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Developmental systems and psychopathology

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Abstract
Efforts to understand the etiology of adult mental disorders by studying children has produced unanticipated changes in our understanding of pathology, individual development, and the role of social context. Among these are the blurring of the division between mental illness and mental health, the need to attend to patterns of adaptation rather than personality traits, and the powerful influences of the social world on individual development. Current developmental views place deviancy in the dynamic relation between individuals and their contexts. At another level, when we view the history of developmental psychopathology, dialectical developmental processes are evident as we trace how patterns of adaptation of researchers, expressed in theoretical models and empirical paradigms, increasingly have come to match the complexities of human mental health and illness.

The emergence of developmental psychopathology as a unique perspective on mental health and mental illness is the outcome of a dialectical transaction between attempts to understand human psychological problems and the problems themselves. After each effort to support an explanatory model by collecting a set of data, the results have required modifications in the model, forcing the field to evolve from a concern with causes and effects to an increasing appreciation of the probabilistic interchanges between dynamic individuals and dynamic contexts that comprise human behavior. Surprisingly, progress in the study of developmental psychopathology has resulted from a set of unresolvable dialectical contradictions. Some of these contradictions are inherent in the study of psychology, some in the study of development, and some unique to the study of developmental psychopathology. A basic contradiction in each of these domains is between the labels we use to divide and categorize our phenomena and the dynamic reality that comprises the phenomena themselves. Unique to the study of pathology is the contradiction between the abstracted diagnostic schemes we use for categorizing individuals and the complex dynamic processes of the individuals themselves.

Another tension we must confront is the contrast between the study of serious mental disorders and mental health. Whereas clinicians have needed to center their attention on individuals who are in the greatest therapeutic need, most developmentalists in the field have viewed the study of pathology in the few as a means for understanding the roots of mental health in the many. The dialectic here is that when we focus on mental health we discover that there are extremes on every behavioral dimension that look like pathology, and when we focus on mental illness we discover that that there are areas of competence that look like health. The study of mental disorder may be inseparable from the study of mental health, and it may be that the study of each is required for the understanding of the other (Sroufe, 1990).

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Our field is labeled with a concern for pathology—that is, disease. Here we find another important dialectical contradiction in the name "developmental psychopathology." By using a developmental approach in the study of pathology, we may find that the disease disappears when understood as one of many adaptational processes between an individual and life experiences.

The final contradiction lies in the nature–nurture dichotomy where we find that by studying the environment we obtain a better understanding of the individual and by studying the individual we obtain a better understanding of the environment. The better we understand the sources of these contradictions, the better will we be at understanding and changing the mental health of children.

Dominant Issues for the Field

The theoretical issues in developmental psychopathology can be captured in three major questions about conceptualizations of pathology, individual development, and the role of the environment.

First, how do we define pathology? Is it a qualitative or quantitative judgment? Can individuals be placed on universal dimensions, or are there qualitative distinctions to be made that place people in one category or another? Here we find the important developmental issue of continuity and discontinuity, not only between one kind of individual and another, but between the individual at one point in time and another.

Second, how do we understand individuals and their development? Is it through a search for stable characteristics either in the individual independent of context or in the context independent of the individual or is it the search for patterns of functioning in context? Moreover, do these characteristics change over time as the unfolding of some maturational pattern, or in reaction to new demands as each individual interacts with an expanding social domain?

Third, how do we conceptualize the environment? Is it a passive set of experiences which maximizes or minimizes innate individual potential as in the concept of genetic ranges of reaction or is experience transforming as it interacts and transacts with dynamic individual developmental processes?

Developmental psychopathology arose as a new orientation to the etiology of psychopathology necessitated by the failure of more customary models to explain how disorders arise and are maintained. The traditional medical model of disorder is based on the presumption that there are identifiable somatic entities that underlie definable disease syndromes. Although within psychiatry the current dominant view of disease is still strongly biomedical, there is an increasing place allowed for social and psychological factors in the etiology of mental illness that may have an important role in the initiation, maintenance, and treatment of mental disorder. Historically, individuals were not seen as integrated systems of biological, psychological, and social functioning, but rather as divided into biological and behavioral selves. If the biology changes, either through infection or cure, the behavior changes. Three problematic principles emerge from this model that are frequently applied to the study of psychopathology (Sameroff, 1995): (a) the same entity will cause the same disorder in all affected individuals, whether it be children or adults; (b) the same symptoms at different ages should be caused by the same entity; (c) specific disorders of children should lead to similar adult disorders.

