only a few months old and expect full continence by the sixth *month* (Whiting & Child, 1953).

The Functional-Organic Distinction

In diagnosing elimination disorders a fundamental distinction is made between those that are *functional* and those that are *organic*. Organic disorders are caused by an underlying physical illness or abnormality, whereas functional disorders have no identifiable physical basis. Although organically caused elimination disorders are quite rare (accounting for less than 10% of all cases), they can be extremely serious. Therefore, every child with an elimination disorder should be checked by a physician before a functional diagnosis is given or psychologically oriented treatment is begun.

Prevalence

Table 1 presents a summary of what is known about the prevalence of the elimination disorders, and several points about these data are especially important. First, note the very high prevalence of primary nocturnal enuresis (simple bedwetting) relative to the other forms of elimination disorder. At age 7, 12% of the population is enuretic; of these, four fifths of all cases are of the primary type. Breaking the data down further according to gender, it can be seen that roughly 11.5% of all boys

Table 1. A Comparison of Enuresis and Encopresis[a]

	Enuresis	Encopresis
Prevalence at age 4	20%	3%
Prevalence at age 7	12%	1.5%[b]
Male/female ratio	60/40[c]	75/25
Primary/secondary ratio	80/20	50/50
Percentage nocturnal only	75%	0%
Percentage diurnal[d]	25%	100%

[a]Compiled from data reported in Achenbach and Edelbrock (1981), Bellman (1966), Forsythe and Redmond (1974), Levine (1975), and Miller (1973).
[b]About one third of all encopretics are also enuretic.
[c]The sex difference is due almost entirely to primary nocturnal enuretics; there appears to be no sex difference for the other types.
[d]Diurnal enuresis almost invariably involves some nighttime wetting; diurnal encopresis often does *not* involve nighttime soiling.

and 7.5% of all girls in the early elementary school years still have never been dry at night for any considerable time. They are primary enuretics. (The maximum periods of continuous dryness permitted in applying the diagnosis *primary* is not fully standardized in the literature, but varies between 2 and 6 months.) By way of contrast, the prevalence of any of the other elimination disorders in elementary school children ranges from less than ½% to no more than about 2.5%.

Co-Occurrence of Enuresis and Encopresis

Because enuresis and encopresis may stem from one or more common causes (e.g., struggles with parents over toilet training), it is of interest to ask whether enuresis and encopresis tend to co-occur. Inasmuch as the prevalence of enuresis is quadruple that for encopresis, it is obvious that the overwhelming majority of enuretics cannot be encopretic. But encopretic children *are* more likely to be enuretic than are other children (Bellman, 1966; Levine, 1975; O'Regan, Yazbeck, Hamberger, & Schick, 1986).

Spontaneous Remission

Virtually all of the parents with whom we deal have already spoken to a pediatrician about their children's elimination problem. Once organic causes have been ruled out, the usual advice they have gotten is to be patient, because the child will eventually outgrow the problem. Is this advice sound? As Table 1 shows, only one half of those suffering from an elimination disorder at age 4 will have it "take care of itself" by age 7; the probability of spontaneous remission does *not* accelerate during middle childhood (Houts & Liebert, 1984). This means that, on the average, an elementary school-aged child with a toileting problem has only about a 15% chance of seeing it resolved by itself within the succeeding 12 months. The best guess is that from any hypothetical starting age in middle childhood, it will take, on average, more than 3 years before the child becomes fully continent. Left untreated, a few unfortunate individuals will still be wetting or soiling in their late teens. Given this information, most parents and children prefer not to wait for a spontaneous remission to occur.

Nonretentive versus Retentive Encopresis

With encopresis, an important distinction is made between its *nonretentive* and *retentive* forms. Nonretentive encopresis is the diagnosis given to individuals who simply defecate in inappropriate places, when not accompanied by significant constipation or holding back of stool. For these individuals, bowel movements occur with normal frequency. Retentive encopresis is the term applied to those whose soiling is related to withholding stool; a history of constipation is common among these individuals. It is estimated that between 80% and 95% of all encopretics are retentive (Levine, 1975; Silverberg, 1983).

Retentive encopresis can become a vicious circle. In its normal state, the rectum is empty. However, when a person withholds stool voluntarily, the rectum and lower colon become filled with fecal material. If withholding continues, a huge quantity of stool will accumulate, and, as a result, the lower colon will become very distended. Concurrently, the body will absorb almost all the moisture from the fecal mass, leaving hard, impacted stool. In this condition, passing stool is extremely painful or impossible. The urge and ability to defecate are therefore significantly reduced. Fresh, moist stool from the upper colon now can no longer be passed at all, but a brown liquid from the fresh stool almost invariably leaks around the impacted mass, producing soiled underwear and a fecal smell.

Clinical Diagnosis

Thus far, we have described the elimination disorders in abstract terms. The data, however, are only as good as the clinical diagnoses on which they are based. Our enuresis/encopresis program at Stony Brook is a collaborative effort of the departments of psychology and of pediatrics. The approach to clinical diagnosis that we have evolved over the years is briefly summarized here.

We employ a highly structured initial consultation, including a thorough toileting history. We ask for such details as age of onset of the problem, correlates of onset (e.g., parental divorce, start of school, illness), and frequency of the problem. Usually, the history is taken in an interview fashion and, in addition to specific information, provides a sense of the family's ability to carry through a treatment plan in the home.

Our initial diagnostic goal for patients with either enuresis or encopresis as a presenting complaint is to rule out the presence of an organic disorder (or to make an appropriate medical referral). So, when parents report that their child has an elimination disorder, the first question we ask is whether

the problem has been brought to the attention of a physician? If the answer is yes, follow-up questions about physical examinations, laboratory tests, and history of pharmaceutical or surgical treatment are used to determine if the problem is organic rather than functional. We always request a physical examination if the child has not had one within the past year. We also find it essential to learn what the family is doing about the problem *right now* and to ask them to discontinue any treatments or practices that seem inappropriate.

Finally, the initial consultation provides our first opportunity for educating the family (e.g., by pointing out to the child that many other children have toileting problems and that they, too, have felt very embarrassed and done everything they could to keep the problem a secret).

Etiology

As mentioned earlier, enuresis and encopresis can be the result of specific physical illnesses and structural defects (see Kelalis, King, & Belman, 1985, and Levine, 1983, for reviews of the relevant literature). When organic causes are ruled out (as they are in 90% of all cases), the question of etiology becomes a matter of searching for environmental and psychological causes. Theoretical accounts of the elimination disorders are structured along parallel lines for enuresis and encopresis.

There is widespread agreement that functional enuresis and encopresis are *familial disorders;* they are closely associated with a child's family background. In fact, the two pieces of firm etiological evidence that we have about these disorders is that (1) they all run in families (Bellman, 1966) and (2) except for primary nocturnal enuresis, they occur more often in troubled families than in better-adjusted ones (Olatawura, 1973).

At the theoretical level, three broad etiological models have been suggested, with numerous variations on each of the basic themes.

The Psychodynamic Model

According to the psychodynamic formulation, toilet training is a psychological battle between child and parents; the battle must be resolved in the child's psyche before he or she becomes or remains fully continent. Inspired by psychodynamic thinking, researchers have shown that parent–child conflict is characteristic of the family with an incontinent child (Baird, 1975; Hoag, Norriss, Himeno, & Jacobs, 1971).

Baird (1975), for example, described four characteristic features of the family interaction pattern of encopretic children as infantilization, mishandled anger, miscommunication, and withholding of emotion. Other reports highlighted the weakness, absence, or negative influence of father figures (Bemporad, Pfeifer, Gibbs, Cortner, & Bloom, 1971; Schaengold, 1977). Although these data obviously bear on etiology, they are causally ambiguous. It is possible to interpret the correlation between parent–child conflict and elimination disorders in psychodynamic terms, by assuming that the interpersonal conflicts cause the elimination disorders. But there is also evidence that these disorders can sometimes be the cause rather than the effect of parent–child conflicts (Vivian, Fischel, & Liebert, 1986).

The Learning Deficit Model

According to the learning deficit model, individuals with elimination disorders have not received the amount or kind of training they need to become continent. Within this model, the focus is on toilet training *per se,* rather than on broader or "deeper" aspects of the parent–child relationship. The conceptualization of toilet training is that the parents are teachers and the child is a learner. Failure to become continent may occur because the parents used inadequate teaching methods and/or because the child was an unusually slow learner or learned aberrant elimination patterns. In either event, the child is presumed to have not yet learned to notice and respond to bodily cues indicating the need to eliminate.

The Improper Diet Hypothesis

The third etiological model of elimination disorders is the improper diet hypothesis. According to this view, children may be incontinent because of deficits or excesses in their intake of food or beverages. For example, it has been hypothesized that the vicious cycle of retentive encopresis often has its origins in diets that are too bland or contain inadequate roughage, which, in turn, will lead to constipation. In a parallel fashion, enuresis is sometimes blamed on excess fluid intake during the hours just before bedtime.

Predictably, the three etiological formulations lead to different methods and techniques of treatment.

Treatment

In establishing treatment goals and evaluating treatments, an important distinction must be made between a treatment's ability to stop wetting or soiling (*initial arrest*) and its ability to overcome the problem permanently (*lasting cure*). Some treatments are initially quite effective but are associated with high relapse rates; others work only as long as they are being actively administered.

Enuresis

Drug Treatment for Enuresis

A remarkable array of powerful, controlled substances have been been held out as possible treatments for enuresis. These include stimulants, monoamine oxidase (MAO) inhibitors, sedative-hypnotics, major tranquilizers, anticholinergics, tricyclic antidepressants, and even pituitary snuff (Blackwell & Currah, 1973). The only pharmaceuticals shown to be superior to placebo preparations in double-blind controlled drug studies are the tricyclic antidepressants (Forsythe & Merrett, 1969; Shaffer, Costello, & Hill, 1968). Imipramine hydrochloride (Tofranil) is by far the most widely used of the tricyclics for bedwetting, probably because it is the least likely to have a soporific effect. Imipramine produces a noticeable reduction in bedwetting for between 30% and 50% of enuretic children; the therapeutic effect usually appears within 10 days or less (Black, 1983). Unfortunately, for the overwhelming majority of children, tricyclic medication will not totally eliminate bedwetting even while the drug is being taken. Moreover, as many as 90% of all bedwetters treated with these drugs will relapse when their medication is withdrawn (Blackwell & Currah, 1973). Despite its inadequacy as a permanent cure, and the occasional serious side effects which attend its use (Werry, Dowrick, Lampen, & Vamos, 1975), imipramine is by far the most common professionally sanctioned treatment for bedwetting in the United States.

Fluid Restriction and Enuresis

Restriction of liquids for one to several hours before bedtime is the remedy most parents seize on first to treat their enuretic children. Ninety percent of the parents in the Stony Brook Enuresis Project who reported trying a restriction of liquids concluded that it did not help. Moreover, despite the occasional popular article about diet, allergies, and bedwetting (e.g., Kelly, 1985), in almost no instances can bedwetting be traced to diet, nor is there any evidence that drinking large quantities of cranberry juice, eliminating dairy products from a child's diet, or any other popular dietary remedy has any more than a transient placebo effect for a few of the most suggestible children.

The Urine Alarm

For enuresis, the core of all the successful behavior management plans is the *urine alarm,* a device which sounds an alarm to wake the sleeping bedwetter as soon as urine is passed. Although the idea was first introduced in the nineteenth century (Coote, 1965), Mowrer and Mowrer (1938) are often credited with developing the urine alarm and are certainly the ones responsible for popularizing it.

The Mowrers reported a cure rate of 100% for the 30 children described in their classic report. Subsequent studies have produced more modest results, but, in general, 75% of bedwetters can expect to be dry within 3 months of conscientious application of the urine alarm (Doleys, 1977). However, the classic urine alarm treatment has three basic limitations.

First, until a few years ago, urine alarms were clumsy and somewhat unreliable mechanically; the original models all consisted of a large bell, which had to be placed on a nightstand near the child's bed, and a special multilayered pad of metal foil and insulating material, which had to be placed on the bed (hence the common name "bell-and-pad"). A far superior version is the wearable alarm, which weighs less than an ounce, is smaller than a package of gum, and operates with almost complete reliability on hearing-aid batteries.[1]

A second basic limitation of the classic urine alarm was the long time required to achieve initial arrest; not only must the child and parents endure a treatment that often lasts ten weeks or more, but it was typical for no progress at all to occur during the first month of treatment. (Failure to comply with the procedures in a diligent manner on a continuing basis appears to be the single biggest reason for the failure of urine alarm treatment. Probably, this is because the family must make a big, "up front" investment of time and energy before seeing any results.)

[1] We have been using the Wet-stop alarm, manufactured and distributed by Palco Labs, 1595 Soquel Drive, Santa Cruz, California 95065.

Finally, the urine alarm has a high relapse rate. As many as 40% of those bedwetters who are seemingly cured will relapse within the year after treatment and require retreatment (Bollard, 1982). Researchers have therefore sought ways to strengthen the urine alarm procedure or to replace it. The former attempts have enjoyed some good successes, the latter attempts have not.

Overlearning

Young and Morgan (1972) introduced an adjunctive antirelapse procedure to urine alarm training that they called *overlearning*. The procedure requires children who have achieved a preset criterion of dryness with the urine alarm (e.g., 14 dry nights in a row) to drink a significant quantity of water every night in the hour before retiring while continuing to use the alarm. Overlearning continues until the child is able to remain consistently dry through the night even with the liquid overload. In their experimental comparison, Young and Morgan found a relapse rate of about 35% for those who had not received overlearning as opposed to a relapse rate of less than 15% for those who had.

Bladder Stretching and Retention Control Training

On the average, bedwetting children have smaller functional bladder capacities than nonbedwetters (Starfield, 1967). Inasmuch as the bladder is a "stretchy" material, bladder capacity can be increased by regularly filling the bladder to its greatest capacity and keeping it full for longer and longer periods of time. The effect is much the same as would be produced by repeatedly filling a balloon with water in order to stretch it. Research has shown that exercises requiring children to drink and then hold substantial quantities of water can help some of them to achieve dryness (Kimmel & Kimmel, 1970; Starfield & Mellits, 1968); used by themselves, such procedures have a cure rate of less than 30%, but they appear to enhance the urine alarm significantly (Houts & Liebert, 1984).

Dry-Bed Training

Dry-bed training (Azrin, Sneed, & Foxx, 1974) is a very intensive procedure employing the urine alarm, a demanding "nightly waking schedule," and a response cost component called "positive practice," which is invoked whenever the child wets the bed. The child is also made responsible for his wet clothing and linens and is expected to make his own bed, a component of the program which is called "cleanliness training."

In dry-bed training, the price paid for an accident is being made to lie on the bed and count to 50, and then to get up and go to the bathroom and try to urinate. Children are made to repeat this sequence 20 times in a row whenever they wet their beds. Positive practice is also required of the child 20 times just before going to bed on nights following a night in which an accident has occurred.

The nightly waking schedule requires that the child be awakened every hour during the first night of training, taken to the bathroom, asked to urinate, and then be "loaded" with fluids before returning to bed. On the second night, the child is awakened 3 hours after going to bed; if dry, the child is awakened 2½ hours after going to bed on the third night; if dry, he or she is awakened 2 hours after going to bed on the fourth night, and so on, so that the nightly waking schedule terminates itself as long as the child remains dry. If the child wets twice in one week, however, the entire schedule starts over again.

Averaging across a number of studies, the complete dry-bed program (whether administered by a professional home trainer or by parents themselves) produces initial arrest in about 90% of all cases, requires an average treatment time of about 6 weeks, and has about a 25% relapse rate. Thus, it is quicker and more successful than the urine alarm alone. When dry-bed training is employed *without* a urine alarm, however, initial arrest occurs in only about 60% of all cases, and typical length of treatment is extended to 10 weeks. Therefore, without the urine alarm, dry-bed training appears to be not much quicker and somewhat less effective than a urine alarm alone (see Houts & Liebert, 1984, for a complete review of the literature).

The major drawback of the complete dry-bed program is low family acceptance. For example, in one study (Bollard & Nettelbeck, 1981), 60% of families dropped out prematurely. In another study (Fincham & Spettell, 1984), parents who carried out urine alarm training rated the program much more favorably than those who carried out dry-bed training.

Full-Spectrum Home Training

Full-spectrum home training is a newer behavior management plan for the treatment of enuresis (Houts & Liebert, 1984; Houts, Liebert, & Pad-

awer, 1983). Like dry-bed training, it was conceived to enhance the effects of urine alarm training by the inclusion of additional components. The treatment is built around a parent manual and incorporates retention control training and overlearning procedures as well as an Azrin-type cleanliness-training component. These components are organized into a contract between parents and children ("the family support agreement"); all procedures are home-implemented by parents, following a single 1-hour training session, which can be administered individually or in groups. Full-spectrum home training has been shown to produce an initial arrest of slightly more than 80% in an average of less than 10 weeks; the relapse rate is under 25% (Houts, *et al.*, 1983). Direct experimental comparison has shown it to produce significantly more rapid initial arrest and a lower relapse rate than a urine alarm alone (Houts, Peterson & Whelan, 1986). Parent and child acceptance of the program is high and, in our experience in New York and Tennessee, dropouts are rare. Thus far, however, there have been no published replications by other researchers.

Encopresis

Encopresis treatments have received nowhere near the research attention that enuresis treatments have enjoyed, perhaps because encopresis is the less common and more variable disorder. Although a variety of treatments have been tried for encopresis (see Doleys, 1983, for a review of the full range of techniques, from mechanical signaling devices to psychotherapy), the most successful efforts to treat encopresis all appear to be founded on behavior management principles used in conjunction with laxative and dietary prescriptions (Levine, 1983; Wright & Walker, 1978). These behavior management plans differ from one another in their particulars, but all seem to include the same five components; namely, record keeping, scheduled toilet sitting, structured toilet-skill training with systematic reinforcement of appropriate behaviors, family education about the problem, and laxative and/or dietary intervention.

In the remainder of this section, we describe the behavior management plan for encopresis that has evolved out of our clinical work at Stony Brook and explain the rationale for each of its components. It has not been possible to come up with the type of fixed treatment package for encopresis that has prevailed in the treatment of enuresis. In our experience, a custom-tailored plan is required for each family because every child has his or her individual training needs. (For example, the child's customary soiling times and places must be taken into account in scheduling laxative use.)

Ensuring Soft and Frequent Stool Passage

For retentive children, our first goal is to clear the rectum and lower colon, using enemas or suppositories as needed. Dietary changes to increase roughage or bulk and a daily schedule of laxatives are frequently established to ensure soft stool and to enhance the urge to defecate. Also, we begin immediately to help the parent and child understand the basic physiology of defecation and carefully explain the rationale for each step in our program.

Record Keeping

As with enuresis, record keeping for encopresis is instituted at the end of the first visit and is continued throughout treatment. The record is usually in the form of a daily calendar on which the parent and child monitor two things: (1) the occurrence and time of day of soiling incidents and (2) the occurrence of each *successful* toileting episode. The child and parent are asked to do all record keeping together.

Scheduled Toilet Sitting

It is important to establish a daily schedule of toilet sitting times for the child. Ordinarily, two to four sits a day are required. The first daily sit should occur just after breakfast. In scheduling the others, attention should be given to relevant aspects of the child's routine. For example, if a laxative is given in the morning, a toilet sit in the middle or late afternoon is usually desirable.

Systematic Reinforcement

Consistent with previous literature (Levine & Bakow, 1976; Wright & Walker, 1978), our primary behavioral technique for controlling encopresis is systematic reinforcement of appropriate behaviors. The preferred reward is immediate social praise, which is always given for successful stool passage on the toilet and for appropriate toilet sitting, including both scheduled and spontaneous sits. For nonretentive encopretic children, days of no soiling may also be rewarded, but it is important not to reward retentive children for "no soiling"

days *alone,* as this may only exacerbate retention. Social praise is usually supplemented by applying stars or other marks of success to the calendar record. Except for symbolic rewards, such as stickers or stars for their record-keeping charts, we do not typically use material rewards in our program. However, tangible rewards and sometimes punishments are used in some other behavior management plans (Wright & Walker, 1978).

A day of soiling should conclude with a warm bath in the evening, because soiling often signals incomplete evacuation of feces. The bath should be followed promptly by a toilet sit, which will often result in a successful bowel movement, especially for younger children.

Cleanliness Training

Cleanliness training for encopresis parallels that in our enuresis program. From the time treatment begins, children are expected to be responsible for taking off soiled undergarments and linens and putting them in an appropriate place, as well as cleaning and changing themselves.

Providing Appropriate Expectancies

Unlike urine-alarm-based management plans for enuresis, we have frequently noted a dramatic initial reduction in soiling behavior in the early weeks after the behavior training session for encopresis. This is often not maintained over the longer term, however, without continued use of the plan's several components. For this reason, families are continually reminded that achieving full bowel control is a gradual process and that ups and downs are to be expected along the way.

Two General Treatment Considerations

Monitoring for Compliance

In treating any of the elimination disorders, the most important role for the therapist is to assure that the parent faithfully maintains *all* aspects of the plan until treatment has been successfully completed.

Follow-Up

We try to follow up children in our clinics by phone every 3 months for at least a year after initial arrest of the disorder. Relapses are given prompt attention through retraining (which is almost invariably successful if treatment has been successful initially).

A Final Word about Treatment

The most encouraging fact about the functional elimination disorders is that successful treatments exist for the most common forms, especially those that fit a learning deficit model. On the other hand, based on our clinical experience, we have little doubt that in certain cases, notably those involving intermittent secondary enuresis or encopresis which persists in the face of adequate diet and structured behavioral guidance, a psychodynamic formulation may be more useful. Unfortunately, these latter cases tend to be unresponsive either to verbal psychotherapy or to behavioral techniques (DeLeon & Mandell, 1966; Werry & Cohrssen, 1965).