Unfortunately, none of these three principles can be generalized, especially with respect to the study of psychopathology. Regarding the first principle, the same biological problem can be related to quite different behaviors in children and adults (e.g., the genetic deficit thought to underlie schizophrenia). Second, the same symptoms may be caused by quite different processes at different ages. The sadness that is a primary characteristic of adult affective disorders is a common reactive condition in childhood. Third, for many emotional and behavioral problems in childhood, there is little evidence of continuity into adulthood. Many childhood emotional problems do not persist, and there is little empirical evidence that connects adult disorders with childhood conditions. Even when continuities of symptoms are found, the

connection to underlying entities is complex. Despite the fact that modern biology has moved beyond such a model in its own domain, there is a strong residue of such thinking when applied to the unknowns of behavioral functioning.

Developmental Psychopathology

The discipline of developmental psychopathology has been promoted as the foundation for major advances in our ability to understand, treat, and prevent mental disorders (Cicchetti, 1989). One assumption underlying this expectation is that the perspectives of developmentalists and psychopathologists offer different conceptualizations of the same phenomena and that their unification would produce a clarification of the appearance and etiology of psychological disturbances. In this vein Rutter and Garmezy (1983) characterize this difference as the developmentalist's concern with *continuity* in functioning such that severe symptoms are placed on the same dimension as more normal behaviors in contrast to the pathologist's concern with *discontinuity* where the abnormal is differentiated from the normal. The division of the field into those who approach the problem from a developmental perspective and those that approach from a clinical perspective has served to mask the fact that there are many different kinds of developmentalists and many different kinds of psychopathologists. These differences arise in contrasting interpretations of behavioral development and ultimately in contrasting views of the sources of behavioral deviation.

Recent progress in the technology of molecular genetics has led to a hope that the etiology of mental disorders will soon be revealed and that their treatment and prevention will follow. For example, Koshland (1993, p. 1861), a former editor of the journal *Science,* presents an optimistic picture in which the future will be better than the past because of expected "insights into the effect on complex processes such as IQ, bad behavior, and alcoholism by single genes or chemical reactions." Although we may view this as a technological statement of fact, it can alternatively be interpreted as the expression of a particular belief system about the nature of the child and especially the nature of pathology. The basis for such linear hopes as Koshland's is a view of humans as determined by their biology and a view of development as an unfolding of predetermined lines of growth. Among these lines of development are those that produce the emotionally disturbed, such as persons with schizophrenia and depression, the cognitively disturbed, such as the learning disabled and the retarded, and the undisturbed (i.e., normal individuals).

But would this model fit those individuals who do not stay on their predicted trajectories? There have been many full-term healthy infants who were predicted to have a happy course but instead ended up with a variety of mental disorders later in life. In these cases one could argue that we have not yet developed the sophisticated diagnostic tools to identify their inherent deviancy at birth. However, how would one explain those infants who had already shown major disabilities and yet somehow did not progress to adult forms of disturbance (Sameroff & Chandler, 1975)?

The case of Helen Keller is probably the best known counterpoint to the maturational view of development (Keller, 1904). The story of this deaf and blind woman required a model of development that went beyond the maturational blueprint to incorporate the powerful effect of environments on human potential. The biographies of many individuals that were certain candidates for a life of institutionalization but whose fate was altered to a happier end have been well documented (cf. Clarke & Clarke, 1976, Garmezy, 1985).

Because the fulfillment of most of the promises of molecular biology are still in the future, there is time to examine the gap between our current scientific knowledge and the elimination of mental disease. On the one hand, we can view this gap as a technical one that will be closed by the accomplishments of empirical initiatives such as the "decade of the brain." On the other hand, one can view this gap as a conceptual one, which will continue to exist despite major advances in the biological understanding of developmental processes.

Notwithstanding vast scientific and techno-

logical advances over the last century the solution to problems at both the individual and social level seem no closer. At the same time that there are major advances in our understanding of the biological underpinnings of such disorders as cancer, the rate of cancer increases. At the same time that major advances are made in the understanding of ecological systems, the rate of environmental devastation increases. And at the same time that major advances are made in our understanding of economic processes, the rate of poverty increases.