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CHAPTER 33

Physical Disorders

Gabor Barabas

Tourette Syndrome and Tics in Childhood

Introduction

Tourette syndrome is a neurologic syndrome that during childhood presents with tics. Generally, tics are involuntary actions that are felt to be secondary to dysfunction of the basal ganglia. The basal ganglia are deep regions of the brain, and various lesions of this area result in involuntary movements. During sleep, however, tics tend to disappear.

Gilles de la Tourette first described the syndrome in 1885 (de la Tourette, 1885). Tourette syndrome is recognized as an organic disorder that is related to abnormalities in the metabolism of neurotransmitters in the basal ganglia. At one time, it was viewed as a psychiatric disorder. Symptoms can be almost imperceptible or extremely severe, often resulting in social ostracism and debilitation. Studies have not demonstrated any relationship of tics to perinatal insults or trauma to the nervous system. In approximately one third of patients, there is a positive family history of Tourette syndrome or other tic disorders, and the condition affects all racial groups, with a male-to-female sex ratio of 3 to 1. The Tourette Syndrome Association estimates that there are approximately 100,000 individuals in the United States affected by the condition.

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Diagnosis

The diagnosis of Tourette syndrome is made clinically, and there are no existing laboratory tests to confirm the diagnosis. Several clinical criteria have to be fulfilled in order to make the diagnosis of the syndrome reliable (see Table 1). Multiple motor and vocal tics appear between the ages of 2 and 15, and the symptoms wax and wane in intensity and change in nature over time. However, usually it is required that the symptoms be present for at least one year. In adhering to the criterion that symptoms be present for at least one year, habit spasms, which result from transient psychological stresses, are excluded.

There are children who are affected by chronic multiple tic disorders, in whom vocal tics do not occur. Such individuals are differentiated from children with Tourette syndrome. However, it is quite likely that the biochemical disturbance in this condition is similar to that found in Tourette syndrome. This finding is supported by the fact that in those family members in whom there is Tourette syndrome, frequently there are some members who not only have the syndrome but also have chronic multiple motor tic disorders.

Symptoms

Most frequently, motor tics involve the face, and there is blinking of the eyes, facial grimacing, movements of the mouth, jerking of the head and shoulders, and various movements of the tongue.

Table 1. Clinical Features of Tourette Syndrome

1. Multiple motor and vocal tics
2. Onset of tics occurs between the ages of 2 and 15
3. Tics wax and wane in intensity
4. Tics change in nature over time
5. Symptoms are present for at least one year

Because tics can also involve the trunk and extremities, there may be tensing of muscles as well as posturing and jerking of the limbs. Vocal tics take the form of various rudimentary sounds, such as sniffing, clearing of the throat, coughing, hissing, barking, snorting, shrieking, honking, squeaking, and burping. Also, there may be repetition of letters, words, or phrases. Some children have explosive outbursts of vocalization, and others, *echolalia. Coprolalia,* or involuntary cursing, is a classic symptom that occurs in approximately 25% to 35% of children, and generally has its onset during adolescence. Frequently, coprolalia involves the repetition of four-letter words, and the children may attempt to cover up such cursing by forming various words and by making other noises. Approximately 10% to 15% of children with Tourette syndrome exhibit what are termed *complex tics,* which may include kicking, squatting, jumping, dancing, or repetitive touching and kissing of objects and people. Essentially, these complex actions are obsessive-compulsive symptoms, and, on occasion, there can also be self-injurious behavior with biting, scratching, or head banging. There may be *copropraxia* (obscene gestures) or the touching of the genitals. Some children may have relatively mild-to-moderate motor and vocal tics but can still be debilitated by what are termed *mental tics.* Because they are silent, these tic symptoms frequently go undiagnosed but typically involve the repetition of numbers, words, and phrases that interfere with cognitive processes. Thus, it can take a child with a mental tic several hours to complete a 15-minute assignment, resulting in significant problems either in school or in various other social situations.

When viewing the multitude of symptoms that can occur in Tourette syndrome, especially the more bizzare symptoms, it is not surprising that this condition has been regarded as being of psychiatric origin. The point at which the obsessive-compulsive symptoms and the mental tics are psychiatric phenomena, versus organic phenomena, obviously cannot be delineated. A child living with this condition, especially when it is misdiagnosed or undiagnosed, will probably also have serious secondary psychological problems complicating the situation even further.

Tourette syndrome often goes undiagnosed for several years, and in many instances, the diagnosis is made by the family when they encounter articles in the newspaper or on television. Repetitive blinking in a child may result in a consultation with an ophthalmologist, whereas excessive clearing of the throat with sniffing and coughing may lead to a consultation with an allergist. Sometimes children are seen by psychiatrists or psychologists and receive behavioral therapy and other modalities that may have no positive effects on the tic symptoms.

Associated Symptoms

Tourette syndrome is associated with attention deficit disorder or learning disabilities in approximately 50% of children (Nee, Caine, Polinsky, Eldridge, & Ebert, 1980). All too often, there is also a discrepancy between verbal and performance IQs in these children, contributing further to the difficulties of living with the syndrome. It is important to note that in children with Tourette syndrome who are hyperactive, stimulants are generally contraindicated because they have the potential for aggravating the tics (Golden, 1974). It appears that there is an increased incidence of migraine in children with Tourette syndrome (Barabas, Matthews, & Ferrari, 1984b). Other studies indicate that various disorders of arousal such as somnambulism and night terrors, (Barabas, Matthews, & Ferrari, 1984c; Glaze, Frost, & Jankovic, 1983), as well as motion sickness (Barabas, Matthews, & Ferrari, 1984a), are also increased. It is likely that the biochemical disturbance that predisposes individuals to tics also predisposes them to these associated symptoms.

Biochemistry

In the past 25 years, a neurotransmitter disturbance in Tourette syndrome and in various tic disorders has been recognized. Dopamine appears to be most affected. Haloperidol, a dopamine receptor blocking agent, has been found to be beneficial in approximately 80% of affected children (Shapiro & Shapiro, 1981). Because this drug is efficacious, it has been hypothesized that Tourette syndrome is

associated with hyperactivity of dopaminergic systems in the basal ganglia. However, haloperidol may cause various side effects that are intolerable so that, after one year of therapy, only approximately 40% of treated individuals still derive beneficial effects from the drug. Also, the drug may cause changes in mood and concentration in the patient and result in lethargy and weight gain. Another agent that is utilized in the armamentarium for Tourette syndrome is clonidine. This drug is a noradrenergic blocking agent that is helpful in approximately 40% of patients (Cohen, Detlor, Young, & Shaywitz, 1980). The positive effects of this drug indicate that in some individuals there is a hyperactivity of noradrenergic systems in the brain. Although clonidine may result in lethargy, it does not seem to have the side effects that haloperidol does. Other drugs that have potent effects on neurotransmitters include pimozide and clonazepam. Although many children derive significant benefits from the drugs mentioned above, approximately 15% to 20% do not respond to any medication. In other individuals, initial positive effects may be supplanted by a recurrence of symptoms. As noted, there can also be an intolerance to side effects, making drug therapy impossible over a long period of time.

The long-term efficacy of these drugs is often difficult to assess because of the waxing and waning of tics over time in the untreated state. Supportive psychological and behavioral therapies are often necessary to help children and families adjust to the pressures of living with Tourette syndrome. Teachers may discipline children because of their vocal tics if they are not aware of the involuntary nature of these symptoms. And the children themselves are ridiculed and ostracized by their peers.

The decision to treat a child who has Tourette syndrome depends not so much on the severity of the tics but rather upon the degree of social disability. Although it is a general rule that the more severe the tics the greater the social disability, this is not always the pattern. Some children are extremely effective in accommodating the symptoms and in being able to maintain social activities. Other children can be debilitated by relatively mild symptoms so that enhancement of coping skills can be quite helpful. Some children are able to suppress their tics for several hours while at school or at social gatherings, and then let the symptoms out at home. Before treating with drugs, it is always important to consider whether one is treating the child or the family.

Prognosis

In approximately 10% to 15% of children, the tics eventually will disappear with time, usually in late adolescence or in early adulthood. In 40% of individuals, the tics continue to decrease into middle age and do not present a significant problem and in the remaining 40%, the symptoms present a significant problem throughout life. In the initial clinical descriptions of Tourette syndrome, it was felt that there was a neurologic and intellectual deterioration with time. However, prolonged observations have demonstrated that there is no such deterioration. The few pathologic specimens of brain that have been examined from individuals who have had Tourette syndrome have not demonstrated any structural or microscopic abnormalities in the brain or in the basal ganglia.

Differential Diagnosis

Tics have to be differentiated from other neurologic conditions that result in involuntary movements. These are complex and generally rare conditions most of which are associated with various degenerative processes. Therefore, it is helpful to refer individuals with involuntary movement disorders to a neurologist to rule out the above entities. Tourette syndrome also has to be differentiated from habit spasms that are of psychogenic origin. Generally, in this situation, the symptoms do not persist beyond 3 to 6 months, usually manifest as blinking and grimacing, and tend not to evolve into other motor tics. Vocal tics are infrequently seen. "Transient tics" of childhood and chronic motor tic disorder also have to be differentiated from Tourette syndrome. However, as has been alluded to, they may all represent merely different points on the biochemical continuum leading to Tourette syndrome.

Conclusion

Tourette syndrome is a complex disorder and its etiology is not well understood. There appears to be a genetic component in a significant number of affected individuals. The condition can range from being a very mild disorder to being a totally debilitating one. The social and psychological pressures may be intense and may require therapy. Education of parents, instructors, and employers is often necessary. Although effective medications are available, the frequent side effects and the

breakthrough of symptoms will make therapy less than optimal. It is apparent that the further elucidation of the biochemical basis of Tourette syndrome and tic disorders will shed great light on the complex mechanisms that are involved in various neurologic and psychiatric disorders.

Sleep Disturbances and Disorders of Arousal

Introduction

Abnormalities in children's sleep behavior and sleep pattern represent a relatively frequent chief complaint. Parents often complain of their children's having difficulties falling asleep or of restlessness during sleep with frequent early wakening. Also, nightmares constitute a common symptom. Generally, such sleep disturbances are transient and are related to stages in children's development and to certain environmental and psychological factors. For instance, it is not unusual for a young child to develop sleep disturbances upon moving to a new home, starting school, or after the loss of a family member or a close pet. On occasion, sleep disturbances may be symptomatic of more serious psychological conflicts or of problems within the family unit. A recent study (Simonds & Parrage, 1982) found that 27.6% of children were restless during sleep, 1.7% had frequent nightmares, 3.6% were fearful of going to sleep, 7.5% engaged in bedtime rituals, 5.3% insisted on sleeping with others, and 10.7% required a security object. The frequency of these symptoms usually will decrease with age.

As mentioned, these disturbances are typically viewed as "behavioral" phenomena and are categorized as *sleep disturbances* or abnormal sleep behavior. In contradistinction, there is another group of sleep abnormalities that is considered to be independent of environmental and psychological factors and are categorized as *disorders of arousal.* These symptoms tend to occur during specific stages in sleep and are more often thought of as being "organic" and associated with instrinsic biochemical disturbances. The primary factor differentiating disorders of arousal from sleep disturbances is that there is amnesia for the event upon awakening.

Physiology of Sleep

The sleep–wake cycle represents an endogenous circadian rhythm of the body. It is not understood why sleep is necessary. Until relatively recently, sleep was viewed as a passive phenomenon during which there was an absence of consciousness and mental function. The active character of sleep was recognized when it was observed that there are specific sleep stages that are manifested in a predictable order during each sleep cycle. These stages are independent of the environment and are dependent upon complex and specific neural and biochemical mechanisms.

Slow-Wave or Non-Rapid-Eye-Movement (NREM) Sleep

During the initial phases of sleep, the electroencephalogram (EEG) shows a progressive slowing of brain-wave frequency and an increase in voltage. This progressive slowing correlates with the depth of sleep. The stages are progressively numbered 1 to 4, and each stage is identifiable on the EEG by certain characteristics. In the initial cycle of sleep, it takes approximately 45 minutes to progress successively from Stage 1 to Stage 4 slow-wave sleep. Once Stage 4 sleep is achieved, there is gradual arousal through lighter states back to Stage 1 over 30 to 45 minutes. These transitions keep recurring throughout the night. Slow-wave sleep is also called non-REM (NREM) or non-rapid-eye-movement sleep, in contrast to rapid-eye-movement (REM) sleep. In REM sleep, rapid eye movements are characteristic. In addition to lack of rapid eye movements, slow-wave sleep is also characterized by relaxed muscles, muscle movements (change in posture), decreased heart rate and blood pressure, and an increase in gastrointestinal motility.

Rapid-Eye-Movement (REM) Sleep

After approximately 90 minutes of slow-wave sleep, REM sleep appears. This stage is distinguished from Stage 1 sleep by rapid eye movements. In addition, there is an increase in heart rate and blood pressure, and a decrease in gastrointestinal motility. There is loss of muscle tone and lack of truncal muscle movements (see Table 2). Upon forced awakening, dream recall is most characteristic of REM sleep whereas it is rather infrequent upon awakening from non-REM sleep.

During a normal night of sleep, there is a cyclical transition between NREM and REM sleep. The ratio of NREM to REM sleep changes with age. A normal infant sleeps about 16 hours per day with approximately 50% of that time spent

Table 2. Characteristics of Sleep Stages

	EEG	Heart rate	Blood pressure	Gastrointestinal motility	Skeletal muscle movements	Eye movements
Slow-wave of non-rapid-eye-movement (NREM) sleep	Progressive slowing, high voltage	↓	↓	↑	+	−
Rapid-eye-movement (REM) sleep	Fast activity, low voltage	↑	↑	↓	−	+

in REM sleep. By the time the child reaches adolescence, total sleep requirement decreases to 8 hours per day, of which only 25% is spent in REM sleep. Stage 4 NREM sleep also tends to decrease with age.

Biochemistry

The lower region of the brain stem reticular system, a phylogenetically primitive area of the brain, is the center involved in the active induction of sleep. This region also regulates and maintains the smooth transition through the various stages of NREM sleep. The primary neurotransmitter involved in such regulation is serotonin. Disturbances in serotonin activity can lead to erratic transition through the stages of slow-wave sleep and can lead to *disorders of arousal.*

Disorders of Arousal

Disorders of arousal are distinguished from sleep disturbances by the fact that there is amnesia for the event upon awakening. These disorders characteristically occur in the transition from Stage 3 to Stage 4 slow-wave sleep to lighter stages of NREM sleep. It has been hypothesized that disorders of arousal are primarily associated with disturbances in serotonin metabolism.

Somnambulism. Sleepwalking may affect males more frequently than females. Recurrent sleepwalking has been estimated to have a prevalence of 1% to 6% (Kales & Kales, 1974). There is an association with enuresis (Pierce & Lipcon, 1956), and it appears to manifest in certain families (Andre-Balisauk & Consetto, 1956). Although it has been traditionally assumed that somnambulists are dreaming, sleep studies have demonstrated that sleepwalking occurs in Stages 3 to 4 slow-wave sleep when dreaming is least likely to occur (Broughton, 1968; Jacobson, Kales, Lehman, & Zweizig, 1965). The episodes tend to last several minutes. Affected children are unaware of their surroundings and are unarousable. As with all disorders of arousal, the individuals are amnesic for the event.

In questioning parents regarding sleepwalking, it is important to make certain that they understand what constitutes the symptom. Some parents feel that their children "walk in their sleep" if they get up at night to go to the bathroom or come to the parents' bed in the middle of the night seeking comfort. All too often, the sleepwalking event goes unwitnessed, and the child is found lying on the floor in another room or doorway.

In most children, sleepwalking disappears with age correlating with the decrease in Stage 4 sleep; however, it can persist into adulthood. Studies have not demonstrated any characteristic psychological disturbances in children who sleepwalk (Kales, Paulson, & Jacobson, 1966). This is in contradistinction to adult sleepwalkers in whom psychiatric disorders are prevalent. In rare individuals, sleepwalking is an epileptic phenomenon. These symptoms are called epileptic *nocturnal wanderings* and can be identified on sleep EEG tracings and may be responsive to anticonvulsant therapy. The management of sleepwalking can pose problems to the therapist and to the family. On rare occasions, it can present a physical threat to a child so that all windows and doors have to be secured. Some children may climb out of a window or wander into the street and are at risk of serious physical harm. In such situations, relocating the child's bedroom to the ground floor when possible is advisable. In children with frequent somnambulism, the administration of Valium before sleep can dramatically suppress symptoms.

Night Terrors (Pavor Nocturnus). Night terrors constitute a rarely encountered disorder of arousal. Simonds found that 9.7% of sleepwalkers also experienced night terrors (Simonds & Parrage,

1982), usually occurring during the first half-hour of sleep. The affected child may sit up in bed and appear to be terrified. There may be screaming or moaning, and the child is inconsolable and unarousable. Excessive perspiration and rapid respirations may be noted. Episodes last several minutes. The event ends abruptly and is followed by sleep and, in the morning, amnesia. Like somnambulism, night terrors occur during sudden arousal from Stage 4 sleep and must be distinguished from nightmares. Nightmares occur during REM sleep and are frequently recollected to varying degrees upon awakening. Generally, an individual who is having a nightmare can be aroused.

Night terrors, like sleepwalking, may respond dramatically to Valium prescription by the suppression of Stage 4 sleep (Fischer, Kahn, Edwards, & Davis, 1973). Families need to be reassured of the benign nature of this symptom because to be witness to it can be a frightening experience. Furthermore, parents need to be reassured that night terrors are usually not associated with psychological problems and that, except for rare exceptions, children invariably outgrow the tendency. In contradistinction, persistent nightmares in a child warrent further investigation into family dynamics and potential psychopathology.

In general, pharmacotherapy for sleepwalking or night terrors should be reserved only for severe cases in which the potential for bodily harm exists or in which significant emotional pressures are noted to warrant such intervention.

Nocturnal Head Banging and Sleep Rocking. Nocturnal head banging and rocking in sleep are very rare disorders. They may be related to disturbances in the deep stages of slow-wave sleep, and are therefore disorders of arousal. Very little research exists on these unusual symptoms.

Like night terrors, these disorders tend to affect children younger than those who engage in sleepwalking. Usually, therapy does not appear to be necessary. Parents need to be reassured regarding the head banging. It must be kept in mind that some retarded children engage in stereotypic rocking and head banging. On rare occasions infants and toddlers who are suffering from headaches may bang their heads. However, this tends not be to limited to sleep alone, and it is obvious that such a diagnosis is an extremely difficult one to entertain and almost impossible to substantiate. Of assistance is the fact that head banging from pain is associated with overt evidence of pain, whereas nocturnal head banging is not accompanied by any apparent discomfort.

Sleep Talking. This disorder appears to be the most frequently encountered disorder of arousal, affecting 12.7% of children (Simonds & Parrage, 1982). Because of its benign nature, it does not pose a management problem and is viewed by most families with amusement. Sleep talking has been found to occur during Stage 4 sleep (Anders & Weinstein, 1972).

Bruxism. Nocturnal teeth grinding can be a bothersome symptom and has occasionally been linked to temporo-mandibular joint disease and resultant facial pain. This association, however, has hardly been established. In some children, a dental appliance may be necessary to prevent the teeth-grinding activity. Bruxism, which occurs in 7.5% of children (Simonds & Parrage, 1982), is noted primarily in Stage 2 sleep (Karacan, Salis, & Williams, 1973). Not much research exists on this symptom.

Associated Conditions

Recent clinical research has demonstrated that in certain clinical situations there is an increased incidence of disorders of arousal. In children with migraine syndrome, there is an increased frequency of somnambulism when compared to control populations (Barabas, Matthews, & Ferrari, 1983a). In children with Tourette syndrome, there is an increased frequency of migraine (Barabas *et al.*, 1984b), and in some an unusual increase in somnambulism and night terrors (Barabas *et al.*, 1984c). The basis for this unusual association of disparate symptoms has not been elucidated. However, it has been suggested that it reflects derangements in neurotransmitter metabolism, in particular, those affecting serotonin.

Conclusion

Sleep disturbances and disorders of arousal represent frequently encountered symptoms in childhood. In general, the symptoms do not require active intervention and, except for enuresis, families and children rarely view the symptoms as problems. Persistent enuresis, frequent somnambulism, and, rarely, night-terrors may require a trial of drug therapy. Recurrent sleep disturbances may warrant a psychological evaluation. On occasion, an EEG obtained during sleep is indicated to identify unusual nocturnal epileptic phenomena. Significant research in this area is still needed because such studies will help clarify not only the nature of sleep

abnormalities but also the complexities of neurotransmitter systems in normal and pathologic states.

Enuresis

Enuresis is defined as involuntary urination during sleep. There has been significant controversy regarding the etiology of enuresis. In some cases, it appears to be a disorder of arousal. The association between somnambulism and enuresis gives credence to this possibility. In one study, 22.6% of sleepwalkers experienced enuresis (Gastaut & Broughton, 1964). In general, the term should be utilized only when involuntary wetting extends beyond the age of 6. Enuresis is more often found in boys than in girls (Cohen, 1975). Daytime or diurnal urinary control is usually achieved first, between the ages of 3 and 4. Nighttime or nocturnal control is achieved later, by the age of 6. There are individuals who continue to have nocturnal enuresis into early adulthood.

It appears that a certain degree of central nervous system maturation is necessary for voluntary bladder control to be present. In infancy and early childhood, reflex mechanisms dominate that sense a full bladder and that result in involuntary bladder contractions and emptying. These mechanisms are modified as certain brain and spinal cord centers mature and begin to exert their influence in inhibiting reflex responses. Once these neurologic pathways have developed, learning is still necessary for achievement of total control, as is a favorable environment and parent–child interaction.

Children with primary enuresis are those who have never achieved bladder control, whereas those with secondary enuresis have had partial or total control for a period of time, only to regress into incontinence. Some investigators have found that up to 25% of children may have secondary enuresis at some time in their life (Oppel, Harper, & Ryder, 1968).