How can one explain such contradictions? At one level the explanation may be that achievements in the laboratory are not readily translated into achievements in society at large, and that, eventually, scientific reason will prevail. At another level the explanation may be that scientific reason itself may be at fault. A belief that scientific knowledge directly changes social behavior may be akin to a reductionistic belief that the action of atoms, molecules, and genes directly change human behavior. If such scientific reasoning is at fault, a different model of human and social action that respects the complexity of both may be necessary as an alternative view.

In recent years a number of respected developmental psychologists have argued about the basic nature of the child and the correctness of our scientific models. For example, Kessen (1979, 1993), supporting the expansion of the contextualist view, proposed that the technological shifts in society are altering our scientific view of the child from an isolate that develops independent of experience to an image of the child as a continuous creation of social and biological contexts. In contrast, Scarr (1985) is able to reinterpret environmental influences from a reductionist perspective, arguing that theories that give context a major role in human development are based on underestimations of the power of genetic influences. She sees genes as not only directly influencing the characteristics of the child but also the characteristics of the child's environment.

My own view is that the appropriate model for understanding developmental psychopathology is one that matches the complexity of human behavior. Such a view is in accord with the beliefs of most of the founding voices of developmental psychopathology (Cicchetti, 1989, 1993; Cicchetti, Toth, & Maughan, 2000; Sroufe & Rutter, 1984). Lewis (2000), as well, describes the need for more complex explanatory models as he lists the inadequacies of linear models that focus on individual traits or simple environmental action. Many complexity approaches are based on general systems theory and similar attempts to integrate individual and contextual processes in dynamic ecological models. Such models may be essential for understanding the sources of health and disorder that are the central concern of developmental psychopathology. In these views if we find simplicity, it is an artifact. Although our goal should always be to find the signal in the noise, we need to make sure that it is noise and not music we have not yet learned to appreciate.

Historical influences on developmental psychopathology: High-risk studies

Developmental psychopathology has become a mainstream enterprise during the past few decades. It began as an attempt to add some developmental content to the study and treatment of child psychopathology (Achenbach, 1974) which had, and still has in many camps, its main foundation in downward extensions of adult psychopathology. These simple beginnings represented an emerging revolution. Although a life span perspective was implicit in the writings of Freud and Adolph Meyer, they were not explicit in general psychiatry. It took the high-risk movement to put the first cracks in the clay feet of traditional psychopathology. Although risk now is a field in its own right, in the late 1960s its meaning was restricted to risk for psychiatric disorder and grew out of an effort to understand the etiology of the then most diagnosed serious mental disease, schizophrenia.

Mednick and McNeil (1968) had argued that studying people with schizophrenia had not and would not illuminate the etiology of the disease because there were many things that happened to people after they got the diagnostic label that had little to do with how

they got it. For example, they now had a label which led themselves and other people to think about and treat them differently, and the course of their treatment from drugs to institutionalization had its own iatrogenic effects. Mednick argued that in order to really understand the factors directly associated with the disease, one had to study people before they got the label.

The strategic question was who to study. It was not efficient to examine everyone because only 1% of the population would become schizophrenic. The problem was to find a subsample who were more likely to get the disorder than the general public, a subsample that was at "high risk." Mednick's answer was to study the offspring of schizophrenics whose risk for the disorder was 10 to 15 times higher than the general population. This answer was adopted by the majority of the 14 or so projects that united in the Risk Research Consortium for the study of schizophrenia (Watt, Anthony, Wynne, & Rolf, 1984) under the intellectual leadership of Norman Garmezy (1974). Garmezy's career characterized the movement and evolution of the field from research wholly concerned with the roots of incompetence in a small segment of the population with serious mental disorders, to the study of the roots of competence in everyone.

There were several stages in the growth of the field that were consequences of the Risk Research Consortium. Each became a consequence of the dialectical contradiction between the question one was asking and the means one was using to answer it. In this case the question was what the etiological sequence leading to a disorder, schizophrenia, was that did not appear until late adolescence. The means to an answer required studying younger children. Previously, there was little interest in children because they were presumed either to be too immature to have serious mental health problems or, in whatever they did have, to be identical to adults with psychopathology. The surprising results of actually studying children, in contradiction to old views, was that some children did have mental health problems and these problems did not readily map onto adult categories of mental illness.

The presumption in the case of later appearing schizophrenia was that there was an underlying biological continuity of disease, the schizotype, which manifested itself differently at different ages. Children with a schizotype would have different symptoms than adults with the same schizotype who had the disorder. The psychopathologist's research strategy was to identify markers of the schizotype during earlier periods that would be correlated with later schizophrenia. A number of hypotheses had arisen as to the nature of these markers, including birth complications (Mednick & Schulsinger, 1968), particular patterns of motor tonus (Fish, 1984), attentional patterns (Nuechterlein, 1984), and eye movement patterns (Holzman, Levy, & Proctor, 1976). What is interesting about this strategy is that these markers need not be developmental links in the etiological chain, but only markers of some underlying pathogenic process that had not yet been identified.

The Rochester Longitudinal Study (RLS) that my colleagues Melvin Zax, Ronald Seifer, Ralph Barocas, Alfred and Clara Baldwin, and I have been involved in for almost 30 years (Sameroff, Seifer, & Zax, 1982; Sameroff, Seifer, Baldwin, & Baldwin, 1993) is an example of an old research model that centered on a linear analysis of the effects of parental psychopathology on child behavior. During the course of the study, however, adaptive changes were forced upon the investigators because of the lack of congruence between hypotheses and data. This dialectical process produced changes in the analytic strategy as well as the investigators' understanding of development—from a study of genetic influences on behavior to an investigation of the interaction of complex dynamic processes between individual and context. Bridging the gap between the unlimited complexity of dynamic developmental conceptualizations and the limited complexity of possible empirical investigations continues to characterize the scientific problem for a discipline of developmental psychopathology.

In 1968, we (Sameroff & Zax, 1973) initiated a study using the high-risk approach developed by Mednick and Schulsinger (1968) in Denmark. We examined the early develop-

ment of children of parents who had a variety of psychiatric diagnoses with special attention to schizophrenia.

At the outset we considered three major hypotheses: (a) that deviant behavior in the child would be attributed to variables associated with a specific maternal diagnosis (e.g., schizophrenia); (b) that deviant behavior would be attributable to variables associated with characteristics of mental illness in general, like the severity and chronicity of the disorder, but no diagnostic group in particular; and (c) that deviant behavior would be associated with social circumstance, exclusive of parental psychopathology.

In general, the first hypothesis found little support. Most of the significant differences found for the schizophrenic group occurred during the prenatal period, and these differences were in the mothers, not in the children. The schizophrenic mothers were the most anxious and least socially competent. They also had the worst prenatal obstetric status.

The second hypothesis, that mental illness in general would produce substantial effects, was supported more strongly. In almost every instance where there was a difference between diagnostic groups, it could be explained by a corresponding difference in the severity or chronicity of the illness. In addition, there were a large number of developmental effects produced by severity or chronicity differences that did not have corresponding diagnostic differences.

When the number of significant outcomes was compared for differences in the diagnostic, mental illness, and social status dimensions, the highest density was found in the social class contrasts, the third hypothesis. One of the more interesting results was that the differences found between offspring of women with psychiatric diagnoses and those without were almost the same as those between offspring of lower and higher social status women. From these analyses a relatively clear picture could be seen. Among the mental illness measures, severity and chronicity of maternal disturbances were better predictors of outcome than were specific diagnoses, but even stronger effects on development were found from social status variables.

Lessons from high-risk research

The excitement of the decade of the 1970s was devoted to high-risk research targeting the offspring of schizophrenic women. As with most fads the achievements rarely match the expectations, and the high-risk study of schizophrenia was no exception. The last two major conference reports of the Risk Research Consortium (Watt, Anthony, Wynne, & Rolf, 1984; Goldstein & Tuma, 1987) contain sobering appraisals of the difficulties inherent in such efforts. Watt (1984) notes in his summary of this work that studies in the Risk Research Consortium had found hundreds of significant differences between children at high and low risk for schizophrenia. Unfortunately, this sensitivity does not extend to specificity because the same differences were found for children at risk for a variety of other psychiatric disorders. The inability of the RLS to find the roots of schizophrenia was not an exception in this research area. I will return to this issue of the universality of risk conditions for a variety of pathologies.