Nocturnal enuresis is much more common than daytime wetting, which occurs in only 5% of enuretics.

Enuresis not only tends to be more common among lower educational and socioeconomic groups (Blomfield, 1956), but there also appears to be a genetic component to the disorder. In children with enuresis, wetting is found in another family member at least 70% of the time (Hallgren, 1958). In one study, it was noted that in monozygotic (identical) twins, there was concordance for enuresis more frequently than in dizygotic (nonidentical) twins (Bakwin, 1973). There is a widely held belief that enuresis is due to psychological factors especially related to abnormalities in child–parent relationships (Gerard, 1939). There are very few studies, however, demonstrating a significant psychological basis to the problem. Enuresis has also been linked to passivity, aggression, environmental stress, feelings of abandonment, and an immaturity of personality. Abnormalities in training methods can be associated with enuresis; for example, ignorance of appropriate training methods, infantilization, the failure of parents to indicate that enuresis is unacceptable, and excessive punitive training can result in incontinence (Bregar, 1963). Brazelton (1962) found that in a training approach in which neurologic maturation was present and in which parental attitudes were appropriately fostered, urinary control was achieved in 98.6% of children by the age of 5.

Physical Causes

Abnormalities of the urinary tract, resulting in obstruction of urine outflow, can result in enuresis. Urinary tract infections can also result in enuresis and tend to affect girls more than boys. Of greater significance is the fact that certain spinal cord lesions affecting bowel and bladder function can lead to wetting. Spina bifida or developmental abnormalities of the spinal cord are a potential cause. In addition, certain tumors acquired at a later age can result in secondary enuresis. Diabetes mellitus and diabetes insipidus (a hormonal disorder affecting the pituitary and hypothalamic regions of the brain) can also lead to frequent urination.

Under what circumstances should the physician become concerned that enuresis relates to organic or physical abnormalities as opposed to a developmental delay in the maturation of urinary control? When there is associated encopresis (fecal soiling), a progressive gait disturbance, weakness in the lower extremities, or an inability to sense and appreciate bladder distention or bowel movements, the physician needs to be concerned, because these signs are especially suggestive of a spinal cord lesion. In addition, sensory deficits in the lower extremities may also be found. In such situations, a referral to a pediatrician, urologist, or neurologist is appropriate. The earlier that intervention is sought, the better the prognosis. In such children, there is usually both nocturnal and diurnal enuresis because the control of urination is affected irrespective of the time of day. Children with urinary tract infections tend to have a burning sensation when urinating and will often have frequent urination and feelings of urgency. Those with diabetes mellitus and

diabetes insipidus may have prominent thirst as a frequent concomitant, because these children are losing fluid to a great degree. When epilepsy, in the form of unappreciated nocturnal seizures, results in urinary incontinence, there may be tongue biting and other signs of a convulsion. This possibility does need to be considered in some children, and an EEG may be diagnostic. Spinal cord lesions are diagnosed by the use of a myelogram or noninvasive magnetic resonance imaging (MRI) of the spinal cord. Diabetes mellitus and diabetes insipidus are diagnosed by analyzing blood for glucose, electrolytes, and various hormonal components. When a specific urologic obstruction or abnormality of the urinary tract system itself is suspected, studies of the kidney and bladder are indicated.

Psychological Factors

When exploring potential psychological elements, it is important to consider whether enuresis is more prominent during periods of stress. The physician needs to explore whether there are conflicts in the home of the patient and what management techniques are being utilized. It is important to ask parents at what age was training started since conflicts over premature training can predispose to enuresis. If punitive methods are being utilized, this, too, needs to be discussed. The degree of resistance on the part of the child is important to note. If there has been an inconsistency in training, resulting in confusion on the part of the child, it has to be noted.

Treatment

The simplest form of treatment of enuresis is to attempt to restrict fluids after dinner. Giving the child this responsibility is especially helpful; so, too, is taking the child to the bathroom to urinate prior to the parents' going to bed. Both modalities may contribute to substantially maintaining dryness. Certain drugs that increase bladder capacity have been utilized in treating enuresis. Imipramine is the most frequently used agent and is given one hour prior to bedtime. However, this drug should not be used for prolonged periods of time.

In the past decade, various conditioning devices have been utilized to treat enuresis. Instruments sensitive to moisture that are connected to an alarm awaken the child upon initiation of wetting. There is controversy as to what degree this is efficacious over time, because relapses are encountered relatively frequently.

When dealing with a child with enuresis, it is important to approach the difficulty with optimism since most cases of enuresis eventually resolve. Enuresis can be quite refractory to all therapeutic modalities, and only time and maturation of physiologic mechanisms and nervous system pathways will result in improvement.

Encopresis

Encopresis is defined as persistent fecal soiling or incontinence after the age of 4 years. Generally, it is viewed as a more serious condition than enuresis because it often results in significant social ostracism. Fortunately, enuresis resolves in most cases spontaneously, but encopresis may be a much more difficult and persistent problem. Although psychological causes are rarely implicated as being the primary cause of enuresis, such factors are often involved in encopresis. For example, there may be a significant disturbance in the child–parent interaction, and problems related to encopresis may become especially apparent once a child begins to attend school and peer ridicule is manifested. Encopresis occurs more frequently in boys than in girls.

Some affected children may also have attention deficit disorder or other learning problems. For example, toilet training for bowel continence requires that a child learn how to defecate, as well as how to prevent defecation under certain circumstances. Maturation of neurologic pathways involved in defecation is a prerequisite for success, and the role of the parent is to enhance the learning process. Early on, infants and young children recognize that there is a negative aspect to defecation by observing changes in parental facial expression and hearing negative statements. Thus, positive statements related to potty-training ultimately will result in the child's relating approval to defecating only under appropriate circumstances. Interestingly, the most frequent physical basis for encopresis is chronic constipation which leads to obstruction of the lower bowel by fecal matter with passage of fecal fluid around the obstruction resulting in fecal soiling. In children with chronic constipation, bowel movements may be painful, and anal fissures and hemorrhoids may develop. Once the child begins to associate bowel movements with pain, there may be further attempts at retaining fecal matter, thus complicating the constipation even further, and a vicious cycle eventually develops. As the lower parts of the bowel are stretched by the chronic

constipation, the ability of the child to sense fullness also begins to decrease. Rectal examination by a physician will demonstrate impacted fecal matter. In severe cases, suppositories and enemas are necessary to "retrain" the bowel. In some children, fecal soiling is related to specific neurologic lesions, especially those of the spinal cord. As noted with enuresis, there are frequently other associated neurologic symptoms, such as gait disturbances and disturbances in sensation of the lower extremities. Such children require a myelogram or magnetic resonance imaging studies of the spinal cord to demonstrate the lesion. Since the innervation of the bladder is also affected, such children tend to have enuresis as well.

Another condition leading to encopresis is Hirschsprung's disease. This condition is usually diagnosed by a barium enema. Children who are born with this condition inevitably will have constipation and chronic fecal soiling, because there is a lack of development of nerve cells in the lining of the bowel and, therefore, the bowel does not contract properly. On occasion, this condition can lead to life-threatening complications. Certain metabolic disorders, such as hypothyroidism (absence or decrease in thyroid hormone), and abnormalities in calcium metabolism can also result in chronic constipation with encopresis. Also certain drugs containing codeine can lead to fecal soiling. Abnormal narrowing of the rectum or anus can result in chronic constipation and overflow fecal soiling.

As has been noted, a psychological basis for encopresis is more frequently encountered than with enuresis. Mother–child conflicts surrounding bowel control can result in phobias surrounding the toilet (McTaggart & Scott, 1959). Environmental and other psychosocial stresses related to bowel training can also be contributing factors to encopresis. Children who are impulsive and distractible are more prone to have this problem, and any evidence of child abuse, marital strife, and abnormalities in parenting have to be explored. Encopresis can become a source of major family conflict, therefore, it is important to treat the disorder as early and effectively as possible. When no specific physical abnormality has been delineated, a consistent training method must be established. The child has to be encouraged to sit on the toilet at least twice a day at the same time whether he or she feels the urge to defecate or not. The child should sit for approximately 10 to 15 minutes. The use of various charts or even small gifts may help to reinforce this training. Also it should be explained to the child that the need to sit on the toilet is related to building up the muscles. Lastly, introducing foods with fiber into the diet may be helpful.

When fecal soiling is related to a neurotic disorder or to a more severe psychopathology, generally there are other symptoms, and encopresis is not an isolated facet of the problem.

In summary, it is important to intervene early in cases of encopresis. Because most children respond to conservative medical approaches and psychological therapy, it is important initially to rule out organic causes. Prognosis is generally favorable in most cases, and, in all instances, it is important to approach therapy in a family setting.

Stuttering

Stuttering is a speech disorder that develops between the ages of 2 and 5. It is characterized by a difficulty in the smooth flow of speech, with typically a repetition of sounds and syllables and an impairment in progressing from phoneme to phoneme. In most children, stuttering is a transient phenomenon but in some it may become a chronic problem. It occurs in approximately 4% of the population. By late childhood, most children have spontaneously recovered from stuttering. The prevalence of stuttering, at any given time in the general population, is approximately 1% (Guitar, 1985). Studies suggest that stuttering may have a hereditary component (Kidd & Records, 1979). Studies of monozygotic twins, for example, have demonstrated a concordance to stuttering (Howie, 1981). The etiology of stuttering in unknown. Although it has been attributed to various psychological factors, the evolution of stuttering as a manifestation of serious emotional problems is extremely uncommon. Even when significant emotional problems and conflicts are delineated, it does not mean that they relate directly to stuttering. Certain environmental factors, increased self-consciousness, and stress can aggravate stuttering. It is of interest that ambidexterity has been documented in some studies of stuttering as being associated (Bryngelson, 1935). This finding has led to the hypothesis that abnormalities in the establishment of cerebral dominance are related to stuttering in some individuals; however, this hypothesis has not been demonstrated in well-controlled studies.

Although the etiology of stuttering is elusive, certain theories have focused upon a developmental problem in which the affected child has difficulty in learning the temporal patterns required for smooth speech, thus implying that there may be a develop-

mental problem in the functioning of regions of the brain that are responsible for processing language and oromotor movements. In adults, bilateral cerebral hemispheric injury, in association with aphasia, can lead to language disorders with stuttering (Canter, 1971). In children with certain rare degenerative diseases, such as juvenile lipidosis, similar disfluencies have been documented (Schain & Wiley, 1965).

Children who stutter need to be referred to a speech and language pathologist if the problem persists for longer than 6 months. If there is excessive parental concern and stress related to the problem, psychological counseling may be necessary. In many children, it is observed that certain disfluencies and stuttering result when the child is attempting to talk rapidly to get a parent's attention. An understanding attitude, and establishment of a relaxed atmosphere whenever ideas are being communicated, will help to alleviate this problem and facilitate recovery.

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CHAPTER 34

Sexual Development and Sexual Psychopathology

An Object Relations Point of View

David E. Scharff

The lifelong phenomena of sexuality have been at the center of theories of personality development and psychopathology since Freud's early psychoanalytic researches (Breuer & Freud, 1895; Freud, 1905). His revolutionary discovery of the world of infantile sexuality through reconstructions from adult analyses were later complemented by the psychoanalysis of young children (A. Freud, 1959; Klein, 1932). More recently, direct observations of infants and young children have clarified the dynamic aspects of psychosexual development and added to our understanding of a specifically sexual developmental line (Roiphe & Galenson, 1981; Scharff, 1982). Crucial aspects of adult sexual functioning were unknown until Masters & Johnson's research revolutionized our knowledge of sexual physiology, highlighted by the publication of *Human Sexual Response* in 1966. With the publication of their second book, *Human Sexual Inadequacy* in 1970, and the work of Kaplan (1974) and others, adult sexual dysfunction has been described and treatment approaches have been developed. The description of normal and abnormal adult sexuality has allowed us to begin investigations of the contributions of development to childhood and adult sexual disorders.

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This chapter will explore sexual development from an object relations point of view and relate it to sexual symptomatology at various stages of development, to aspects of strain in sexual functioning, and to common sexual problems in everyday life. Such an attempt, although based centrally on psychoanalytic object relations theory, draws also on child development, sexual physiology, sex therapy, family therapy, and group analysis. It is necessary to range widely in gathering viewpoints that will help us explain these phenomena, because the complexity of human personality and interaction ensures that no single theory can explain experience fully.

Object Relations Theory

Fairbairn (1952, 1954) proposed that what motivates the development of infants is not the unfolding of inherent drives of sex and aggression as Freud (1905) had proposed, but the need for a relationship with the mother (or mothering person), who, in Freud's theory, had been seen as merely the "object" of the sexual and aggressive drives. Within this relationship, the infant's psychological structure is built up by internalizing the experience with the mother. The experience, which is mainly satisfying, contributes mostly to more conscious

and rational ego structure; whereas those aspects that are painful and unsatisfying are split off and repressed precisely because they are too painful to be borne in consciousness. When experience is internalized, it is taken in as two basic structures: an image of the mother or other primary person (the internal object) and an image of the self or ego in relationship to that object. In addition, this paired image carries the affect that characterizes that part of the relationship. Two categories of aspects of relationship to the mother are too painful to be borne consciously: the mother which the infant feels is rejecting of its needs or is persecuting; and the mother who triggers a painful longing for unmet needs. Part of the infant is in relationship to all three aspects of maternal experience, the satisfying mother with whom the infant feels calm and safe, the rejecting or frustrating mother, and the mother who tantalizes and elicits the painful longing. These "internal object relationships" do not represent the reality of the exchanges with the mother, but the infant's internalized versions of them, modified by the lack of maturity of the child's understanding at each stage of development and by the child's fantasy life.

Klein (1975a,b; Segal, 1973), who was a pioneer in object relations theory, introduced the idea of introjective and projective identification. Introjective identification results "when the object is introjected into the ego which then identifies with some or all of its characteristics." Projective identification "is the result of the projection of parts of the self into an object. It may result in the object being perceived as having acquired the characteristics of the projected part of the self, but it can also result in the self becoming identified with the object of its projection" (Segal, 1973, pp. 126–127). Introjective and projective identification provide us with a language for unconscious communication, because most sexual difficulty should be understood as an attempt to communicate with an external object and thereby to impose an aspect of internal object relations onto relationships in the outer world.

All development takes place within the family context. Attempts to establish a relationship sexually and otherwise involve attempts to match external objects to existing internal object relations, both with the purpose of reestablishing what has been familiar or good and of repairing what has been painful. As the child grows in the family environment, aspects of multiple projection and introjection that occur in the family group become part of its permanent internal record. In later relationships, either with the same family members at a later time, or with new external objects, the history of previous relations, as carried forward by internal object relations, determines the way relationships are understood and modifies them.

Sexual Development

Sexual development occurs as the child attempts to establish and maintain an attachment to its mother and then to move into a stage of being separate but attached, which characterizes the rest of life (Bowlby, 1969, 1973, 1980; Mahler, Pine, & Bergman, 1975).

In the beginning, the mother and infant have a relationship that is at the same time totally physical and totally psychological. The mother (or mothering person) organizes the interior of the baby—the baby's psyche—through her physical handling of the baby, through attunement to the baby's rhythms, the feeding, the positioning of the body, looking into the baby's eyes, making noises which have a particularly maternal characteristic, and by attuning her own rhythms to the baby's. Winnicott (1971) called this relationship "the psychosomatic partnership." This partnership begins the history of the intertwining of physical and emotional development. It is the root of all development and it is the root of the involvement of the body in the organization of the psyche, as well as the involvement of the other person in the organization of the psyche. Out of this developmental trunk springs the branch of sexual development, which always maintains a fundamental relationship to all other issues of growth.

Thus, sexual development begins between the baby's mouth and the mother's breast, in the surface contact of their skin, as mother and baby negotiate the baby's sense of position, and in eye and voice communication. The baby takes the mother in through its mouth, skin, eyes, and ears, and all of these in the healthy, normally functioning baby contribute to an integrated relationship. Defects in any one of the senses must be overcome by compensation from other areas.

Freud (1905) postulated that sexual development was at first oral in the first year, with its incorporative and biting aspects, and later anal. Only in the third year does the child become specifically organized around its genitals. The parents focus on the genitals with great feeling at birth, because of their role as signifiers of gender. They may feel approval and love if satisfied with the gender of the child, or disappointment if not. Even if they are

initially disappointed, the genitals may have a role in helping them mourn the loss of the gender they wished for.

One mother was bitterly disappointed to have a third daughter because she had felt this was her last chance for a son, and the absence of a penis hit her in particular. But as her infant daughter won her over, this mother began to feel that her daughter's genitals were indeed lovable. The loss, mourning, and restitution focused on her daughter's vulva.

Genital activity is a feature in infancy. Usually, touching genitals begins during accidental exploration or in imitation of the mother's handling of the genitals. This is pleasurable and is repeated. The absence of genital play by the end of the first year is a sign of inadequate parenting. However, in the first 18 months, genital play does not have the characteristics of masturbation. But at 18 months or later, a more organized activity begins, which can be called masturbation (Kleeman, 1966, 1977; Spitz, 1962; Spitz & Wolf, 1949). However, even though some genital manipulation occurs in the first 12 to 18 months, the infant's sexual activity normally focuses primarily on other areas of the body. Freud held that the focus shifted from oral sexuality to anal zonal sexuality in the second year. Increased feeling and control of the anal and urethral zones and toilet training are regular features of the second and third years. The sexualization of these areas occurs to a greater extent in some cases than in others. Roiphe and Galenson (1981) described the early phase of "body-genital schematization" at the onset of the rapprochement crisis at about 18 months. Heightened sexual urgency is demonstrated by an increase in object-directed masturbation, at a time when self and object are still closely intertwined. The child is especially affected by recognition of anatomical differences during this period and often responds aggressively. This phase is crucial both in the development of gender identity and in the choice of sexual object (Roiphe & Galenson, 1981). When the child has difficulty in the relationship to the mother in this critical period, it remains vulnerable to anxiety around separation, body image, and sexual differences. These factors are crucial in the development of the sexual line: in gender identity, object choice, and the splitting of self and objects, which later lead to paraphilias.

It is not until late in the third year that the child's bodily focus shifts to the genitals, but it then does so dramatically, introducing the phallic-narcissistic phase (Edgcumbe & Burgner, 1975). In the first phase, boys and girls have concerns about the adequacy of their genitals and compare them to the opposite sex. They show off, brag mercilessly, and/or mourn a perceived inadequacy. For example, at 2¾ years, one girl went to nursery school for the first time. Suddenly she began to sit backwards on the toilet, in imitation of boys who stood up. She announced that boys had "wiggly bottoms" and she explored who in her family had what sort, saying about her father's wiggly bottom, "I wish I had one." Another three-year-old girl told her father "I have a penis inside my pee-pee. It is sleeping and when it wakes up, it will come out."

This is the phase in which boys and girls focus anxiety on what they perceive to be the threat of castration, the absence of the penis in girls. Boys feel threatened in seeing this, whereas girls feel a loss which must be mourned. The child focuses on the genital qualities of each parent and relates to each parent with a new consciousness about its own genitals.

A few months later, Oedipal development *per se* joins in with phallic development, and the child now relates to the parents in terms not only of their genital characteristics but also in terms of their being a pair. Thus, the child often tries to form a pair with the opposite sex parent and to dispose of or denigrate the same sex parent. But the child experiences conflict, also wanting a relationship with the same sex parent. In part, each child would therefore also like to dispose of the opposite sex parent—but this remains a minor wish except in those individuals who move toward homosexual object choice. During the Oedipal phase, previous history is rewritten in terms of sexualized triangles, because the child is now sexually energized. The child has now advanced cognitively to the point of understanding the importance of the parents' relationship, and to the use of sexually defining characteristics as the major attribute of relationships (Scharff & Scharff, 1987).

The urgency to resolve these conflicts and loyalties are relieved considerably during latency. The internal forces of sexualization recede and cognitive growth continues, allowing the child relative freedom. Latency is a time when a child tries to keep down his or her sexual life and contain it within bounds, while building peer relationships and developing new skills. But there are many breakthroughs that betray the active presence of sexual issues. For instance, girls' jump rope rhymes often express active sexual concerns, while channeling sexual themes into the bodily excitement of the exercise activity, a sublimated derivative of masturbation. The themes are often present in the rhymes themselves: Cinderella/dressed in yellow/went up-

stairs to kiss her fellow. /By mistake,/she kissed a snake,/how many doctors did it take?/One, two, three, four, etc. (Goldings, 1974). The latency child is far from asexual, even on the surface of his life (Sarnoff, 1976). Sexual concerns continually break through, turmoil is often not far away, especially in the children who make up the clinical population.

At the age of 10 or 11, the child becomes once again sexually energized. The first movements often constitute a regression away from sexual material and often toward anal or "bathroom" material with an overt disparagement of sexual issues (Blos, 1967). However, the child becomes more and more curious. During early adolescence, the child talks a great deal about sex, with the beginning of urgent concerns for the opposite sex. There is a great deal of variation of the time adolescents actually begin to have intense relationships with the opposite sex. The initial interest is expressed in a great deal of talk with the same sex peers about the opposite sex. Later in adolescence, perhaps normally in mid-adolescence, sexual relating moves to the center of experience. There is a great deal of cultural and historical variation in the period during which genital relating normally becomes central, but a *concern* with genital relating becomes of interest sometime between early and mid-adolescence. Adolescents use their whole bodies in the same way that young children use toys as the opportunity to play at being adult. Sexuality, beginning with masturbation, often carries the urgency of relating in adolescence—those adolescents who are most afraid of it often feeling that they are falling behind their more easily sexual peers.

The Causes of Sexual Symptomatology

Several issues in development can be identified as constituting strong predispositions to sexual symptomatology and sexual disorders, either in childhood or in later life. Whether the resulting difficulty is manifest sooner or later depends on many factors: the strength of the predisposition, the constitutional factors in the individual, and the family encouragement of such a direction in individual development.