For most of the Risk Research Consortium the initial concern with the etiology of schizophrenia was neither developmental nor social, nor was it concerned with understanding psychopathology in the light of normal behavior—the hallmarks of developmental psychopathology. But by engaging in the search for symptoms in children, the dialectical contradictions between hypotheses and reality led to new attention to the role of environmental experience and the importance of studying developmental processes in normal as well as abnormal children. The nonsymptomatic behavior of most children thought to be at risk led Garmezy (1974), for one, to transfer his concerns to the roots of competence in conditions of adversity. In the RLS we were struck by how our attempts to study the child out of context were defeated by the profound effects of social variables on the lives of the children in our investigation. The contradiction here was that research devoted to understanding the nature of children at risk for schizophrenia brought to the fore information that it may be the nature of the environment that was as im-

portant as any biological heritage for their future mental health.

Conceptualizing Pathology

There are two basic questions that need to be addressed for understanding childhood psychopathology. What does it mean to be disordered? Are disordered children different in kind or in degree? These questions have been addressed by Zigler and Hodapp (1986; Zigler, 1969) in their interpretation of mental retardation. In their view there are two kinds of children with low intelligence scores. One group is dimensional and identified by the diagnostic test. They are part of the normal distribution of any attribute and represent, in the case of mental retardation, the less than 3% of individuals who are 2 standard deviations below the mean. Labeling them as retarded is an artifact of the normal distribution and not of the individuals themselves. It also produced the artifact of the 6-hour retarded child, who only manifests the difficulty when assessed through the lens of scholastic standards yet shows adequate social competence in the worlds of work and social relationships. This categorical view of retardation is further undermined by the major reduction in the percentage of mentally retarded individuals after 18 years of age, when they leave the academic environment and are no longer subject to normed tests of development (Berkson, 1978).

There is a second group of individuals who score in the retarded range who are indeed different in kind from the first. They are organically impaired and the correlates of their low scores on the IQ test are different than those who are only at the low end of the normal distribution. Because their biology is different, the processes by which they develop and the therapeutic treatments required to improve their status may be different from the first group of children who are at the low end of the normal distribution. Behavioral genetic research has provided some confirmation for this dichotomy in that siblings of severely retarded children with IQs less than 50 tend to have normal, average IQs of around 100, whereas siblings of mildly retarded children with IQs in the 60s had a lower average IQ of 85 and 20% were themselves retarded (Nichols, 1984).

When we move from mental retardation to mental illness we are struck with the same question: Do the children with whom we are concerned represent the extreme of a normal distribution, or are they different in kind from the rest of the population? The answer to this question will have powerful implications for our understanding and treatment of their mental health problems. Community surveys of mental health routinely diagnose many more individuals as having psychopathology than make their way to clinical facilities. Are these results because of the lack of adequate services or because their aberrant behavior is compensated by their life circumstances? Are there mental health criteria that distinguish those who are "really" deviant from those who are not? Moreover, will these criteria apply to individuals regardless of their context or only reflect deviance in individuals in specific life circumstances?

Illness is generally associated with suffering. Although adult mental disorder is usually associated with suffering, in the case of children it is usually the pain of others that brings them to clinical attention. It is the parents, teachers, and other caregivers who are the referral agents, especially for young children (Achenbach, 1974). The pain for them is that the child does not fit in. This is not to say that children are not in distress, but when they are it is usually the result of abuse and neglect. In these cases we place the responsibility and the diagnosis on the parents and not on the child. In either case we are confronted with the fact that mental illness in children is not an individual problem; it is a relationship problem in the conflict between the child and the context.

Conceptualizing Individual Development

Although absolutely necessary for scientific progress, one of the biggest problems for the field of psychology, in general, and for developmental psychopathology, in particular, is the use of operational definitions. These definitions require dividing the world into categories that can be easily grasped and catering to reductionist tendencies to view behavior as

traitlike characteristics of the behaver. A number of developmental psychopathologists have been trying to counter this tendency.