1. At certain points in development, sexuality is at the center of the individual's developmental thrust. Such periods as the genital focus and the consolidation of gender identity occur at approximately 18 months, with phallic and Oedipal development between 33 and 48 months, and in early adolescence. At these points, disruptions are often translated into a variety of sexual symptoms.

2. At these points in development, general aspects of loss may be turned into sexual symptoms or may be accompanied by sexualization. For instance, a child whose mother is depressed in the first two years or who loses a primary caretaker at 24 months may compensate by increased masturbation, an autoerotic attempt to soothe when the physical soothing of the mother is absent.

3. If a parent has an internalized form of conflict about sexual issues, these will often be transmitted to the child through projective identification. In addition, conflictual aspects of the parent's internal object relations will operate in the relationship to the child. If these involve sexuality specifically, for instance, in fear about sex, which leads to a sense of danger in intimate relationships, this is usually conveyed to the child through the parent's anxiety during the periods of sexual emphasis during development. But issues not specifically involving sexual matters may be translated into sexual symptoms by the child: a boy may read his mother's fear of aggression as if it meant she were afraid of his turning into a man rather than about aggression specifically.

4. The child does not only internalize the relationship with each parent, but also the quality of the parents' relationship to each other; that is, each child has an internalized image of the parental couple. This is built out of what is visible of their relationship as it is modified by the child's fantasies, fears, and wishes for that relationship. Therefore, real and imagined vicissitudes of the parents' marriage and sexual relationship have a marked impact on the growing child.

Sexual dysfunction in the parental couple is one of these factors that strongly affects the child. Despite Oedipal ambivalence, each child wants to have two parents who have a loving, resilient marriage with a good sexual life. Children in single-parent families will fantasize having the other parent and such a relationship. When the parents suffer from sexual dysfunction, one or both parents' anger and frustration are often handled by attempting to get more emotional satisfaction from the child to compensate for feeling unloved. This may sexualize the relationship to the child, even, if as is true in most cases, there are not specific genital attempts to get satisfaction from the child. In these nonincestuous families, there is often, nevertheless, a heightening of a sexualized interest when there is parental sexual frustration. This factor of parental sexual frustration often adds to the internalized is-

sues of one or both parents, which lead both to their own sexual difficulty and which constitute the unconscious environment of the growing child.

5. Some families sexualize development by generally overemphasizing the sexual aspects whenever possible. These families will read sex into everything, so that the smallest gesture is seen as a seduction and that even the child's earlier developmental steps will be interpreted to mean the child is sexy. Conversely, other families underemphasize sexuality. Normal infantile masturbation, or the most open Oedipal seductive moves of the child will be read as being aggressive, or will be overlooked, as the family attempts to repress sexual development. The same will be true in later stages, perhaps in early adolescence, when the breakthrough of open sexuality or a sexual symptom will be viewed as bad or disruptive.

6. The child grows in the environment created by the parents. This includes the general aspects of the way their internal object life is transmitted to the child through the practical provision of an environment in which to facilitate the child's development and through projective and introjective identification. If the parents have a sexual failure, it impinges on the child's environment. The same internal object issues that contribute to the adults' sexual failure also are present as they try to raise their children. It is primarily these issues that determine the quality of sexual conflict within which the child develops and that will influence the development of personality and of specific symptomatology in childhood, adolescence, and adulthood. These developments are subject to the normal processes of splitting the arrangement of object relations into part objects, which predominate in the early and more primitive management of relationships, and of idealization and denigration, which represent failures to maintain a more realistic view of the object (Klein, 1975a,b; Scharff & Scharff, 1987).

Sexual Symptomatology in Childhood

The baby's sexual functioning is not well differentiated from its total relationship to its mother. Behaviors that prefigure as specifically sexual also mark the beginning of other nonsexual trends. Nevertheless, a number of specific sexual symptoms can be seen in later childhood.

Childhood Masturbation

A four-year-old was brought to the clinic by her mother because of a diffusely anxious picture that included urgent masturbation. The child rhythmically stimulated her genitals with her hands, often while others were present, and would only stop if she were held in the lap of one of the parents.

This girl's parents had both worked, leaving her with a sitter who was the primary caregiver. At 30 months, the family moved, and the child lost the sitter. In addition, the mother had a history of being neglected and sexually abused as a child and had withdrawn sexually from the father.

The little girl's father tended to sexualize events, whereas the mother hoped to minimize the sexual meaning of them. Both parents turned to the little girl for an excited and anxious compensation for their anxious and sexually troubled marriage. Thus, all the factors were present that tend to produce sexual symptomatology: deprivation, change and loss in a sexually critical period, anxiety in the relationship with her parents, sexual anxiety on the part of the mother, and sexual dysfunction in the parents' relationship. With treatment, the masturbation stopped, but even so, her subsequent development was anxiously sexualized.

Effeminate Behavior and Enuresis

A five-year-old boy was brought to the clinic because he was wetting his bed and refusing to go to kindergarten. His appearance was strikingly effeminate, and he preferred dolls and tea parties to boy's games. His father was an alcoholic fireman who was largely absent from the home, whereas his mother was teasing and supportive of his feminine identification. In play therapy, the boy was himself teasing and coy, afraid to express anger. He served the therapist tea and played with the doll family. Over time, he was more able to be angry and to form an affectionate bond with the therapist. As he made an identification with him as a man, he began to play with circus animals who fell and broke their legs, acting out his castration fear. He was now more outspoken at home, and his mother had great difficulty in tolerating his newly found aggression. The therapist now had to work with the mother by helping her to develop the capacity to enjoy her son as a little man, so that she would not discourage the boy's development of a masculine identification.

These examples give a small sampling of a variety of sexual symptomatology appearing in young children. Other pictures that form the precursors of adult symptomatology can also be seen: young children who prefer the clothes of the opposite sex or the taking of an unusual object as a

compulsive object of intense sexualized fascination—the precursor of fetishism.

Sexual Symptomatology in Adolescence

In adolescence, sexual symptomatology is again interwoven with general symptomatology. Promiscuous sexual acting out puts the child at risk for emotional abandonment, pregnancy, and sexually transmitted diseases, the most serious of which is now AIDS. For many, sexual activity runs ahead of the general capacity to relate, leading to a degraded sense of self as the adolescent throws him or herself furiously into sexual relationships that do not foster the solidification identity (Kalogerakis, 1975; Kestenbaum, 1975). Although many adolescents worry about their sexual adequacies and difficulties in adult terms, in this age group the underlying concerns reflect the unfolding of adolescent issues of identity and especially of sexual identity (Erikson, 1950; Sarell & Sarell, 1979). Adolescent pregnancy is a common sexual symptom in this age group.

A 15-year-old girl who felt she could never win her parents' approval began having unprotected intercourse with an 18-year-old boy who had been his family's black sheep and wanted to compensate by taking care of a family of his own. When she became pregnant, she wanted to improve on her own life by taking care of her unborn child. Her refusal to have an abortion despite her parents' wishes and in the absence of a moral objection in the girl was meant to communicate to her mother that she was independent and could win the control battle of adolescent separation. Because the boy could not support a family, the couple planned to live in a mobile home in the girl's parents' driveway, perpetuating a kind of umbilical connection to her parents.

Here, the issues of adolescent sexual identity formation were confused with each adolescent's difficulty in working out an adolescent separation from the parents (Blos, 1967). Their sexual bond and the meaning of the resulting pregnancy reflected these developmental blocks.

Sexual Difficulties in Adulthood

The psychologically caused sexual disorders of adulthood have a developmental base. This is not to underestimate the number of sexual disorders that have a physiological basis, as explored in Masters and Johnson's original work (1970), and synthesized by Kaplan (1974, 1979, 1983, 1987). Where there is question of organic contribution, medical study must be done. With advancing age, the percentage of disorders that are physiologically based increases. For instance, in men, impotence in the under-40 age group is mostly psychologically caused, whereas in the over-40 group, impotence is more often biologically caused (Kaplan, 1979).

Early life experience plays a role in the development of sexual difficulty in adulthood just as it does in childhood. The final common pathway for emotionally caused sexual difficulty is anxiety. That anxiety which is closest to the event of an attempt to perform sexually (so-called proximal anxiety) has been described by Masters and Johnson (1970) as the "spectatoring" and self-judgmental attitudes that constitute the immediate interference with sexual performance. However, behind proximal anxiety lies a series of life experiences carried by the internal object organization of each individual. These constitute a history that can make sense of why sexual difficulty has developed. The quality of early relationships is a primary factor in the determination of current difficulties. This is not to say that the old relationships are "the cause," but that they often form the models for internal object relations that are imposed on the current reality. These early models continue to influence the present, because they constitute an important and inhibiting piece of the internal psychological structure.

Sexual Dysfunctions and Adult Sexual Symptomatology

Adult sexual dysfunctions have been classified by Masters and Johnson (1970) and by Kaplan (1974, 1979, 1983, 1987). Generally, these are seen as relating to the phase of the sexual response cycle, as described by Masters and Johnson, with the addition of the first phase of the cycle of desire. The phases of the sexual response cycle are: (1) desire, (2) arousal, (3) plateau period during which arousal is maintained (no specific disorders), (4) orgasm, and (5) resolution (no specific disorders).

The desire phase disorders (inhibited or exaggerated desire) are most regularly associated with unconscious developmental issues, whereas orgasmic phase disorders (anorgasmia or premature ejaculation) are most likely to be a combination of learning disorders, sexual naïveté, and cultural or

religious interference. Those in the arousal phase (delayed ejaculation, vaginimus, psychological impotence, lack of enjoyment despite arousal) are a mixture. Each disorder has to be assessed by an individual case method in order to determine the degree to which symptomatology is (1) physiological versus psychological, (2) reflects difficulty in the current relationship as opposed to the overall capacity of each of the partners to relate, and (3) is a function of conscious difficulties as opposed to unconscious difficulties.

Concerning the Lack of Specificity in Cause and Effect

In these disorders, we cannot make exact links between a specific insult and the specific sexual difficulty. This inability remains because although we examine the effect of family events on the intrapsychic world, the meaning of those events is always mediated by what is in the child's mind at the time something occurs. This, in turn, is already a function of previous events and of the fantasies at the time of earlier events. Despite the resulting inability to predict, an object relations theory contributes a consistent way of understanding how the individual has interpreted and organized his experience.

Sexual Identity

Sexual identity includes both the issue of gender identity—the feeling of which sex one belongs to and the question of object choice—and whether one selects heterosexual or homosexual objects. Both of these have their origin in the earliest psychosomatic partnership. Lichtenstein (1961) held that the mother gives form to an "identity theme" in the baby, beginning with the feeding relationship which is different for boys and girls. He describes the interaction between mother and child "between two partners where each partner experiences himself as uniquely . . . capable of serving as the instrument of the other's sensory gratification . . . a partnership of sensual involvement" (p. 47). The child has an "innate body responsiveness . . . [which is] sexual because it forms the matrix of later sexual development" (p. 280). Stoller (1968) emphasized the central role of early parental handling in the development of what he termed "core gender identity"—the innate sense of being a boy or girl which he sees as occuring in a nonconflictual sphere of early development. In the transsexual patients he described, the mothers had handled their sons in ways that interfered with the development of this core gender identity. Stoller also suggested that feminine development proceeds with a second stage, which is a defensive movement against the girl's increasing awareness of genital difference. Money and Ehrhardt (1972), studying children with anomalous chromosomal and hormonal makeup, also identified the period between 18 to 24 months as critical for the establishment of gender identity and emphasized that it is the sex in which the infant is reared by the parents during the first two years that is the major determinant of gender identity.

Roiphe and Galenson's (1981) observational data on the role of an early genital phase between 15 and 24 months led them to the conclusion that

> it was the differences in the reaction to the awareness of the genital difference that seemed, above all, to mark the divergent paths each sex would take, a finding that strongly suggests that the second half of the second year of life is a critical period for the development of the sense of sexual identity. (pp. 272–273)

They stress both the role of genital arousal and the differentiating role of parental objects. For the boy, the availability of the father confirms the phallic body image and supports him in the eventual acknowledgment of the absence of a penis in his mother, a process which continues into the middle or end of the third year (pp. 273–274). For the girl, there is more depression at the recognition of genital difference and a strengthening of identification with the mother. Here, the father offers support to the sense of being loved during this loss.

In all views since Freud, the role of Oedipal organization has been emphasized. However, in Freud's original view (1905, 1931) the role of the observation of genital difference, the reaction to which he termed the castration complex, and of triangular object choice were first observed during the Oedipal era. Recent contributors (Roiphe & Galenson, 1981; Scharff & Scharff, 1987) have emphasized the fact that Oedipal development represents a resexualization of development, a renewed focus on the genitals combined with the child's enlarging capacity to understand the implications of being interested in two parents. The centrality of the child's internal experience—of its internal object relations—is emphasized by the similarity of the developmental path for children with only one parent, children who supply the ingredients of the missing parent through a combination of their own internal world and the representation of the parent

of the other sex provided to them by the one parent they do have.

Homosexuality and the Paraphilias

Initially, homosexuality was considered to be simply the most common among the many perversions (Freud, 1905). However, later work allows us to see that each syndrome is the result of complex factors in the object relations of the individual—an intricate mix of factors centering on the handling of object loss. There has been a good deal of controversy about the role of innate hormonal balance in the predisposition to homosexuality. Meyer (1985a) concluded

> In terms of present knowledge, there appears to be no definitive action of hormonal or other biological factors on sexual identity or object choice. Rather, there appears to be a complex interaction between biological, environmental, and mental factors that affect the expression of sexually dimorphic behaviors. The concept of a "nature vs. nurture" controversy seems anachronistic. (p. 1058)

Although much controversy also exists about whether ego-syntonic homosexuality is a pathological process, we can nevertheless comment on the contributions to development that result in a gender identity that is intact, but accompanied by homosexual object choice. Many authors have contributed to the view that difficult relations with the mother and an absent or weak father contribute to such a picture. Following the work of Roiphe and Galenson (1981), we can now say that experiences from the second year of life on, including Oedipal development (Meyer 1985a), and, I believe, often crucial additional turning points as late as adolescence, all play a role in the development of homosexual object choice.

In homosexuality, as in the paraphilias, the expression of a remnant of childhood sexuality is a required component of adult sexual expression. The person uses this piece of surviving infantile sexuality to allow displaced expression for other, more feared pregenital components and object relationships that are only thereby susceptible to repression. This is known as the Sachs Mechanism (Sachs, 1923). Kernberg (1975) and Socarides (1978) have noted that homosexuality may express a range of internalized object relationships based on increasing levels of ego maturation. In Oedipal level homosexuality, submission of an infantile self is made to a domineering parent of the same sex. In high-level pre-Oedipal homosexuality, the object stands partly for the self and partly is a representative of the pre-Oedipal mother. In the less mature, narcissistic brand of pre-Oedipal homosexuality, the object is purely a representative of the grandiose self. Relationships are brief, and there is little concern for the object as such. In schizo-homosexuality, homosexuality co-exists with schizophrenia. There is a lack of separation of self from object (Socarides, 1978).

The family constellation is clearly crucial in the development of both male and female homosexuality. Bieber and his colleagues (1962) found that in men with severe homosexuality, there was a pattern of a detached hostile father and an overly close, seductive mother who dominated the husband. Homosexual solutions in women share the major characteristics with those of men, involving a mixture of pre-Oedipal and Oedipal issues and failures in relations to both mother and father. Saghir and Robins (1973) noted that a variety of patterns exist from hostile, domineering mothers and distant, unassertive fathers to those lesbians who have had intense, seductive relationships with their fathers, but narcissistic and detached mothers. They conclude that the common underlying factor is the presence of a significant anti-heterosexual pattern in the home.

There are essential contributions to homosexuality by both parents and by their relationship to each other. When family interactions are severely disturbed, the homosexual child emerges as the focus of parental disturbance. Often the relationship with mother is close but ambivalent. The boy who will become homosexual fails to let go of an early identification with her to form an adequate identification with his father. The girl who will become lesbian cannot risk the mother's displeasure in solidifying her father as an object choice, or she faces danger in the relationship with him. Overt homosexuality develops when it is supported and encouraged by the parents' conscious or unconscious processes (Kolb & Johnson, 1955), when parental needs override the child's needs for autonomy in the separation–individuation phases (Socarides, 1978), and, in sum, when familywide conscious interaction and unconscious projective identification support the development of the splitting and sorting of internal objects such that the exciting, safer internal object is identified with the same sex and the threatening object is opposite-sexed.

The Paraphilias

Paraphilias, previously known as perversions, are repetitive, involuntary situations of sexual arousal or masturbation characterized by unusual or bizarre imagery or acts involving the use of nonhuman objects for arousal, accompanied by the imposition of humiliation or suffering, or by involvement with nonconsenting partners. A special fantasy, which has conscious and unconscious components, is inevitably an underlying element. Arousal and orgasm are dependent on the elicitation of this fantasy, and its influence and elaborations extend beyond the sexual sphere to pervade the individual's life (Meyer, 1985b). Sex and aggression are fused in these symptoms, so that the paraphilic always expresses anger toward the sexual object along with sexual arousal. This group includes fetishism, transvestism, sexual sadism and masochism, exhibitionism and masochism, pedophilia, zoophilia, and a group associated with excretory functions, such as coprophilia and urolagnia. These disorders are far more common in men than in women, which Meyer (1985b) attributes to the threat posed to boys' genitals due to the obviousness of their anatomy.

> Male perversions are external, often flamboyant structures with concrete props that tell the story of triumph over a castration threat. Female perversions are largely unobtrusive, being revealed by a particular willingness to accommodate to the perversion of sexual partners . . . a clandestine insurgence against a sense of genital inferiority. (p. 1069)

For a given family and a given individual, one compromise formation allows expression of individual and family conflict. A function that ordinarily has an important but minor role in sexual expression comes to the forefront. Anxiety and aggression are inextricably bound with sexual desire and arousal. In classical psychoanalytic theory, it has been suggested that the driving force is the boy's concern with castration and with using unconscious fantasy to calm his anxiety by establishing over and over the existence of the maternal phallus. Roiphe and Galenson (1981) described an infantile fetish whose development echoed the attempt to develop a secure sense of genital identity in a boy. They postulated that the ordinarily transient phase of such issues exists on a continuum with the adult persistence of paraphilias. Such unconscious fantasies can be unconvered only when in-depth exploration can be used as in psychoanalysis or detailed observation of child development. The paraphilias represent ordinary transient pieces of early development, fixated as required by stereotyped components of arousal through the confluence of many unconscious factors during development. What has not been as well described, but is clearly crucial, is the family support and encouragement, through projective identification, of such a solution to familywide problems in relating. Thus, a transvestite boy can be seen to be internalizing and expressing his parents' shared fear of the phallic woman and of the threat of castration, as well as his own developmental fears. A girl who loves suffering in sexual relationships can be seen to have incorporated the suffering of both father and mother in sexual terms, which express their disappointed sexual longing.

Case Example

> A couple, married twelve years with two children, illustrates some of these points. The wife could no longer tolerate the husband's insistence that she dress in wrestling clothes and consent to "wrestle" before intercourse. The husband could not achieve arousal without these circumstances. A joint interview revealed that this obligatory quality to their sexual life had not been present in the beginning of their marriage, but had become pressing after the birth of their son, eight years ago. The husband had experienced this child as a potential rival, reactivating his fear of exclusion from his own parents. His father had been largely absent from the family although he had rough-housed with the boy when he was very young. The husband's mother had been attentive but harsh, preferring his two sisters. He had spent many boyhood afternoons watching wrestling on T.V. with his beloved grandfather, and had engaged in an arousing form of "horsing around" with male peers during puberty, a time when he felt most excluded from his family.
>
> The wife felt lost in her own large family. Her father was also largely absent, except that her parents argued vociferously in her presence. She also felt excluded by her mother's preference for others. When her mother became ill, she cared for the younger children.
>
> For the husband, the wrestling had come to symbolize the required conditions of being loved while being able to discharge rage at feeling excluded. A resurgence of sexuality and aggression during puberty contributed to his choice of an act required for arousal. For the wife, the wrestling had originally been a form of suffering which was not too great a price to bear if it insured her husband's interest. With time and maturation, her tolerance decreased, at which time the husband's dependence on her prompted him to ask for help. This couple gave up stereotyped sex within a few sessions of therapy, but stayed to explore anxieties about rejection.

This vignette illustrates that (1) in couples, there is often a mutuality to the paraphilia that satisfies the object relations needs of both partners. (2)

Paraphilias express and symbolize the object relations and fantasies of the person in the deviant piece of sexuality. If a developmental history of the individual and couple can be obtained, it will make sense of the symptom. (3) Like all sexual symptoms, the severity of paraphilias varies along a continuum from the situation in which they are normal variants to those of severe pathology. In the mild cases, they will be more distressing to the patient and partner and will be more easily treated. Severe cases will often be more difficult to treat. Alertness to the picture will uncover the milder cases that will respond more readily to treatment.

Object relations explorations of families of incest have uncovered the multigenerational transmission of incest, often accompanied by subtle or overt forms of physical abuse, which correspond to the combined object relations of the parents (Kaufman, Peck & Tagiuri, 1954; Lustig, Dresser, Spellman, & Murray, 1966; Winer, 1988). The mothers in such families have often been sexually abused themselves, whereas the fathers come from similarly deprived families characterized by poor boundaries and untamed aggression. The parental marriage and sexual relationship may be poor, while a boundary around the family is rigid, restricting the family members to getting satisfaction of frustrated needs inside its walls.