One of the more articulate redefinitions of psychopathology in developmental terms has been provided by Sroufe and Rutter (1984) who saw the discipline as "the study of the origins and course of individual patterns of behavioral adaptation" (p. 18). Cicchetti (1986) enlarged this concept by rooting it in Heinz Werner's (1948) classic organismic–developmental approach. He argues that:

> . . . it is necessary to engage in a comprehensive evaluation of those factors that may influence the nature of patterns, and the different pathways by which the same developmental outcomes may be achieved. It is important to map out the processes whereby the normal course of development in the social, emotional, and or cognitive domains, in dynamic transaction with the "inner" constitutional and "outer" environmental characteristics, may lead to outcomes that either inhibit or exacerbate early deviations or maintain or disrupt early adaptation. (p. vii)

However, such complex descriptions are not easy to understand. Traits and their psychopathological analogs, diagnoses, are much easier. An individual is examined and a descriptor is applied—sad, manic, hyperactive, oppositional. Much like physical characteristics such as skin color, height, body shape, they are thought of as inhering in the individual. If the individual moves from Situation A to Situation B, these attributes remain the same. A *pattern of adaptation* is far more complicated to assess. The implication of adaptation is that when situations change the individual changes. Here, categories are not inherent in the individual but in relationships between individuals and situations.

Consider the practical difficulty in using such a system for understanding child development. One would have to know the general adaptive problems of children as they go through various life phases. Then one would have to know the specific cultural variations and expectations for emotional and behavioral expression. Then one would have to know the unique family parameters related to parent figures, caregiving figures, and sibship patterns. And finally, one would have to know the stresses and supports provided by the neighborhood, community, and historical epoch.

Traits

A simpler alternative would be to use an assessment of traits of the child which are more easily classified through responses to a diagnostic interview or a behavioral questionnaire. The initial foray of psychiatry into child mental health involved a downward extension of adult categories with the expectation that children would fit these classifications—to the extent that they would fit any categories. Costello and Angold (1996) in a review of the history of childhood psychopathology point out that the first most basic distinction for adults and children was between "imbeciles" and "lunatics," between the mentally retarded and the mentally ill. For children the next distinction was between instinctive insanity, which was an aberration of instincts and passions, and moral insanity, which was a defect in moral qualities. Today we have a much more elaborate schema with clear categories and descriptions and criteria for classifying children. Depression and conduct disorder are two of the most active research areas in developmental psychopathology, so I would like to use these disorders as illustrations of problems in diagnosis.

Depression

The criteria that have been used to identify children with depression vary from high scores on a parent checklist to careful diagnostic interviews. Compas and Hammen (1994) have done an extensive analysis of the meaning of such scores. They raise three questions overlapping with our present concerns. The questions are whether a depressive disorder in childhood takes the same form as a depressive disorder in adulthood, whether high depression scores are different in quality or merely quantity from low depression scores, and a new issue involving the high correlation between symptoms of depression and symptoms of other disorders.

Their answers increase the complexity of the diagnostic problem because there appear to be three levels of depressive phenomena with similar degrees of sadness—depressed mood, depressive syndromes, and depressive disorders. It is only the latter, with criteria for a extended duration and accompanying functional impairment, that qualifies for the categorical diagnosis. But the bigger difficulty is that it is rare for children who have depression problems to only have depression problems. There is a tendency for emotional and behavioral problems to cluster or co-occur in the same individual. This co-occurrence can be variously thought of as covariation, interrelatedness, or comorbidity.

Comorbidity is a fascinating issue. It should be rare for an individual to have one serious disorder, much less two. Because one has diabetes should not make it more likely to have cancer. But for psychiatric disorders this seems to be the case. For depression comorbidity is the rule not the exception. A review of community epidemiological studies found the range of comorbidity to between 33 and 100% (Flemming & Offord, 1990). Anxiety conditions are most frequently comorbid with depression, so one might think that this could be easily explained because they are both internalizing disorders. But the co-occurence with exernalizing disorders is equally as high, ranging from 17 to 79%, including conduct disorders, oppositional–defiant disorders, attention deficit disorder, and alcohol and drug abuse. Moreover, the worse the course of the child's depression the more likely that she or he would have a concurrent nonaffective comorbid condition (Keller et al., 1988).

For a while when depression was first being discovered in children, it was believed that everything was a symptom of depression. The concept of masked depression was posited as an explanation for all these other symptoms (Cytryn & McKnew, 1979). Now we understand that these other conditions are not simple expressions of underlying depression. They are symptoms and disorders in their own right.