Sexual Maturity

What constitutes normal sexual maturity, the favorable outcome of sexual development? Balint (1948) suggested that mature love combined the pregenital elements brought into a sexual episode in foreplay and integrated the tenderness of a successful infantile inheritance into genital expression between mature sexual lovers. Levay and Kagle (1977) characterized three levels of sexual achievement: those with the ability to achieve (1) pleasure for the self, (2) intimacy with another, and (3) cooperation with the other to reach higher goals. Sexual maturity is not, however, a fixed quality. It is the development of a capacity for the level of sexual exchange appropriate to each stage of life (Scharff, 1982). At each stage this involves the capacity for pleasure for the self, exchange with the other, and cooperative efforts that influence the wider family. The earliest level relates to sexual interest stemming from an interest in one's own body as experienced with the mother of early attachment. The second level is that of sexual interest stemming from an interest in the mother-of-separation as object. The aim is not sexual satisfaction but the securing of a safe, pleasurable attachment to the mother. The third level is sexual interest stemming from complex relationships, where cooperation, mutual satisfaction, and flexible interchange are combined with genital expression. The fourth level uses the achievement of a loving relationship to build toward the family, a sublimated investment in children, work, and the creative products of intimacy, cooperation, and secure self-reliance.

In adult life, sexuality has the potential to revitalize the connection between the internal figures of the past and current family, gathering the myriad aspects of physical and emotional growth, as it brings the problems and reassurances of past relationships forward into current life. Sex forms the bodily link in this process, acting at an unconscious level to maintain a loving attachment to primary objects despite life's many assaults on that attachment. The many faces of sexual psychopathology represent the struggles of individuals to continue that task in the face of the roadblocks to love and satisfaction erected in the course of development.

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PART VI

Intervention

CHAPTER 35

Institutionalization Revisited

Expanding Views on Early and Cumulative Life Experiences

Sharon Landesman

The study of the effects of institutionalization has been affected profoundly by institutionalization practices themselves and by changing conceptualizations of what comprises optimal mothering. Dramatic descriptions of child maltreatment in orphanages, from Dickens's *David Copperfield* and *Oliver Twist* in mid-nineteenth century England to Gray's "Little Orphan Annie" created in the 1920s, captured vividly the plight of many children who were placed in the care of strangers. The motives of nonrelative care providers always have been suspect, based on opportunities for monetary gain and for deriving perverse pleasures from abusing children outside the purview of the conventional world. In the late 1930s and early 1940s, disturbing clinical observations surfaced about the grave effects of institutionalization on children. These led to the seminal studies of Bowlby (1940, 1944, 1951, 1958, 1969), Spitz (1945, 1946, 1947), Bender (1947), Bender and Yarnell (1941), O. L. Crissey (1937), Skodak and Skeels (1949), and Skeels (1966), who opened the field of systematic inquiry about the early care-providing environment.

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Early Themes in the Study of Institutionalization

Two themes dominated the early scientific forays into this field. The first was concern about the child's tragic loss of mother, synonymous with a signaled disruption in the child's primary source of nurturance. The mother–child relationship was assumed to be essential, universal, and unique in setting the stage for the child's emerging trust in others, confidence in self, responsiveness to cognitive and linguistic stimulation, and willingness to explore the world beyond this first and compelling dyadic relationship. What effects did maternal loss have on the child's psyche and subsequent ties to the social world? The second theme revolved around the significance of environmental variables and what actually happened to children when they faced life in an accidental context rather than in a nuclear family. The accidental context of child institutions was characterized by a complex array of worrisome environmental conditions, including (1) the fluctuating presence of multiple care providers (usually referred to as custodians, only rarely viewed as surrogate mothers), (2) large and often strongly influential peer groups, (3) regimentation and restriction of daily activities, (4) lack of appropriate and diverse cognitive stimulation and feedback, (5) absence of privacy, (6) generalized deper-

sonalization of social interactions, and (7) the ever-present possibility of neglect and abuse—a possibility all too frequently realized and concealed from public knowledge. The paramount aim of these investigations was to measure the nature and extent of damage inflicted upon children who experienced such deviant and depriving environments and ultimately to learn how to provide effective social and cognitive remediation within these nonfamilial environments.

The early investigations sought to provide a clinically rich documentation of the effects of institutionalization, including both immediate and long-term consequences, ranging from apathy, failure-to-thrive, or death from "tristeza" (infant depression), to later social withdrawal, intellectual deficiency, and rebellion. At that time, orphanages existed in almost every American and European community, as places of refuge for children who lost one or both parents through death, insanity, illness, or poverty. Institutions for individuals with mental retardation also were starting their ascent to a peak occupancy in the mid-1960s, an increase largely attributable to two successive factors: a vigorous eugenics movement in the early part of this century, which favored massive institutionalization of "intellectual and moral degenerates" to prevent their continued procreation; and physicians' endorsement of institutionalization for "defective" children, directed mostly toward white middle-class parents who were urged to make this decision as early as possible to avoid inflicting harm and probable pathology on their entire family. In fact, the institutions for "mentally retarded" children became society's refuge for a very heterogeneous group of children, all too frequently including nonretarded and minimally retarded children who simply needed a place to live. These prevailing and highly visible practices contributed to the interest that child psychiatrists and developmental psychologists expressed in understanding the consequences of institutionalization, especially on infants and very young children.

This interest in the effects of institutionalization was never restricted to the academic community. In 1949–1950, Bowlby was invited by the World Health Organization to summarize the mental health aspects of the world's homeless children. His conclusions (Bowlby, 1951) affirmed the importance of early bonding in the first 6 months of life, the vigorous protest and subsequent emotional retreat displayed by well-attached infants when they lost their mothers, and the range of severe disturbances in older orphaned children who were not permitted to mourn openly and to share their loss with a sibling or close relative. The child's feeling of abandonment was hypothesized to be central, lifelong, and extremely difficult to resolve—a feeling later manifest in adult symptomatology, especially severe separation anxiety, recurrent bouts of depression, and psychosomatic problems. More extreme reactions, usually apparent by adolescence, included open rebellion and antisocial acts, suicidal inclinations, and the failure to establish or maintain intimate relationships with others. Indeed, Bowlby's review contributed in a major way to an enhanced construct of "attachment," one which recognized variation in the quality of the mother–infant relationship and posited a far-reaching influence on development, extending beyond the immediate provision of physical care to a dependent infant.

Controlled Environments and the Search for Causal Relationships

A rich naturalistic and experimental animal literature on the effects of early experience complemented the (fortunately) more limited studies of human development in private orphanages and large public institutions. Here, the theoretical focus was a broader biobehavioral one (e.g., Harlow & Harlow, 1965; Harlow & Zimmerman, 1959; Hinde, 1974; Lorenz, 1935; Rosenblum & Kaufman, 1968; Suomi & Marlow, 1978), in contrast to the predominantly psychoanalytic one in the human literature (see Bowlby, 1958, for a sharply delineated psychodynamic view of the child's attachment to mother). The central questions asked were: (1) What are the effects of specific types of social and sensory deprivation (alone and in combination) on the developing organism? (2) Do these effects occur only during critical or sensitive periods in development? and (3) Can subsequent normative environments provide compensatory experiences sufficient to offset earlier deprivation? The European ethologists had demonstrated convincingly that altered environments could disrupt normal maturational processes, and that the nature and irreversibility of these disruptions related to the timing of the events (i.e., the organism's stage of development). Extreme environments undeniably created extremely aberrant or species-atypical forms of behavior. Beyond this, however, what investigators sought was a more precise identification of the environmental variables that represented the minimal elements

necessary to support normal development, especially in the social and cognitive domains.

The collective results of the animal and human research did not yield clear or consistent answers. The important ethological construct of a critical period (a time when certain environmental input must occur in order for the organism to develop particular normative behavior patterns—i.e., subsequent exposure would not offset earlier deprivation), evidenced most elegantly in ducks, never received equally compelling support in the study of primate behavior. What complicated the field was the accumulation of data demonstrating (1) species-specificity in response to social and sensory deprivation (e.g., closely related species showed wide variation in the form and severity of effects, with some species apparently remarkably resilient to negative effects of extreme isolation and deprivation), (2) considerable individual differences (most notably, sex differences) in response to early deprivation, and (3) the effectiveness of compensatory environments and social challenges—that is, the demonstration that alternative environments and experiences could substitute functionally for good "mothering." For example, in rhesus monkeys, the early presence of a peer group or a stable cadre of mother surrogates appeared sufficient to prevent the emergence of an "institutional syndrome" in animals reared apart from their parents and naturally occurring social groups. (The institutional syndrome refers to the behavioral pattern rhesus monkeys displayed when reared in total isolation, a pattern strikingly similar in topography to that shown by retarded individuals in deprived institutional settings: high rates of bizarre, stereotyped, seemingly nonpurposive, self-directed and sometimes self-injurious behavior, as well as grossly inept social behavior.)

Another remarkable finding that presented a substantive challenge to earlier reports was that the apparent intellectual deficiency of isolation-reared monkeys was, to a considerable extent, an artifact of the high arousal or distress they experienced when placed in novel test situations. When granted sufficient time to adapt to the new test environment, these severely deprived animals showed surprisingly higher levels of cognitive competency in solving problems they previously had failed.

The three decades of vigorous human and animal research generated findings that were studied meticulously for clues about basic processes of adaptation and the environmental elements that represented universal essentials in promoting social, intellectual, and emotional development. More than enough evidence existed to confirm the observation that extreme neglect, abuse, and deprivation could cause permanent damage in infants born without defects. The general conceptual framework about the social meaning of early attachment had both evolutionary and psychological appeal, and thus was adopted without rigorous testing. The ethological notion of critical periods was modified to that of "sensitive periods," times during which organisms were more receptive and more responsive to certain types of stimulation, usually with less rigid time boundaries. This idea of optimal or favorable (rather than exclusive) time periods for environmental input was a safer and more optimistic one, and received indirect support from multiple sources, ranging from anecdotal and clinical evidence (e.g., from infants born profoundly deaf, data indicated that the timing of first exposure to formal sign language resulted in differential abstract reasoning abilities) to the newly emerging field of infant development, which provided reams of fascinating data about infants' age-based changes in awareness of and response to variables in their environment (e.g., preference for certain forms of visual patterning, interest in objects and their transformations in space, wariness to strangers). The earlier specific hypotheses about what was most important in maternal behavior or in the mother–infant relationship (e.g., the significance of a singular primary relationship with an adult, tactile stimulation, vestibulator input, frequent opportunities to interact with diverse objects) had failed to gain unequivocal support, and no new synthesis could account for the varied findings. In fact, increasing human evidence refuted even the most straightforward propositions about the necessity of certain infant–environment interactions. For example, Piaget's assumption that an infant's frequent actions upon objects and observation of self in means–ends sequences were critical for intellectual maturation was relegated to correlational, rather than causal, status when infants born without limbs (victims of prenatal exposure to thalidomide), and thus denied the opportunity to engage in typical physical exploration of objects and repetitive self-initiated sequences of object play, showed normal intellectual development.

The studies of institutionalization effects on children with mental retardation did nothing to simplify or resolve these general issues, because greater refinement in design and measurement resulted in more complex and less theoretically cohesive findings. Substantial interinstitutional variation became a well-documented reality (e.g., Balla, But-

terfield, & Zigler, 1974; Butterfield & Zigler, 1965; Klaber, 1969). Accordingly, subsequent efforts to make sweeping generalizations about the effects of institutions *per se* were considered far too simplistic. Even more germane was the finding, replicated in several diverse settings, that some children actually showed gains in intelligence *after* institutionalization (Clarke & Clarke, 1953, 1954; Clarke, Clarke, & Reiman, 1958; Klaber, 1969; Strauss & Kephart, 1939; Zigler, Butterfield, & Capobanco, 1970), whereas other children within the same institutions showed declines. Studies that sought to measure the contribution of discrete environmental variables, such as institutional size, staff : resident ratio, and per capita investment in children, variously reported no effects, small magnitude effects, or counterintuitive effects (cf. Landesman-Dwyer, 1981). The only two conclusions that were upheld with any consistency were that traditional institutions supported bizarre and atypical social behavior and that many forms of aberrant institutional behavior could be reduced, at least temporarily, by increasing the individual attention or the environmental stimulation available to residents.

Thorough and insightful critiques and commentaries were written and provided suggestions for future research (cf. Baumeister, 1967; Butterfield, 1967, 1987; M. S. Crissey, 1975; Heal, Sigelman, & Switzky, 1978; Klaber, 1969; Windle, 1962; Zigler & Balla, 1977). To conduct well-conceptualized and well-designed studies about institutionalization was truly challenging, especially given the heterogeneity of children served by institutions, the biases in placement of subjects in different types of institutions, the difficulty in documenting children's preinstitutional experiences, and the lack of sound instrumentation to guide the measurement of the adaptation process and the environment.

Since the early 1970s, when deinstitutionalization was widely practiced and an increasing number of alternative community-based residences were established, another major wave of environmental effects research was conducted. The questions addressed remained essentially the same, although the focus was on a distinctive population and phrased in more positive, rather than negative, terms. That is, investigators asked (1) what comprises an optimal social environment to support normative experiences, developmental progress in adaptive behavior, and decreases in undesirable behavior? and (2) what variables predict who will benefit from particular types of residential experiences? Consistent with the earlier human and experimental animal literatures, however, most studies comparing community versus institutional placements suffered from the same serious flaws in design, measurement, and analysis as did earlier research. That is, the subjects who entered into different environments were not comparable before moving there. Further, the dynamic components of the community-based residences were rarely monitored and objectively described at a level adequate for understanding the differential outcomes observed. Finally, the field of inquiry still was characterized mostly by a desire to prove that "community living" *could* work (i.e., opposite to institutionalization effects), rather than to understand the developmental processes and person–environment transactions that occurred under diverse environmental conditions. Only recently have there been efforts to hypothesize plausible mechanisms to account for individual differences observed in responses to similar environments (Landesman & Vietze, 1987).

Individual Differences in Response to Environments

Interestingly, the repeated finding of variation in response to institutional experiences or to community living is in some ways quite positive, though seldom discussed as such. That is, some children appear to survive quite well, despite tremendous environmental oppression or lack of normative growth-promoting opportunities. More systematic inquiry into the processes by which these "invulnerable" children achieve positive outcomes has important theoretical and clinical value. Are these children truly *not* affected by extreme environments or do they have special coping strategies to offset the difficulties, obstacles, and inadequacies identified in their environments? Alternatively, would more refined assessment of these children's environments reveal the presence of supportive elements that previously were not recognized? To the extent that compensatory behavioral and/or environmental variables can be identified, for certain children or certain situations, then new treatment interventions can be designed to incorporate these protective elements and can be empirically tested to determine whether they can minimize negative effects of nonoptimal environments for other children as well.

Although the effects of institutional environments were related in various studies to retarded children's earlier experiences and to such variables as age, sex, and level of retardation, a consistent

pattern did not emerge regarding long-term prognosis (Windle, 1962). Because of life circumstances, the preorphanage lives of infants and young children were rarely documented with any credible details, thus preventing *post hoc* study of how temperamental and previous environmental experiences contribute to individual differences in responses. Perhaps most impressive are the biographies of accomplished people who survived the loss of one or both parents early in life, and frequently endured a series of nonparental family homes and/or orphanages or simply entered the world independently before reaching adolescence. Simpson (1987) wrote an interesting popular book on orphans that highlights many of these lives and provides thoughtful insights about individual differences and coping mechanisms, including the use of fantasy, denial, and fabrication. She also posited that all children may endure some "degrees of the parentless state," as seen in Dickens's *David Copperfield,* who starts life with a mother who is still a child and then traverses through many disruptions and disappointments, with mother's love being more or less available at different times (e.g., due to other lovers and being sent away to school). Somewhat ironically, his mother's true death permitted David to see his mother idealistically, returning to the mother of his infancy. This interpretation is compatible with the increasingly endorsed view of a continuum of care-providing environments, which is associated with differential effects depending on individual and historical variables (Sameroff & Chandler, 1975). Even outside the walls of institutions, institutional experiences and a sense of parental abandonment may occur; what contributes to these feelings and how they are transformed over time remain uncharted.

Modern Forms of Institutionalization: Distinguishing Features

An explicit definition of what comprises an institutional environment or an institutional experience was seldom provided in the earlier scientific literature. Children's institutions were so obviously distinctive from natural family homes and so inextricably associated with a relatively constant set of negative qualitative attributes ("blandness, drabness, uniformity, and lack of individual attention," Flexner, 1987) that further definition seemed superfluous. The oppressively inhumane institutions and impoverished orphanages of the mid-twentieth century have been closed or transformed markedly (e.g., Rothman & Rothman, 1984; Wolins & Wozner, 1982). Efforts to provide truly habilitative and individualized care within group settings for children with special needs have increased. At the same time, the success of these modifications in residential care settings has been uneven, and frequently structural reforms have not resulted in functional improvements or a higher quality of life (Landesman, 1987; Landesman & Butterfield, 1987). Further, the possibility that institution-like environments can exist outside traditionally recognized large institutions has been raised (Edgerton, 1988; Landesman, 1988). That is, the environmental elements hypothesized to contribute to negative behavior and poor developmental progress may be present in such places as foster homes, group homes, or day-care settings. From this perspective, the *functional characteristics* of children's primary care-providing environments (e.g., amount and quality of social interaction, developmentally appropriate activities, opportunities for choice and variety) are hypothesized to be the critical mediating factors in producing institutional effects, rather than the structural characteristics (e.g., administrative category, adult-to-child ratio, size of facility).

From this functional perspective, the most salient negative features associated with institutional environments, including modern day facilities, are (1) a rigid and slow-to-change administrative organization with inflexible operating policies, (2) lack of frequent and sensitive social interactions, (3) instability and unpredictability in the presence and behavior of adult care providers, and (4) poor resource utilization, often associated with a closed system and relative isolation (Landesman, 1988). A definition of institutional environments that underscores the child's day-to-day experiences, the social and physical resources within the environment, and the opportunities for responsiveness to individual children places the study of institutional effects within a much broader developmental framework.

One of the most prevalent forms of institutional care in present times is poor quality day care for infants and toddlers. The recent surge of systematic study of day-care environments has resulted from the same concerns that guided much earlier research concerning the effects of institutionalization. Scientific inquiry on the topic of day care would be well informed by carefully reviewing the serious flaws that plagued decades of research on institutionalization. To understand the role of young children's early and multiple care-providing environments, the scientific questions must be integrated within a biosocial and ecological framework

that seeks to discover dynamic patterns of individual patterns of development that emerge within functionally cohesive environments (Bronfenbrenner, 1977; Landesman & Ramey, 1989). A child's day-care experiences need to be evaluated within the context of his or her other life experiences, and the rate and nature of environmental changes (both social and physical) over time. Somewhat disturbing is the paucity of knowledge about how children's everyday transactions with the environment at one age affect their transactions at later ages. What has been absent from most scientific study of young children is a cumulative and contextual life experiences perspective, despite the fact that all major developmental theories posit the importance of earlier environments for later adaptation.

In theory, there are many other care-providing environments—including family homes—at risk for having at least some of the institutional qualities of regimentation, insensitivity in social interactions, instability, unpredictability, and inadequacy in resources. It is important to remember that environments always represent the joint product of people and physical resources, and that environments are not static. A care-providing environment may be highly effective at one time, but not necessarily at other times. Similarly, a certain combination of environmental features (i.e., type of environment) may produce positive developmental outcomes for the majority of children, yet fail to do so for a defined subgroup. What is vitally important is the search for explanatory mechanisms to account for such individual variation in responsiveness to environments.

Although traditional institutions for handicapped children and holding institutions for adoptive and foster children have been reconfigured so that their institutional structural features are much less apparent or eliminated, the functional experiences of children in these altered environments remain negative, with corresponding psychological scarring comparable to that observed by the pioneering investigators in this field. Even community-based family care homes, group homes, and natural family units sometimes display the depersonalized care historically associated with very large public institutions.

Toward a Developmental Taxonomy of Care-Providing Environments

That the measurement of care-providing environments and environmental variables has lagged far behind the measurement of individuals has been well recognized and vigorously lamented. The number of standardized tools is few, and the range of environmental variables considered is limited. Yet the developmental literature continues to rely on relatively simplistic, and predominantly structural and demographic variables to describe children's environments. Further, the cultural biases in selecting environmental variables to include in longitudinal research are not adequately recognized as restricting our theoretical advances about child–environment transactions. For example, the present-day values and parenting practices of upper middle-class white families are those that are most frequently assessed, because these are hypothesized to be the environmental variables that promote children's development. Although children in these environments typically display better than average developmental progress on the outcome measures obtained—notably IQ scores, language skills, and school achievement—there is a serious flaw in conducting an essentially ahistorical and ethnocentric study. That is, the evidence is compelling that the structural and demographic features of upper middle-class white families have changed dramatically within this century. Many children today are living in environments considered pathological by the normative standards of earlier generations (e.g., only children, single-parent families, working mothers), yet these children survive these environments well—that is, they do not show major negative effects (at least as defined by the investigators and parents) and they do progress well cognitively, socially, and emotionally. What then have been the surviving functional features of successful children's early environments, despite the marked structural changes? Have there been new (alternative) environmental variables that have been added that, in turn, have provided functional equivalents to elements no longer assured in children's everyday lives? Are there some classes of environmental variables that have developmental consequences only when certain other resources are present or absent, or only for certain types of children?

Increasingly, answers to questions about the effects of maternal loss and institutional environments indicate the potential value of a more formalized taxonomy of care-providing environments (Landesman, 1986). Arbitrary categorizations of home and other care-providing environments that are based primarily on current standards or labels are highly vulnerable to cohort effects and thus limit the potential accumulation of valuable scientific in-

formation about human adaptation. Similarly, classification schemes derived from statistical groupings of a large number of environmental variables have not demonstrated good utility or validity. To date, no single broad-based theory has been transformed into a method for measuring and describing (1) the objective external features of children's home and out-of-home environments; (2) the subjective or experiential environments of children, which may or may not be compartmentalized by geographical location; and (3) the relationship between objective and subjective environments. Increasingly, children spend large amounts of time in two or more primary residences during their early development, with significant changes in the adults who are their primary or shared care providers. Assessing the continuities and changes in children's environments, across settings and sequentially, is vital. We must incorporate the multidimensional and variable environment as a significant codeterminant (along with biological and maturational influences) of a child's development, a codeterminant worthy of developing theoretically cohesive and psychometrically strong measurement tools and classification schemes.

Conclusion

Institutionalization as an exemplar of an extremely aberrant environment for the development of children provided an important historical opportunity to identify important themes related to normative experiences and their correlates within specified environmental contexts. The deep psychological consequences that motivated investigators originally to study the effects of institutionalization remain worthy of continued scientific inquiry. What happens when children's individuality is ignored? If feelings of loss and anger, especially toward parents and other primary care providers, cannot be expressed, in what ways will children's emotional sensitivity and subsequent interpersonal attachments be affected? When cognitive challenges and environmental stimulation are substandard, how effective are subsequent remedial efforts to correct for these environmental deficiencies?

Reflections on the combined human and animal literature on the effects of early deprivation, especially maternal deprivation, and subsequent studies on institutionalization confirm three broad conclusions. Interestingly, these conclusions have important implications for understanding person–environment transactions and the consequences of experiences within a contextual life-span framework. They are not unique to orphans or handicapped children, nor to those in formally identified institutions. These conclusions are: (1) environments are not inherently good or bad; (2) the consequences of a given environmental experience are as much determined by what follows and what precedes it as by the experience itself; and (3) the negative consequences associated with disruptions and environmental changes can be offset, at least in part, by providing some stability in at least one primary close human contact. These findings now need to be integrated with a biosocial functional taxonomy of environments, to further specify the means by which children develop competencies and emotional maturity and trust.

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CHAPTER 36

Perspectives and Interventions

Psychoanalysis and Psychoanalytic Psychotherapy, Family Therapy, and Community Psychology

Mary D. Laney

Timothy, a dark-haired child with large brown eyes, is noticeably small for nine years of age. He rarely smiles, does not have friends, and according to his mother, "will never be Mr. Popular." In addition to problems at school and difficulties sleeping, Timothy has ulcerative colitis. Like thousands of other children, Timothy suffers from one of many forms of childhood psychopathology.

Introduction

Today, mental health professionals, families, and communities have available a wide range of options from which to choose psychological interventions for those children who are at-risk for the myriad problems of daily living or who, like Timothy, suffer from psychopathology. In order to best determine the most responsive form of intervention for such children, it is helpful to view a child's manifest psychological difficulties from a perspective of developmental psychopathology. From that perspective, psychological disturbance, symptom formation, and problems of maladaptation or coping in daily living may be viewed against the salient tasks of the given developmental period in which they emerge and progress. Moreover, from a longitudinal perspective, transactions from prior periods, environmental challenges, and the secular accumulation of biological influences impinging on an individual become more lucid.

The child who is at-risk for psychological disturbances differs greatly from the child who exhibits pervasive and long-term psychological disturbance. Both children differ from the child who exhibits transient and reactive symptoms related to environmental changes, chronologic age, and increased coping demands. In view of the level and course of pathology in each of these children, what may prove to be an effective form of intervention for the former child may prove less effective for the latter. In most cases, the final choice regarding the selection of a particular form of treatment is multiply determined and includes the consideration of treatment costs, the onset and severity of the problem, and program availability. To the extent that a knowledge of developmental psychopathology can be integrated in that decision, treatment selection will be enhanced.

Psychoanalysis and psychoanalytic psychotherapy, family therapy, and community or systems interventions are among the dominant psychological treatment choices. Major and looming points of distinction among these various forms of interven-

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tion lie not only in the object (recipient) of treatment, but also in the means of facilitating or fostering mental health. Those distinctions are of paramount importance in treatment selection. In the remaining portions of this chapter, the approaches to treatment characterizing each of these intervention options will be explored in relation to their underlying theories of psychopathology.

Psychoanalysis and Psychoanalytic Psychotherapy

> When one encounters characteriological disorders which are based on early life fixation or personality disorders indicating internalized conflicts, psychodynamically oriented psychotherapy or psychoanalysis is often the recommended form of treatment. While the tendency of the disorder to interfere with the further development of the child establishes the need for treatment, the level of fixation and the degree of internalization determines the choice of treatment. (Neubauer, 1972, p. 223)

Psychoanalysis and psychoanalytic psychotherapy are based on an understanding of the child from a developmental point of view. From that perspective, psychopathology usually is conceptualized along designated lines of development and their relationships to one another (Freud, 1962). Such lines of development include sources of strength and weakness, internal conflicts and defensive modes, congruences and incongruences, ego functioning, and superego course (Rexford, 1982).

During an analytically oriented assessment, the therapist or child analyst seeks to determine the child's strengths and to identify sources and characteristics of the problems that brought the child into therapy. The initial evaluation explores the child's capacity to relate to another person, the state of various ego functions, physical growth and intellectual capabilities, resourcefulness, and relationships with family and friends. Information gathered through the initial evaluation and parental history is used to conceptualize the child's pathology according to the level and nature of developmental fixation and internalized conflict(s). Standard criteria that are used in establishing the indication for psychoanalytic treatment for a child typically include the presence of internalized conflict, the capacity for verbal expression, the ability of the family to participate minimally in the child's treatment plan, and the family's capacity to accept the child's improvement.

Approaches to Treatment

The psychoanalytic treatment of psychological disorders in infancy and childhood is usually long-term psychoanalysis (3 to 5 sessions per week for several years) or psychoanalytic psychotherapy (1 or 2 sessions per week for 1 to 2 years). Although both treatments are designed to remove barriers to the child's progressive development, child psychoanalysis is more intensive and requires more time than psychoanalytic psychotherapy. The goals of child psychoanalysis, however, are more ambitious and seek to achieve a resolution of unconscious conflicts and rearrangement of the underlying cognitive structures in order to enable the child's development to procede undistorted by fixations at earlier periods in life or by anxieties and fantasies.

The means of achieving the aims of psychoanalysis and psychoanalytic psychotherapy with children are, in general, the analytic techniques of reconstruction, affective reliving, and the attainment of affective and cognitive insight. Analytic tools include the therapist's respect for the child, the use of the analyst as a primary object, the use and analysis of transference, and the consideration of developmental, environmental, and biological factors (Chess & Thomas, 1984). Although the techniques used are frequently the same or similar to those used with adults, resistances to therapy are usually stronger in children, and psychoanalysis or psychoanalytic psychotherapy may be more difficult with children for several reasons. Because the child does not choose to be in psychoanalytic treatment, has no long-term view and hence no rationale for tolerating the difficulties of therapy, he (or she) may feel more threatened by analysis or wish to flee therapy, especially during the intense periods of negative transference. Moreover, the child may experience strong resistance to the unconscious return to former developmental states.

Analytically oriented play therapy has become a major means of accessing the child's inner world and achieving the goals of psychoanalytic treatment of children. Although young children are unable to "free associate" and often lack the language necessary to verbalize their internal experience, daydreams, drawings, and active play with figures, sand, or clay, are used as avenues to the child's unconscious (Sullwold, 1984). In its most classical form (Klein, 1955), play therapy seeks to bring into the child's awareness those fantasies, anxieties, and defenses that are exhibited in play and in the child's transference to the therapist. Interpretations are

made through play first, and in those instances in which play becomes inhibited, the causes of inhibition are interpreted. In contrast to that approach, the techniques developed by Anna Freud (1979) rely heavily on the analysis of the child's various reactions, the strength of the child's aggressive impulses or sympathies, and his or her attitudes toward things and people represented by toys.

Recent Developments

Although play therapy continues to be a major mode of individual psychoanalytic treatment of children, more recently, and in large part as a result of Fraiberg's influential work with mothers and their infants (Fraiberg, Adelson, & Shapiro, 1980; Fraiberg, Lieberman, Pekarsky, & Pawl, 1981), there has been a proliferation of psychoanalytically oriented treatment programs for mother–child dyads (see Greenspan & Weider, 1984; Loewald, 1985; Stern, 1985). Fraiberg's modification and expansion of psychoanalytic techniques resulted in an intervention that, in addition to psychotherapy, provided home visits, developmental guidance, and supportive services to impoverished and disturbed women and their psychiatrically at-risk infants. Split-off affects associated with these women's painful early experiences were activated in the transference and worked through in the therapeutic relationship. As a result, these mothers no longer needed to ward off emotional attunement with their infant's experience. Developmental and psychiatric fixations in their infants abated, and progression toward mental well-being and normative development ensued. Although some of the subsequent analytically oriented models of dyadic intervention have provided treatment to the mother and child conjointly with liaison services provided (Musick, Clark, & Cohler, 1981), others have treated the dyad as a unit with one individual serving as primary therapist to both mother and child (Greenspan & Weider, 1984).

In order to illustrate the psychoanalytic conceptualization of and intervention with a disturbed mother–child dyad, the following vignette of a dyad treated by the author is offered.

> Sarah, a very pretty 3-year-old child with blue eyes and sparse blond hair, created an initial image of brightness and precosity. Despite that seeming precosity, a shallowness underlying her verbalizations was quickly evident. Expressions of positive affect appeared superficial, lacking depth and meaning. Sarah was often sharply negative toward adults, ordering with emotional vehemence, "You shut up, you get out of here, I hate you." During her play and interactions with others, Sarah could be seen secretly pulling out strands of her hair and injesting them clandestinely from a hidden space between her thumb and forefinger. In play activities, Sarah was poorly coordinated and markedly destructive; play was undirected and obsessive with an absence of theme or development. At the imposition of any limit, Sarah would fall to the floor, bang her head wildly, and pull her hair out in handfuls as she slapped both herself or others who tried to calm her. Sarah did not tolerate being separated from her mother.
>
> Although Sarah's mother appeared genuinely concerned for her daughter, her efforts to be involved with Sarah's play were intrusive and designed to exhibit Sarah's capabilities. She did not appear adaptive or resourceful in providing the emotional nurturance and structuring which her daughter needed. Nonetheless, she did exhibit the capacity for empathy and a strong motivation to help Sarah.
>
> Sarah had a history of emotional lability and symbiosis with her mother. Born following a preceding still birth and a problematic pregnancy, Sarah had since birth spent all or most of her time with her mother. For this dyad, what would have been the normal period of "preoccupation with the infant" continued. Sarah's relationship with her mother was characterized by her mother's indulgence, Sarah's emotional intensity, an absence of clear boundaries between Sarah and her mother, and a destructive form of affective attunement in which Ms. M. "took over" Sarah's experience. Both head banging and pica began when Sarah was old enough to move away from her mother and may be viewed as manifestations of Sarah's efforts to establish a "body self" or calm herself.
>
> Psychodynamic interventions focused first on developing therapeutic relationships with both Ms. M. and Sarah. Efforts were made to stabilize Sarah's affect through empathic responses to her actions and mirroring of her affective expressions during play therapy. Over time, those interventions enabled Sarah to begin to express her feelings rather than act them out in an injurious manner. Interpretation of Sarah's actions eventually led to Sarah's verbalization of some of her fears and frightening feelings (death and abandonment).
>
> Maternal interventions focused on empowering Ms. M.'s ego through the use of the therapeutic relationship and the therapist's empathy for her current situation and life experiences. Ms. M.'s increased awareness and the working through of those experiences and fears which had prevented her from treating Sarah as separate from herself created a more relaxed and enjoyable tone within the dyad.

In the work with the dyad presented, the use of the therapeutic alliance, the analytic techniques of working through, and analytic play techniques facilitated a less conflicted and symbiotic parent–child relationship. Although Sarah's temperament may continue to be difficult, resolution of individual conflicts that interacted with her dispositional tendencies allowed a healthy separation between Sarah and her mother and fostered reductions

in both the child's uncontrollable emotional outbursts and injurious behavior toward herself and others.

Treatment of Adolescents

Consonant with the developmental view of pathology, the psychoanalytic treatment of psychopathology in adolescents focuses on those issues that are most incumbent in the developmental tasks of adolescence *per se*. These tasks include identity, the change in a person's attitude toward his or her body, and the dynamic and conflictual anxiety activating libidinal reorganization and repression. Capitalizing on the flux of the adolescent process, therapeutic interventions at this developmental stage may be particularly effective in the treatment of characteriological disturbance associated with the more severe forms of developmental psychopathology (depression, substance abuse, antisocial and conduct disorders) (Blos, 1962). In some ways, the dynamic and economic conditions of prolonged adolescence are opportune to the frequent therapeutic sessions and long-term treatment associated with psychoanalysis.

As suggested, the primary goal of psychoanalysis or psychoanalytic psychotherapy with adolescents is the successful resolve of the conflict of adolescence. With a central and dominant function of the establishment of a final sexual organization (Laufer, 1976), fantasies, daydreams, and feelings that are associated with this developmental stage typically involve the adolescent's body and his or her experience as the owner of that body. Pathological symptomatology is viewed against that developmental challenge. The focus of therapy becomes the adolescent's attainment of a consolidated picture of himself or herself as a sexual being with dependency needs integrated with genital sexuality and the needs for assertive, protective, and intimate love.

Family Therapy

Since its inception as a defined and discrete form of treatment in the decade between the 1950s and the 1960s, family therapy has grown into a major subspecialty within the mental health profession. The emergence of family therapy may be viewed as an outgrowth of several independent and notable research projects concerned with schizophrenia. Those projects included the work of Bowen (Bowen, Dysinger, Brodey & Basamanie, 1957) and Wynne (Wynne, Ryckoff, Day, & Hirsch, 1958) at the National Institute of Mental Health, and Bateson and Jackson (Bateson, Jackson, Haley, & Weakland, 1956) in Palo Alto. Each of these projects initiated the treatment of entire families as a means of achieving greater understanding and new leverage by focusing on the family of the schizophrenic individual.

One of the most important directions ensuing from the early work with families of schizophrenics was the observation of what has come to be known as the *double bind* hypothesis of the etiology of schizophrenia. According to this hypothesis, the schizophrenic patient is born out of continual contradictory communication toward the child in which acknowledgment of the contradictions contained in the communication is not permitted. An important feature of this conceptualization has been the cohesiveness with which therapists could look beyond the behavioral dysfunction of an individual to the influence of the family and other components of the social context (Tittler, 1985). Although the notion of schizophrenogenic families subsequently has been shown to lack empirical substantiation (Frank, 1965), the outcomes of early work with families of schizophrenics have had lasting consequences for family therapy.

Central Concepts

Since its origin, family therapy has branched into diversified schools that afford varied approaches to the treatment of families. Despite their theoretical and practical differences, the various approaches to family therapy share an ideological and therapeutic commitment to the treatment of families and a loyalty to the entire family system. From the family therapy perspective, symptomatology in any member of the family is viewed as an outward manifestation of family dysfunction. As such, any particular symptomatology is analyzed not only in terms of family interactions that may have precipitated the onset of disturbed behavior, but also in terms of the contextual and intrafamily aspects of family functioning that may limit or amplify the existing problem. That is, the treatment of emotional illness of an "identified patient" is conceptualized against the larger kinship background and social milieu of the family. From that expanded view, a dysfunctional family is one in which members are unable to attain the desired closeness, self-expression, and meaning.

A central contribution of the family systems

perspective to the understanding of the psychological disorders in children has been the identification of the child-focused family (Tittler, 1985). In such families, the constant focus on the child serves to fix and accentuate the child's problems. Moreover, the preoccupation with the child dilutes the marital system, and the relationship between the parents becomes confused with each parent's relationship with the child. That confusion results in the parents' loss of perspective and good judgment regarding the guidance of the child.

The needs and temporal crises associated with the emotional transitions from one *life stage* to another and the influences of *ethnicity* on family functioning and values are important in the practice of family therapy (McGoldrick, Pearce, & Giordano, 1982). Similarly, the notion that early life experiences within the *family of origin* play a decisive role in the formation of personality and affect later behavior has received widespread support from family research workers (Fish & Biller, 1973; Rabkin, 1965). As expressed by Rapoport and Rapoport (1975), over time, the interplay of demands and the simultaneous ability of families to provide the appropriate resources will govern the well-being of family members, shape their interactions with their social context, and play a crucial role in the lives of family functioning.

Recently, family therapy has been criticized for its overemphasis on an interpersonal family process and reliance on anecdotal case histories and methodologically flawed research studies (Johnson, 1986). Nonetheless, family therapy researchers have related change in the family's general life situation and marital conflict to problem behaviors in children at home and in school (Safer, 1968).

Several additional concepts are central to the underlying theory and practice of family therapy. The concept of *openness* is used to convey the interconnectedness and interdependence of parts within the family system as well as the stability and manner of relatedness that characterizes the family system. *Wholeness,* when used in relation to the family system, refers not only to the sum of the parts taken separately, but also to the interactions among them. The concept of *equifinality* of structures of the family system implies that not only are transactions open in the present, but, more importantly, that interventions or alterations, which are made in the present functioning of the system, may have lasting effects. That is, in distinct contrast to more psychodynamic conceptualizations of pathological symptomatology, family therapy theorists are not governed by what may be referred to as the "genetic fallacy" of the past. Although past origins of symptoms and problems are not denied from this particular perspective, the primary emphasis is placed on those conditions, relationships, or alliances that maintain the symptomatology in the present. From a family therapy perspective, the system has no memory, "conscious or unconscious." The family therapist is interested in *what* is transpiring rather than *why.*

Schools of Family Therapy

An array of different models of family therapy abound; however, the four major approaches to family therapy are Family Systems, Object-Relational, Structural Family Therapy, and Strategic Intervention schools (Foley, 1974). Although each of these schools uses real-time interventions and treats the family as a unit, differences in their underlying philosophies and theoretical orientations have consequences for both the conceptualization and treatment of psychopathology.

The *Family Systems School* is a dominant form of family therapy. Heavily influenced by Murray Bowen, this school places primary importance on differentiation of the self within the family, the generational analysis of relationships, and the value of responding rather than reacting to the family system. Central to this approach is the importance of clarifying relational influences from past generations and the larger "family field." From this perspective, pathology is conceptualized as a result of family dysfunction, fused or cutoff relationships, or the over- or underfunctioning of individuals within the family.

The concept of triangles, a central tenet of family therapy, originated with Bowen (1976) in efforts to label the tendency for two-person systems (or emotional networks) to move toward three-person systems in order to reduce the emotional intensity of the system. According to Bowen, triangles are activated by stress and anxiety. Family systems therapy may strive toward family members' attainment of a less anxious, more flexible manner of relating with other family members of the triangle. Additional goals of family systems therapy include individual family member's increased differentiation of the self and decreased vulnerability among those children who become heirs to a disproportionately large shares of the family fusion or unresolved emotional attachment.

The *Object-Relational School* is most resonant with psychoanalytic theory through its heavy emphasis on "objects" (people) in the patient's own family of origin and hypotheses regarding difficul-

ties that appear to be carried over to the new family. The identified patient is often viewed as the carrier of the unacceptable or "split-off" impulses of other family members. According to this particular school, family pathology is seen as "a specialized multi-person organization of shared fantasies and complementary need gratification patterns, maintained for the purpose of handling past object loss experience" (Boszormenyi-Nagy, 1965, p.310).

Structural Family Therapy, associated with Salvador Minuchin (Minuchin, 1974; Minuchin, Montalvo, Guerney, Rosman, & Schumer, 1967), places emphasis on the power structure of the family and conveys organization of the family system through subsystems (the sibling subsystem, marital subsystem, or parent–child subsystem) and the use of boundaries. The concepts of "alignments and splits," characteristics of the structural school, were introduced into family therapy by Lyman Wynne in the early 1960s. From this perspective, psychopathology is viewed as a result of faulty structure, disengagement or enmeshments, weak generational boundaries or disrupted hierarchies, and dysfunctional alliances and splits within the family. Through joining with family members, enactment, physical restructuring, unbalancing, and challenging, the family therapist works to achieve the treatment goal of restructuring the family to facilitate symptom removal.

The *Strategic Intervention School* is perhaps most distinct from other schools of family therapy. Originating from the thinking of Don Jackson, the problem-focused approach to treatment that is espoused by this particular school reflects the beliefs that (1) the symptom is the problem, (2) such problems are caused by faulty life adjustments, (3) problems continue because attempted alterations only intensify the problem, and (4) the cure, paradoxically, is often found in intensifying the problem (Weakland, Fisch, Watzlawick, & Bodin, 1974). The use of paradox and "prescribing the problem," though not indigenous only to this particular school, are strategies that are used to induce people (within families) to behave differently.

Approach to Childhood Psychopathology

The treatment of childhood psychopathology by a family therapist focuses on the organization and structure of the family (Foley, 1974). Because psychopathology is conceptualized as the overt expression of the incorrect organization of the system *per se,* the family therapist intervenes to facilitate change within the family system. Interventions are designed to increase familial adaptation and equilibrium in lieu of family dysfunction and/or stability based on symptom formation within the family. Change in the disturbed individual(s) upsets the "pathological equilibrium" and thereby necessitates change throughout the system. Likewise, changes in relationships, alliances, and systems outside of the individual can bring about relief in an individual and/or facilitate adaptive functioning. Although the outcome may change in a particular person, that change is achieved through modifying the structure of family relationships.

The particular form of family therapy provided by any family therapist in the treatment of various forms of psychopathology varies as a result of (1) the theoretical stance of the therapist, (2) the therapist's personal style of relating, and (3) the type of family being treated. Often therapy is a "drama" between the therapist and the family. Family sculpting, prescribing the symptom, reframing problematic behavior or symptoms, making assignments designed to empower parents or facilitate individuation may be used to bring about changes in the family alignments.

The following vignette exemplifies the conceptualization of disturbance from the family therapy perspective and the implementation of interventions to ameliorate the underlying relationship patterns that maintained family dysfunction.