Compas and Hammen (1994) end their review with a provocative idea that high rates of covariation and comorbidity of depressive phenomena are the result of the exposure of high-risk children to multiple sources of risk that contribute independently to negative outcomes. We will return to this idea when we consider the whole issue of risk and resilience.

Conduct disorder

Externalizing problems are much more intrusive into the lives of those around affected children than internalizing problems. Conduct disorders did not require a psychiatric revolution for their discovery and have long been a social as well as clinical concern. Crime is mostly committed by teenagers and young adults, but it does not easily fit in with mental illness categories because for most individuals it is self-limiting. For one reason or another children start and then stop, most within a 1-year period of time (Elliott, Huizinga, & Ageton, 1985).

Although adult antisocial behavior is generally preceded by childhood antisocial behavior, most antisocial children do not become antisocial adults because most adults are not antisocial (Robins, 1978). There does appear to be a group of early offenders who are persistent through early adulthood. Stattin and Magnusson (1991) found that this group accounted for only 5% of their sample but 62% of the crimes. If there was going to be a valid diagnosis of conduct disorder, this would appear to be the group that would have it. Yet this group also had the highest levels of comorbidity. For example, boys who were only aggressive were less likely to become persistent offenders than boys who were aggressive and hyperactive. They were also more likely to have a variety of nondiagnostic problems, including academic deficiencies, poor interpersonal relationships, and deficiencies in social problem solving skills.

Developmental patterns associated with conduct disorder are best described in a developmental analysis of boys from childhood to adolescence by Loeber et al. (1993). They were able to distinguish three statistical pathways: (a) an early authority conflict pathway characterized by stubborn behavior, defiance, and authority avoidance; (b) a covert pathway

characterized by minor covert behaviors, property damage, and moderate to serious forms of delinquency; and (c) an overt pathway characterized by aggression, fighting, and violence. This information is very important for appreciating the developmental trajectories that children follow, but does it throw light on any trait for conduct disorder in these youth? Not as much as we would hope. The worse the disorder, in this case delinquency, the more likely the boys were to be in more than one pattern, with the highest rates for youth who were in all three patterns.

As in the other studies comorbidity is rampant in this sample, with attention deficit–hyperactivity and substance abuse especially associated with the overt pathway. The result is that the more serious the disturbance, not only is comorbidity between disorders more likely, but also multiple deviant pathways within a disorder are more likely.

Individual development as an adaptive system

The developmental approach expands upon traditional models of mental disease by incorporating biological and behavioral functioning into a general systems model of developmental regulation. Within this approach underlying entities do not exist independent of developmental organization. The expression of biological vulnerabilities can occur only in relation to the balance between coping skills and stresses in each individual's life history (Zubin & Spring, 1977). Continuities in competence or incompetence from childhood into adulthood cannot be simply related to continuities in underlying pathology or health, because the level of competence is a function of an adapting system.

Children are integrated wholes rather than collections of traits. When they show evidence of serious dysfunction, it is not restricted to single domains unless we only measure single domains. The worse the problems, the more likely it is that more than one behavioral area is involved. The concept of patterns of adaptation does not provide an easy catalog of behavior but may be a better fit for understanding how children are negotiating their lives and the resulting positive or negative consequences. The pattern concept would require us to continually examine each element of the child's behavior in relation to the whole, a more complex but ultimately more useful way of viewing the child (Achenbach, 1995).

The relations between earlier and later behavior have to be understood in terms of the continuity of ordered or disordered experience across time interacting with an individual's unique biobehavioral characteristics. To the extent that experience becomes more organized, problems in adaptation will diminish. To the extent that experience becomes more chaotic, problems in adaptation will increase. What the developmental approach contributes is the identification of factors that influence the child's ability to organize experience and, consequently, the child's level of adaptive functioning.