> Cindy K. was referred for therapy because she suffered from chronic constipation, refused to comply with medication schedules, and exhibited severe temper tantrums. When Ms. K. was confronted by the referring pediatrician regarding why her daughter did not take her medication, she replied, "She's eight years old, what can I do?" At the time of the referral, Cindy was very unruly and in many ways maintained a position of authority within the family. She became violently angry when Ms. K. interfered with the way she felt life "should be." Her frequent temper tantrums resulted in her retreat to her room to be "rescued" by her mother. Cindy "bossed" both her friends and younger sister and had difficulties in maintaining friendships. Ms. K., the youngest of five daughters born to her Jewish parents, was the only one of her sisters who could have children. Ms. K.'s relationship to her husband, an Irish man ten years her senior, was characterized by a lack of closeness and open arguments regarding how their children should be raised. As a parent, Mr. K. was quite uninvolved. His relationship with his only brother was extremely distanced, and he had not had contact with his mother for six years.
>
> From a family therapy perspective, the K. family was characterized by the enmeshed relationship between Ms. K. and Cindy, the lack of boundaries between the parent subsystem and the child subsystem, and the dominating child focus of the family. Moreover, Ms. K. appeared to function as overresponsible in contrast to Mr. K.'s underresponsibility as a parent. The absence of

closeness in the marital subsystem was maintained by the child focus of the family: Ms. K.'s preoccupation with her children, and Mr. K.'s distance stance within the family.

Primary interventions focused on the dissolution of the fused relationship between Ms. K. and Cindy and the establishment of boundaries between the parental and child subsystems. Confrontation of the role of Cindy's interpersonal difficulties and physical complaints in maintaining Mr. and Ms. K.'s distanced marital relationship focused Mr. and Ms. K.'s attention on the state of their marriage and led to their initiation of steps to improve their relation to one another. Coaching of Ms. K. in ways of setting limits and following through helped to establish boundaries between the child subsystem and the parental subsystem. Framing of Ms. K.'s overresponsibility as a means through which she could be the "perfect parent" and thereby, the "perfect daughter" helped Ms. K. to take steps to become more differentiated from her family of origin. Mr. K. was encouraged to reestablish contact with his own family of origin. At the end of several months of treatment of this family, Cindy reported fewer conflicts with peers and decreases in gastric problems. Over time, Ms. K. reported less difficulty with both of her children, a decrease in Cindy's temper tantrums, and an increased ability to function as a parent rather than allow Cindy to have the major voice in the family.

As indicated in the above vignette, the heart and goal of family therapy is change of behavior. Insight, feelings, cognitions, and understanding how things evolved to the point of dysfunction are not important or relevant to the family therapeutic process. Although family history is important, it is important only to the understanding of the genealogy of the family and the identification of patterns, relationships, and functions of individuals.

As shown in the example provided, direct interventions often involve "coaching" individual family members in specific ways of behaving or intervening with either the nuclear family or kinship network. In stark contrast to the stance of therapists practicing from other theoretical perspectives, the practicing family therapist is *directive* in his or her endeavors to bring about change in individuals.

Community Psychology

Despite the frequent use of the term *systems-oriented family therapy,* in general, family systems theorists treat the family as a closed system. That is, the larger social, economic, and organizational suprasystems in which the family is submerged are omitted in the therapeutic process. Those larger, macroscopic worlds in which the individual resides are the domain of community psychology.

The practice of community psychology is readily distinguished from the psychodynamic or family therapy approach to treatment by its underlying values, field of inquiry, and applied procedures. In addition to those distinguishing features, the philosophy and historical antecedents on which the theory of community psychology rests differ from the histories and philosophies of treatments that are associated with psychodynamic and family therapies.

Underlying Theory

Over time, society's definition of psychological problems has expanded to include antisocial behavior, ineffective personal relationships, unhappiness, and waste of talent. The ensuing emphasis on the individual's social well-being and personal development is qualitatively different from the previous emphasis on symptom formation and character neuroses (Zax & Cowen, 1976). That panoramic and extended view of mental health and the concomitant interest in an individual's life space and the social institutions that shape human development are at the heart of community psychology (Cowen, 1985).

Of the three approaches to the treatment of childhood psychopathology discussed in this chapter, community psychology is most sensitive to the larger social, cultural, environmental, and political systems in the prevention of psychological disturbance and the promotion of mental health. In distinct contrast to both family and psychoanalytic approaches to psychopathology, community psychology reflects a strong underlying endorsement of the view that deviant behavior is *culture* and *value* bound. That is, from this perspective, deviant behavior is not viewed as an entity meaningful in and of itself; rather, deviant behavior becomes meaningful only in relation to the culture in which it occurs (Cook, Howe, & Holliday, 1985). Ancillary to this concept is the view that positive mental health and social competence are also culture bound.

Community psychology has been strongly influenced by the action research concepts of Lewin (1946) and Collier (1945) along with the conceptualization of person–environment transactions provided by Kelly (1966). The emphasis on both the environment and the individual, including the individual's style of coping and tendencies toward action versus adaptation, reflected in these theoretical contributions is central to the practice of community psychology. In addition, the community mental health revolution (Hobbs, 1964), with its

commitment to the identification of more effective ways of reaching more people in need as well as its recognition of the ineffectual approach to overcoming mental disease by only treating its victims, has fostered attention to the alternative to promote psychological health and prevent maladjustment. That latter recognition serves as a cornerstone to primary prevention in mental health.

Central Concepts

Community psychology concepts that are central to conceptualization and intervention include "person–environment fit," prevention, consultation, ecological and environmental influences, and phenomenological attitudes. The concept of *person–environment fit* is a major and guiding principle of community psychology. According to this principle, emotional disturbance cannot be identified solely on the basis of individual intrapsychic indicators; rather, the person's adaptation to a particular environment is dependent on characteristics, demands, and features of the environment in interaction with the child. The person–environment focus of community psychology is best exemplified by conceptualization of emotional disturbance as maladaptation between the person and the environment, rather than the mere presence of individual intrapsychic indicators.

Primary prevention in mental health has as its core the dual goals of promoting psychological health and preventing psychological dysfunction. This proactive stance is easily distinguished from the preceding secular efforts to reform hospital care and treatment (a movement inaugurated by Phillippe Pinel in the late eighteenth century) and the psychodynamic revolution (as personified by the efforts of Sigmund Freud), which focused on the treatment of conditions which we now call neuroses. Currently, primary prevention programs, as part of mental health education, are focused toward helping people to acquire knowledge, attitudes, and behavior patterns that foster and maintain psychological well-being.

The twinned primary preventative goals of promoting health while forestalling psychological problems are reflected in psychologically oriented community programs. Such programs are (1) mass or group oriented, (2) directed toward "well" individuals and those who are at-risk for adverse psychological outcomes, and (3) reflective of a knowledge basis which suggests that a program's operations hold promise for strengthening psychological health or reducing psychological maladjustment. Examples of such programs include interventions to strengthen social support (Felner, Ginter, & Primavera, 1982), expressive and modeling procedures, as well as stress inoculation training (Graziano, DeGiovanni, & Garcia, 1979) and modifications in classrooms and social settings (Trickett & Moos, 1974).

Professional Role

The professional efforts of community psychologists are focused on each of the varied levels of interaction between the individual and his or her environment. The role of the clinical psychologist includes community action and advocacy; the development and implementation of group, agency, institution, and community interventions; tertiary prevention approaches; and enhancement of competence and stress resistance building. Recently, Heller and Monahan (1977) have advocated the expansion of the professional role of psychologists to include ecologically oriented case consultant, program planner/administrator/educator, and representative for some social system. Community psychology's open value for cultural relativity and diversity as well as its strong commitment to providing services to the disadvantaged, the disempowered, and the disenfranchised are manifest in the role extensions associated with the practice of this particular form of psychology.

Intervention Model

A compelling community psychology intervention model for clinical child psychologists offered by Cook and her colleagues (Cook, Howe, & Holliday, 1985) provides levels of analysis for viewing psychological intervention. In general, the model depicts a series of progressive interventions emanating from an initial social contract negotiated at the individual (or child) level. At that primary level, assessments and interventions with the child may most resemble other psychotherapeutic models of child treatment. However, a distinguishing feature of this particular approach is the emphasis on the child as an active social agent and active learner in various social environments. Services at the primary level of intervention may not only involve the analysis of the child's resourcefulness and capabilities, but also interventions designed to facilitate skills and strategies to be used in confronting varied social transactions (Krasner & Rubin, 1981). A distinguishing point of the initial assessment conducted from a community psychology perspective

is the inclusion of a dominant focus on the child's transactions with a range of systems.

The second level of intervention involves social contracts within those naturally occurring small social groups that both provide the most immediate social systems for the child and most directly affect the child's development. Primary interventions at this level are focused toward the child's family, classroom, and treatment unit. At this level, classroom competency training, based on core competencies for various age groups and programs designed to teach cognitive and interpersonal skills, may be used to promote adjustment and deter the development of problems. Parent guides to teaching decision-making skills to their children (Clabby & Elias, 1986) are additional means of promoting adaptable environmental functioning and preventing psychopathology through effective mastery of interpersonal difficulties, the attainment of personal goals, and the facilitation of positive self-esteem.

Interventions at the second level may be followed by interventions at the system–system level (including family–school transactions, agency–agency transactions, and unit–unit transactions within the school system). A final level is the sociopolitical level, which may involve community, states, and federal government.

Consultation

A major tool in achieving the goals of intervention at the various levels and particularly those beyond the primary level is consultation. Consultation refers to an approach to social change through improvement of existing community organizations and institutions. The goals of consultation are modification, renewal, and improvement through the delivery of additional information and professional input to improve the knowledge and skills of primary care professionals. The major premises of the consultative model are (1) that primary care-givers can develop psychologically sound programs and can learn to understand and respond effectively to the social and emotional problems of those individuals in their care and (2) there are very few problems in life that are exclusively psychological and that require the intervention of a psychologist. Consultations are rarely client-centered, rather the recipient of consultation is usually a representative of those programs and systems with which the child interacts. Examples of consultations with regard to psychological disturbance in school-aged children may include consultations with classroom teachers, the school principal, the coach of a sports team on which the child participates, or consultation workshops for parents. Program-centered consultation may be offered to school programs and community mental health systems.

Behavioral consultation, a specific form of consultation, is used as a means of increasing the likelihood of desired behaviors occurring in the patient's natural environment. Through behavioral consultations, the principles of behavior modification are made available to those individuals who are in positions to provide positive reinforcement in the natural environment. Part and parcel of behavioral consultation is the consideration of the values, ethical problems, safeguards, and goals of the society at large.

In order to exemplify the use of range and of consultation and community-based interventions, the course of treatment for Timothy, the 9-year-old boy used to introduce this chapter, may be helpful.

> Timothy's treatment utilized a systems perspective with primary interventions focused both on Timothy's family as well as the larger emanating systems. Interventions were designed to provide a less harsh and critical atmosphere. Consultation to the parents in ways they could provide empathic support for Timothy and act to facilitate problem-solving and the development of a helpful relationship between Timothy and his brother gained support for Timothy and facilitated improved self-esteem.
>
> Second order interventions focused on the school system and were designed to facilitate a more appropriate placement for Timothy and an improved sense of himself as a competent and likeable child. Utilization of the Child Study Team resulted in a thorough assessment and the detection of a perceptual problem which impaired Timothy's ability to master written material. A resulting placement in a classroom for the Perceptually Impaired afforded Timothy the opportunity to interact with children who, like himself, were capable and bright but manifested learning difficulties in a prescribed area. Consultations with both classroom teachers and the school principal were used to provide some background information regarding Timothy and his current needs for successful life experiences. As a result, these professionals, particularly the school principal, began to use Timothy to perform "special tasks." Familiarity and growing friendships between Timothy and these individuals have helped Timothy to see himself as a desirable and capable child. Finally, enrollment in a summer camp designed to provide a noncompetitive atmosphere and foster growth through the mastery of tasks and cooperative activities led to new friendships for Timothy. Inservice training provided to counselors in the development of decision-making strategies facilitated more effective means of solving problems and maintaining friendships among all children enrolled in the camp. Though Timothy may never be as large as his peers, through the use of multifaceted interventions he is able to see his many assets and utilize his resourcefulness to attain those things which are important to him. Most importantly, he no longer feels he is alone and incapable.

Summary

In this chapter, the central concepts and underlying theories associated with psychoanalysis and psychoanalytic psychotherapy, family therapy, and community psychology have been presented. Familiarity with each of these alternatives to treatment is crucial to the determination of the most appropriate form of psychological intervention vis-à-vis the development of psychopathology. For the child who is at-risk for the host of psychological and social problems that are associated with living in societies characterized by high unemployment, poor nutrition, and stifled opportunity, the preventive and health-promoting efforts of community psychology offer valid and encompassing intervention approaches. For the child who has a history of psychological disturbance and currently exhibits pathological symptomatology, family therapy and psychodynamic or psychoanalytically oriented therapy are among the dominant alternatives to treatment. In their combination, the curative and preventive potentials associated with each of these treatment options hold great promise for the many children who suffer from the various forms of developmental psychopathology or the myriad problems of daily living.

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CHAPTER 37

Cognitive-Behavioral Approaches to Child Psychopathology

Present Status and Future Directions

Andrew W. Meyers and Robert Cohen

The contributions to the present volume attest to the importance and complexity of childhood psychopathology. Children's behavioral maladjustment has broad effects, not only on the child at home, in school, and in the peer group, but on family functioning, and community systems. Accurate assessment and effective treatment of childhood psychopathology should have immediate and longer term preventive health and mental health benefits for the child, the family, and the community.

Over the last two decades, behavior therapy has had a major and positive impact on the treatment of childhood psychopathology. Recent developments in behavior therapy have encouraged the inclusion of the study of the child's cognitive processes (Kazdin, 1978). This addition, away from—but at the same time encompassing—operant and classical conditioning-based models of behavior, has led to an acknowledgment of the active role of the individual in perceiving, interpreting, and understanding the world. Rather than assume that the individual responds passively to an extant environment, we now hypothesize that the individual responds constructively to a perceived world. This creates a dynamic and reciprocal interaction among the child's behavioral acts, cognitive processes including beliefs, rules, and expectations, and the environment. Each of these factors, behavioral, intrapersonal, and environmental, require our attention if the dysfunctional child is to be understood and helped (Bandura, 1985).

This mediational perspective on human functioning underlies cognitive, cognitive-behavioral, and social learning conceptualizations of childhood psychopathology (Meyers & Craighead, 1984a). In this chapter, we adopt the term *cognitive-behavioral,* and we examine this position on the assessment and treatment of children's maladjustment. We begin by outlining and illustrating the mediational perspective. Then we review the historical determinants of the model and critique it in its present form. Based on this critique, we build a cognitive-behavioral model that recognizes the contribution of a developmental perspective and incorporates the roles of affect and family and social systems. We close with the implications of such a model for the assessment and treatment of childhood psychopathology.

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A Cognitive-Behavioral Perspective

Since Bandura's (1973) early work on children's observational learning and Meichenbaum's

(1974) description of self-instruction training with children, mediational approaches to child psychopathology have flourished. Cognitive-behavioral intervention strategies for aiding learning disabled children, the mentally retarded, psychotic children, the socially isolated, and children manifesting anxiety, attention deficits, aggression, and delinquency have all received empirical support (Meyers & Craighead, 1984a). Later in this chapter we summarize our self-instruction work with children, but it is beyond the scope of the present effort to review child cognitive behavioral interventions (see Kendall & Hollon, 1979, and Meyers & Craighead, 1984a, for reviews of a broad range of relevant intervention areas). To illustrate this clinical paradigm, we present a prototypic example of the development and implementation of a cognitive-behavioral intervention program with a child clinical population.

The Think Aloud program (Camp, Blom, Hebert, & van Doorninck, 1977) was an attempt to apply a mediational model to decrease the inappropriately aggressive behavior and increase the prosocial behavior of aggressive boys. Camp and her colleagues at the University of Colorado Medical School demonstrated that young, aggressive boys showed deficient nonverbal problem-solving performance, more impulsivity, and more task-irrelevant speech when compared to nonaggressive boys (Camp, 1977). From this work, Camp's group concluded that interventions with this population must work to inhibit aggressive behavior and to guide the dysfunctional child to alternative desirable performances.

To meet these goals, Camp designed a psychoeducational training program conducted by teachers. This was done to facilitate dissemination of the program and to allow it to reach both treatment and prevention goals. The cognitive-behavioral intervention strategy for aggressive boys was delivered over approximately 40 sessions, with teachers working from prepared training manuals. The lessons began with a game called "Copy Cat," which prepared the child to imitate the teacher while he or she proceeded through the development and application of relevant problem-solving strategies. In subsequent lessons, the teacher used the Copy Cat game across a broad range of tasks to model the problem-solving approach and strategies for coping with errors.

Once presented the adult problem-solving model, the child was prompted to employ overt verbalizations to guide task performance. The child's use of overt guiding verbalizations and the participation of the teacher were gradually faded to covert levels. Finally, the child was required to apply the problem-solving strategy in a variety of contexts to promote generalization both within and beyond the training program. Toward this end, training began with simple cognitive problems, such as mazes and puzzles, and progressed to social problems and increasingly complex cognitive problems.

The initial goals of the Think Aloud problem-solving model included enabling the aggressive child to inhibit first responses to a problem situation and to develop an overarching, organized approach to problem solving. The program was then directed toward improving the child's understanding of the concepts of cause and effect and increasing his or her repertoire of alternative response solutions and evaluation skills. The child was then prepared to apply these skills to cognitive/impersonal problems and to social/interpersonal problems (Camp & Ray, 1984).

The application of the Think Aloud program to the problems of aggressive boys has received several experimentally controlled evaluations (Camp, 1977; 1980; Camp & Bash, 1981). Typically, measures of the child's cognitive change and teacher ratings of child behavior have been taken. On occasion, observation of the child's classroom behavior was also included. A summary of these studies (Camp & Ray, 1984) suggested that the Think Aloud program produced more desirable cognitive change and, on cognitive problem-solving measures, was more likely to return aggressive boys to normal levels than no treatment control groups or attention placebo groups. Teacher ratings of child behavior indicated that Think Aloud prompted significant improvements in those ratings when compared to no treatment controls. The use of the program also seemed to be associated with less deviant aggressive behavior in class, though other forms of disruptive behavior continued to occur. Camp hypothesized that boys using the Think Aloud principles in the school situation channeled aggressive impulses into less socially disruptive acts.

Camp's Think Aloud intervention program for aggressive boys illustrates the cognitive-behavioral approach to child psychopathology. In the next section, we review the historical development of this model.

The Transition to a Cognitive-Behavioral Model

Behavioral interventions with children had their roots in Watson's work on the development

and extinction of children's fear behavior and Skinner's influential research on operant conditioning. The first clinical applications of learning theory and other research in experimental psychology in the 1950s and early 1960s were dominated by these classical and operant conditioning approaches. At that time, behavior therapy with children centered around operant interventions with autistic children, the retarded, and children with motor deficits and conduct disorders (Meyers & Craighead, 1984b). Increasing success with these child populations permitted behaviorists greater involvement with less severely disabled children in less restrictive outpatient settings. The goals of this clinical work also shifted from children's disruptive and self-injurious behavior to educational and self-management tasks that required attention to the child's cognitive skills. Work with a more intact client population forced behavior therapists to consider the child's internal thought processes both as a target of change and as a mechanism of change.

The growth of a mediationally based behavioral model was fueled not only by these social changes, but also by increasingly apparent empirical and theoretical deficiencies in behavior therapy. Although the conditioning-based model had generated a wide range of applications, Kazdin (1978) noted that, by the late 1970s, there was a growing conceptual stagnation. Advances in behavioral research had been limited largely to extensions to new populations, problems, and settings. At the applied level, the failure to find consistent generalization of behavior change to new responses and settings was extremely disappointing.

However, theorists and practitioners in behavior therapy were beginning to respond to a broader social science paradigm shift, one that Dember (1974) labeled, "the cognitive revolution." Kazdin (1978) highlighted several information-processing research areas for their influence on this shift in behavior therapy. Experimentation in semantic conditioning, symbolic self-stimulation and imagery, the role of awareness in learning, observational learning or modeling, and the impact of perceived contingencies have served to complicate traditional behavioral assumptions.

Subjects' expectancies, self-delivered instructions, or awareness of contingency relationships all affect the acquisition and extinction of behavior. Evidence of this is found in research indicating that inaccurate instructions or beliefs may exert more control over behavior than the actual response–reinforcement relationships (Dulany, 1974). Other investigators have argued that operant consequences are effective behavior change strategies to the extent that they communicate information and instructions to the subject rather than through the direct operation of reinforcement contingencies (Murray & Jacobson, 1971). Often reinforcement consequences have little or no effect on behavior until the subject is informed of the contingency relationship. Typically, such instructions by the experimenter immediately modify the subject's response to conditioning trials.

Craighead (1982) identified three factors as instrumental in the shift from conditioning-based models of behavior to a cognitive behavior therapy. These three factors are cognitive psychology and the information-processing perspective mentioned above; research in self-control; and the development of cognitive therapy. Bandura (1969), working within the context of an information-processing model, laid the cornerstone for the construction of a cognitively oriented behavior therapy with his description of modeling or observational learning. Explanations of learning by the observation of a model stress the involvement of verbal and imaginal encoding of the modeled behavior. Symbolic encodings drawn from the model can then be used by the learner to structure performance and experience.

Bandura's research on observational learning was a central component of his social learning theory (Bandura, 1977). This position assumes that an individual's behavior, cognitive processes, and the environment are involved in a reciprocal determinism, each affecting and being affected by the other two sets of variables. Although conditioning models view the individual as a passive responder to environmental effects, social learning theory assumes that the individual plays an active role in the manipulation of the environment and control of behavior.

A body of research that exemplifies the impact that Bandura's theory has had on behavior therapy is self-instruction. Meichenbaum's (1977) self-instruction training was based, in part, on the cognitive developmental psychology of Luria (1961) and Vygotsky (1962). These Soviet psychologists suggested that control of a child's behavior shifts during development from the verbal control of adults in the child's social environment, to the child's own overt speech, and finally the child's covert speech. From this position, Meichenbaum constructed a training program to teach impulsive children to control their own behavior more effectively by using self-delivered, task-guiding, first-person statements. The child first imitated an adult model who performed the target task while presenting relevant self-instructions. Eventually, the child

imitated the behavior while self-instructing aloud, whispering, and, finally, covertly rehearsing or thinking the instructions. In the past 15 years, self-instruction interventions have been used successfully with a variety of childhood problems, including hyperactivity, aggression anxiety, social competence, and academic deficiencies (Craighead, Wilcoxon-Craighead, & Meyers, 1978). We shall return to the topic of self-instruction in a later section.

The second major factor identified by Craighead as influential in the development of a cognitive behavior therapy was research in self-control. Prior to 1965, the behavioral explanation for so-called self-control behavior still relied on Skinner's (1953) operant view that external consequences determined behavior. In that year, Homme offered the notion of "coverants" or operants of the mind. Homme (1965) argued that thoughts could serve to reinforce and so modify behavior. While still flirting with an operant theme, this work broached the role of internal factors in self-directed behavior. Homme's papers were followed in the early 1970s by Kanfer's (Kanfer & Karoly, 1972) analysis of self-control. He conceptualized the self-control process as being divisible into self-monitoring, self-evaluation, and self-reinforcement components. Applications of this model to clinical problems, such as obesity, smoking, and impulsivity, have come to play a major role in contemporary behavior therapy. Although it is plausible to argue for a causal role for external factors in the self-control process, Bandura's (1977) reciprocal determinism and other systemic perspectives encourage us to acknowledge the interaction of intrapersonal cognitive activity and environmental contributions to self-directed behavior.

Craighead's (1982) third and final influence on the growth of cognitive behavior therapy was the development, in the 1960s and 1970s, of cognitive therapy. Beginning with Ellis's (1962) *Reason and Emotion in Psychotherapy,* and reinforced by Beck's (1976) writings, a body of clinical approaches and procedures developed in the clinic rather than the laboratory gained popularity. These therapies shared two basic assumptions: that psychopathology was produced by maladaptive cognitive processes and that successful treatment must modify those self-statements, expectancies, or beliefs. Research examining cognitive therapies has placed in doubt both of these basic assumptions and supported more comprehensive causal models (Zeiss, Lewinsohn, & Munoz, 1979); however, these approaches have continued to have impact on therapeutic work with children.

This brief historical overview of cognitive behavior therapy has outlined the social, theoretical, and experimental forces that have moved behavior therapy to a cognitive-mediational position. These influences have enabled child clinicians to offer a more comprehensive model of childhood behavioral disorders and, in some situations, more effective therapeutic strategies. However, the cognitive-behavioral view has not flourished without critical comment from its own adherents, and in the next section we examine some of this commentary.

Critical Issues in the Cognitive-Behavioral Model

Bandura's social learning theory and its causal model—reciprocal determinism—have strongly influenced child cognitive behavior therapy. As mentioned earlier, reciprocal determinism assumes that the child's behavior, intrapersonal variables, and environmental contexts interact to affect all factors in the interaction. Although Bandura argued for an attention to a wide range of systemic influences in the study of behavior, cognitive behaviorists have emphasized cognition to the exclusion of other person variables, and typically have conceptualized context as linear antecedent and consequent events (Craighead, Meyers, & Wilcoxson-Craighead, 1985).

Cognitive positions have traditionally assumed that emotion is a product of cognition. However, Zajonc (1980) and others have called for a new recognition of the primary role of emotion in human behavior. Rather than argue over the primacy of cognition versus affect, Craighead, Meyers, Wilcoxson-Craighead, & McHale (1983) have suggested that we be concerned with the pattern of interaction of these variables. Mahoney (1985) has gone so far as to argue that such distinctions as cognition, affect, and behavior may not be helpful, and that these constructs, as communicative interactions with our world, would better be subjugated to notions of personal order and personal meaning. Clearly, a comprehensive cognitive-behavioral model must encompass the child's emotional responses.

A second person variable that is crucial to an understanding of children's behavior, but one that has received surprisingly little attention in the cognitive-behavioral literature, is the child's developmental status. Without an awareness of normal developmental sequences, it is difficult to assess the appropriateness of children's behavior or their response to therapeutic interventions (Meyers & Co-

hen, 1982). The analysis of cognitive functioning in children within the field of child clinical psychology has been dominated by adult-derived, nondevelopmental models (Achenbach, 1978; Cohen & Meyers, 1984). That is, there is an assumed quantitative continuity between childhood conditions (behavior), and adult psychopathology. Analyses of childhood problems and of the etiology of adult problems, then, conceptualize the child's state as deficient—that is, in terms of adult states minus some level of sophistication—and often use age of child as an index of functioning. An alternative approach to these issues, termed here a developmental perspective, is to view the child as progressing through qualitatively distinct stages of development. At each stage, the child has a complete system for interacting within his or her environment, with each stage building upon the accomplishments of previous stages but not simply reducible to the previous stage. Rather than considering the child deficient, the child's functioning is considered different from adult states. This approach is *not* a denial of a continuity in a pathological process; rather it is an assertion that this process may be exhibited in different forms at different developmental periods.

The significance of developmental variables is apparent when one finds age-dependent child performance on tests of conceptual style and concept learning, response to instructions and models, use of memory rehearsal and hypothesis-testing strategies, and measures of planfulness (Craighead *et al.*, 1983). Even more interesting, as we shall discuss at length later, is evidence that same-age children at different Piagetian cognitive levels manifest discriminative cognitive task performance. This research illuminates the contrast between a "deficit" model and a "developmental" model of child behavior.

Finally, we turn to environmental variables. Behavior therapy has concentrated traditionally on immediate antecedent and consequent events. Obversely, contemporary models of child development (Bronfenbrenner, 1979) and child psychopathology (Henggeler, 1982) have stressed an integration of wide-ranging systems influences. These latter perspectives encompass reinforcement contingencies but view the child as embedded in and interacting reciprocally with several social and familial systems (e.g., family dyads, the school, socioeconomic conditions). Although these interactional perspectives have recently had impact on behavior therapy (Glenwick & Jason, 1984; Turkewitz, 1984), an increased awareness of systemic influences would benefit the child cognitive-behavioral model. For example Panella, Cooper, and Henggeler (1982) found that teenagers who receive consistently supportive discipline from parents or guardians are more resistant to the influences of delinquent peers.

That such broad environmental models mesh well with cognitive positions can be seen in both cognitive and developmental perspectives that argue for an inseparability of cognition and action. Piaget's concepts of accommodation and assimilation tied the child's cognitive development to interaction with the environment (Flavell, 1963). Weimer (1977), in a comment on "motor theories" of cognition, stated that cognition must be understood "from the outside inward." In a recent evaluation of cognitive theories of depression, Coyne (1982) argued against assigning causality to cognitive variables on the grounds that depressed adults were both perceiving and creating depressing environments.

Whether we choose to focus on the systemic interaction of the child's behavior, cognitive and emotional processes, and environment, or the inherent inseparability of these constructs, recognition of a more comprehensive mediational model should present us with a framework for greater understanding of child behavior. In the following section, we take advantage of this framework to build a developmental and systems-based child cognitive-behavioral perspective.

A Developmental Perspective for Cognitive Behavior Therapy

Over the past seven years, we have studied the role of individual differences in cognitive functioning for the outcome of self-instruction interventions. Although self-instruction training has produced significant training task improvements for a variety of childhood problems as noted above, prior to our work, the generalization effects from training were not particularly noteworthy (Meichenbaum & Asarnow, 1979).

From our developmental perspective, rather than using age of child as an indicator of developmental functioning, we selected same-aged children who differed in Piagetian-defined level of cognitive development. Specifically, we studied preoperational and concrete operational children as determined by performance on two conservation tasks: conservation of number and conservation of continuous quantity (See Flavell, 1963). The dominant problem-solving mode of preoperational chil-

dren is perceptual, thus there is little integration of past experiences with present circumstances, and physical consequences are considered over social norms. Concrete operational children assume exact answers to problems, are aware of irrelevant variants in problems, and understand and are responsive to the social demands of others. In short, preoperational children live in a here-and-now reality, influenced by their perceptions of their immediate environment. Their thought is characterized as often being egocentric, that is, being unable to take others' perspectives. The thinking of concrete operational children is not tied to their immediate perceptions. These children can integrate past with present experiences, and are not misled by state-changing transformations, such as rolling out a ball of clay, when making judgments about invariant properties such as quantity. It is important to reiterate that we selected children who varied in cognitive level while matching them on chronological age.

For this research, we adapted a model of learning context from Bransford (1979). A full explication of this model may be found in Cohen and Meyers (1984). We conceptualized the outcome of any learning situation to be the product of a complex interaction among four factors: cognitive status of the learner, nature of learning materials, procedures used during learning, and criterial tasks for assessing learning, maintenance, and generalization. We tested this model in a series of studies, defining cognitive status of the learner in terms of Piagetian stage membership, and used the Matching Familiar Figures Test (MFFT) (Kagan, Rossman, Day, Albert, & Phillips, 1964) as a training task and a perceptual perspective-taking task as a measure of generalization. In each of the studies, children were assessed for Piagetian level and then served in two experimental sessions. In Session 1, the child was pretested on the MFFT and perspective-taking tasks and received self-instruction training according to assigned condition. In the Session 2, held 1 to 3 days after the first session, the child received a second exposure to the self-instruction regimen followed by a posttest on the two tasks.

In our first study (Schleser, Meyers, & Cohen, 1981), we manipulated the nature of materials component of the learning context by providing the child with either a self-instruction package designed specifically for use with the MFFT or a general problem-solving package which was applicable to a range of problem-solving situations. Children performed better on the MFFT training task following exposure to the specific content instructions, but, importantly, only those children exposed to the general content instructions showed significant generalization effects. Although concrete operational children outperformed preoperational children overall (i.e., a main effect for cognitive level on both tasks), there were no differential training effects as a function of cognitive level in this first experiment.

Keeping nature of materials constant in our second experiment, using only the specific content instructions, we varied the learning procedures (Schleser, Cohen, Meyers, & Rodick, 1984). One group of children received the traditional fading of instructions procedures, whereby they rehearsed the self-guiding statements through a series of progressively less elaborated prompts. Another group of children was led to "discover" the same set of self-guiding statements through a Socratic dialogue procedure with the experimenter. Again, both preoperational and concrete operational children benefited from training in the same fashion, with both the fading and the directed discovery groups outperforming the control groups. Importantly, only the concrete operational children in the directed discovery group significantly improved on the generalization task following training.

Given that both cognitive level groups demonstrated significant generalization from a general content, fading procedure, whereas only the concrete operational children generalized from a specific content, directed discovery procedure, the logical next study was to assess the effects of fading versus directed discovery when the content of the instructions was general in nature (Nichol, Cohen, Meyers, & Schleser, 1982). As in the second experiment, only the concrete operational children demonstrated significant generalization.

This set of studies, along with our other research on self-instructions (Brown, Meyers, & Cohen, 1984; Cohen, Schleser, & Meyers, 1981; Goodnight, Cohen, & Meyers, 1984; Meyers & Cohen, 1984; Schleser, Meyers, Cohen, & Thackwray, 1983; Thackwray, Meyers, Schleser, & Cohen, 1985) led us to propose the notion of optimal discrepancy (Cohen & Meyers, 1984). Piaget suggested that cognitive growth is best facilitated under conditions of moderate disequilibrium, that is, conditions which are new to the individual, thus requiring accommodation, yet not so novel as to be devoid of meaning. Extending this notion to generalization from training, by *optimal discrepancy* we mean that generalization is best facilitated when the cognitive involvement of the individual is maximally captured. Involvement will be determined

by the cognitive demands of the intervention setting and thus may be manipulated by the materials and procedures used, but will ultimately be determined by the cognitive status of the child. For the problem-solving situations used in our work, preoperational children are optimally involved when the learning procedures are kept simple and the materials used require cognitive integration and effort, as in the case of the general content instructions. When the procedures are difficult, as is the case with directed discovery, the preoperational child is overloaded in a cognitive sense. The child can perform the discovery operations (and in fact shows training effects as a result); but this child is unable to extract a strategy that can be carried into other situations from this overwhelming procedure.

The concrete operational child is optimally engaged by the directed discovery procedure, whether the content of the strategy is specific or general in nature. The procedure puts on display for this child a general plan not only for how to solve a problem, but how to go about solving problems. The concrete operational child can understand this strategy generation procedure (separating form from content), whereas the preoperational child cannot.

We were interested in applying what we had learned from the above studies in an efficient manner to large groups of children. To this end, all first and second graders at a public elementary school were involved in a study in which self-instructions were delivered in the class and/or at home (Cohen, Nichol, Cohen, & Meyers, 1985). Specifically, children served in four experimental conditions: classroom training, parent consultation training, classroom plus parent consultation training, no training control group. Each child was individually pre- and posttested on measures of cognitive ability (e.g., Piagetian level, cognitive tempo [MFFT], Tower of Hanoi, metacognition, tests devised to assess abilities in following directions, self-monitoring, and coping with errors, and tests of math skills), sociometric measures, perceptions of self-control, and locus of control. In addition, teachers and parents gave sociometric ratings of the child and perceptions of the child's self-control.

Training consisted of 8 weeks of biweekly lessons. The classroom self-instruction regimen was embedded within regularly scheduled math lessons and was delivered by a female graduate student. Problem identification, planfullness, self-monitoring, and coping with errors were taught in the first 4 weeks (one component per week, cumulatively rehearsed). During the last 4 weeks these components were further integrated, and the children were encouraged to apply their "plans" to both academic and social situations. The format and materials for the parent consultation groups directly paralleled those of the classroom groups with home visits made to help the parents before the first week of training and prior to the fifth week.

The most pervasive finding was the advantage of concrete operational children over preoperational children across nearly all the measures (e.g., more reflective in style, more efficient on several cognitive tasks, better metacognitive awareness, better in math, more often nominated by peers and by teachers as academically competent, more internal attributions for behavior). In terms of training, the classroom presentation led to numerous improvements on the cognitive measures (e.g., Tower of Hanoi, cognitive tempo, metacognition, and math skills). In sum, delivery of self-instruction strategies in the classroom appeared to be an efficient way to improve behavior, particularly on cognitive tasks. Although the classroom approach, where more control could be exerted over the manner of delivery, was superior to delivery by parents on the cognitive tasks and on sociometric measures by peers and teachers, parents involved in the home presentations reported that their children exhibited a greater degree of self-control at home by posttest than did children in the other groups.

Before a reevaluation of our developmental perspective, consider a recently completed doctoral dissertation by Brown (1986), comparing 443 children's (6- to 14-years-old) self-report of depressive symptoms with parent and teacher ratings of the child's depressive symptoms using a 57-item questionnaire. Discriminant analyses were performed using the factor analytically derived dimensions from each set of raters to discriminate Piagetian stage membership (preoperational, transitional, concrete operational). From the parents' scale, symptoms associated with school performance, social interaction, and somatic complaints were significantly higher for transitional children; symptoms associated with alienation were positively related to cognitive status (preoperational children were low and concrete operational children were high); symptoms associated with anxiety were negatively associated with cognitive status (preoperational children were high and concrete operational children were low); and symptoms associated with withdrawal were associated in a curvilinear fashion (i.e., lower for transitional than for preoperational or concrete operational children). Predicting chronological age, somatic complaints on the child scale decreased with age, and aggression

and self-criticism on the teacher scale increased with age. Predicting mental age, school performance and alienation on the teacher scale significantly increased with mental age. In short, developmental status of child, whether defined as Piagetian level, chronological age, or mental age, led to differential predictions of normative levels of depressive symptomatology as a function of who was rating the child's symptoms.

As related findings in Brown's project, the mean baserates for items on the depression scale varied as a function of rater, and the correlations between child–teacher and child–parent ratings, while statistically significant, were quite low (r = .22 and .28, respectively). Parent–teacher ratings were not significantly correlated (r = .12). Although some of this discrepancy in responses from raters may be due to differences in how children and different adults conceptualized the items or response alternatives, the factor analyses in this study suggest an additional explanation. For the children, the self-criticism factor accounted for a substantial amount of variance (41.3%); for parents, the prominent factors were school performance and social interaction (37.9%); for teachers, aggression was the leading factor (44.7%). Not surprisingly, children are focusing on internal factors whereas adults are concentrating on observable behavior relative to the social contexts in which they see the child.

Elaborating the Developmental Perspective

As noted, we began this research program as a collaborative effort to integrate a developmental perspective with traditional child-clinical concerns, a view we have termed a developmental-clinical perspective (Cohen & Schleser, 1984). We feel that we have adequately demonstrated the utility of such an approach, showing that an assessment of a child's extant cognitive functioning can lead to differential predictions of intervention outcome. While focusing on the cognitive level of the child, we adapted the model of learning context from Bransford as described above. This also has proven quite useful, giving us conceptual tags for understanding variations in intervention materials and procedures and their relationship to variations in cognitive functioning. What we wish to propose in this section is a further elaboration of our position, particularly in terms of examining the role of systemic influences and of affect in understanding a child's behavior.

A growing number of researchers are beginning to examine behaviors as a function of physical and social dimensions of settings. For example, environmental psychologists emphasize that the physical environment is not simply a stage for the performance of behaviors (see Cohen, 1985). Rather, there are complex individual-environment transactions whereby the physical setting in conjunction with the psychological characteristics of individuals serve to determine the nature of behavior. A related position can be found in Bandura's (1977) reciprocal determinism, which hypothesizes an interdependence among behavior, environment, and person characteristics. Family therapists are also interested in the reciprocal interactions among family members (e.g., Minuchin, 1985) and how both proximal and distal social systems impact upon an individual in a given context (e.g., Henggeler, 1982). In developmental psychology, Bronfenbrenner (1979) described the child's development as being influenced by four spheres of social exchange, from direct and immediate influences in the current environment, to more indirect influences, such as social relations not immediately present, mother–father marital relations, and cultural mores.

There is no unifying theory to date on the role of context. Yet each of these positions shares a concern for an examination of an individual's behavior in relation to relevant characteristics of the setting in which the behavior is exhibited. This is not simply a call for naturalistic research; rather, it is an acknowledgment of the complex multidimensional nature of factors that influence and are influenced by an individual.

What does this mean for a cognitive-behavioral perspective? We believe that the basic premise of these positions, that an individual's cognitive functioning can and should serve as the focus for assessment and intervention, should remain as the focal point. The influence of settings, whether that be defined in terms of physical and/or social characteristics of the environment, will be known to the individual in relation to that individual's level of cognitive functioning. What is needed is an expansion of this position to include a variety of additional variables. This expansion will be proposed below with reference to our research on self-instructions.

The case can be made that all knowledge has social origins, a position particularly emphasized by Vygotsky (1962). Parents, teachers, peers, and therapists transmit information and strategies to children through social discourse. Of course, this is

a two-way street, with the giver of the information varying the message as a function of the abilities and behaviors of the learner. In the vast majority of research on clinical interventions with children, we ignore these social exchanges; we focus on the content, or the procedures, or the tasks of interest.

Recall the research above in which we presented self-instructions to classes of children and/or had parents deliver the strategy. It is interesting that the strategy training in the classroom resulted in cognitive gains on cognitive tasks whereas the parent consultation groups led to little change in these assessments. Clearly, the parents were engaged in a different set of relationships with their child than we were in the classroom. Parents did report a change in self-control in their children. Thus, the parents may not have produced cognitive task changes in their children, but the self-instructions they presented to their children led to some change in the child's behavior at home (or at least a change in the parents' perceptions of that behavior).

Similarly, the research reported on the child, teacher, and parents as raters on a depression questionnaire led to findings of differences as a function of rater. The agenda of teachers, children, and parents are certainly not identical. The lack of relationship among the raters should not be viewed as "error variance" but should be viewed as assessments of different conceptualizations of the child's everyday contexts.

Importantly, in both these projects, the cognitive-developmental level of the child strongly influenced the findings. Even though we are expressing a belief that cognitive interventions with children must examine more fully the dimensions that relate to the contexts of interest, again we urge that this study be performed in relation to the developmental status of the child. Piagetian stage membership is an excellent predictor of behavior as shown in our research, but it may not be the best construct in other work. Individual assessments of the understanding of peer relationships or family functioning, or the child's understanding of the power and control of authority figures, may be more appropriate indices of functioning in other projects.

One aspect of individual, developmental functioning, which has received little empirical attention in the developmental, systemic expansion of cognitive-behavioral therapy with children, is the role of affect in the child's behavior. Emotional processing of stimuli may proceed at a faster rate than cognitive processing, and thus emotions may influence cognition at least as much as cognition influences emotion. There is some evidence, reported in Anooshian and Siegel (1985), that the emotional "tags" related to events and situations are particularly important for young children with less robust representation abilities than older children.

Regardless of the position one assumes on the relationship of affect and thought, the influence of affect can and should be included in analyses, assessment, and implementation of intervention strategies. We suggest that the development of affect, as is the case for cognitive development, follows a sequential qualitative pattern; that affect serves the child in communication with the environment and must be considered in an understanding of the child as a unified whole rather than as a miniature adult.

In conclusion, we remain aligned with the more traditional cognitive-behavior modification position in terms of focusing on the cognitive processes of the individual. We expand upon this basic premise by urging the consideration of the child in terms of a developmental perspective, viewing the child as an organism different rather than deficient relative to adult functioning and in terms of the context of the child's behavior. We also urge for the inclusion of the child's social and emotional status in both assessment and intervention considerations. Whether one chooses to intervene upon an individual child, a classroom, or a family from a cognitive-behavioral perspective, we argue for the consideration of both direct and indirect, both proximal and distal, sets of variables.

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