Conceptualizing the Environment

The early childhood data from the RLS had a transactional effect on the course of the rest of the study through adolescence. What had begun as a study of children was transformed into a study of environments. We had discovered, on the one hand, that if the only developmental risk for a child was a mother with a mental illness, that child was doing fine. On the other hand, if the child had a mother who was mentally ill and who was also poor, uneducated, without social supports, and with many stressful life events, that child was doing poorly. But we also found that children whose mothers were poor, uneducated, without social supports, and with many stressful life events had worse outcomes, even if the mother did not have a psychiatric diagnosis. In the Rochester study social circumstance was a more powerful risk factor than any of the parental mental illness measures. What we learned was the overriding importance of attending to the context of the children in the study in order to understand their development, but it was not yet clear what would be the best approach to understanding environmental influences.

The analysis of social ecologies proposed

by Bronfenbrenner (1977) described a range of social influences from the parent practices that have direct influence on the child to community and economic factors that can only impinge on the child through the action of others. Depending on disciplinary background different sets of these social variables have been proposed to explain the sources of mental health problems. Economists have focused on poverty and deprivation, sociologists have implicated problems in the community and family structure, educators blame the school system, and psychologists have focused on processes within the family and its members as the environmental influences that most profoundly affect successful development. Rather than viewing these as competing hypotheses, each can be interpreted as a contributor to a positive or negative mental health trajectory. An ecological model emphasizes the contributions of multiple environmental variables at multiple levels of social organization to multiple domains of child development.

For this discussion of issues in developmental psychopathology I will review two environmental issues, the definition of risk, protective, and promotive factors and the multiple-risk model. Although a central role of epidemiology is the identification of the causes of poor health, Costello and Angold (2000) point out that in the study of complex physical disorders the preponderance of studies have identified probabilistic risk factors rather than the singular causes they were designed to seek. Such comprehensive efforts as the Framingham Study of heart disease discovered that no single influence is either sufficient or necessary to produce the disorder. In the domain of mental illness a variety of studies beginning with Rutter (1979) have made prediction of pathology even more nonspecific by indicating that it may be the quantity rather than the quality of risk factors that is most predictive when data from multiple environmental influences are combined.

In the RLS we combined 10 environmental risk variables to calculate a multiple-risk score for each child when he or she was 4 years old. These included (a) a history of maternal mental illness; (b) high maternal anxiety; (c) parental perspectives that reflected rigidity in the attitudes, beliefs, and values that a mother had in regard to her child's development; (d) few positive maternal interactions with the child observed during infancy; (e) head of household in unskilled occupations; (f) minimal maternal education; (g) disadvantaged minority status; (h) single parenthood; (i) stressful life events, and (j) large family size. The resulting score was highly correlated with child mental health. The more risk factors the greater the prevalence of clinical symptoms in the preschoolers (Sameroff, Seifer, Zax, & Barocas, 1987). These effects were also found when multiple environmental risk scores were correlated to child's mental health at 13 and 18 years of age (Sameroff, Bartko, Baldwin, Baldwin, & Seifer, 1998).

Another opportunity to examine the effects of multiple environmental risks on child development was provided by data emerging from a study of adolescents in a large sample of Philadelphia families (Furstenberg, Cook, Eccles, Elder, & Sameroff, 1999). We took a more conceptual approach in designing the project so that there were environmental measures at a series of ecological levels: Parent–child interaction, parent personality, family process, peer influences, school quality, and neighborhood resources. As in the Rochester study there were linear relations between a multiple risk score and adolescent mental health, problem behavior, and academic performance (Sameroff et al., 1998).

The concern with preventing developmental failures has often clouded the fact that the majority of children in every social class and ethnic group are not failures. They get jobs, have successful social relationships, and raise a new generation of children. The concern with the source of such success has fostered an increasing concern with the development of competence and the identification of protective factors as in the work of Masten and Garmezy (1985). However, the differentiation between risk and protective factors is far from clear, and there continue to be many theoretical and methodological limitations in their identification (Luthar & Zigler, 1991). Here we again find limitations when the study of pathology is separated from the study of health.

Although some have argued that protective factors can only have meaning in the face of adversity (Rutter, 1987), in most cases protective factors appear to be simply the positive pole of risk factors (Stouthamer–Loeber et al., 1993). In this sense a better term for the positive end of the risk dimension would be *promotive* rather than protective factors. To test this simplification we created a set of promotive factors by identifying families in the Philadelphia study at the positive pole of each of our risk factors (Sameroff, Seifer, & Bartko, 1997). For example, where a negative family climate had been a risk factor, a positive family climate now became a promotive factor, or where a parent's poor mental health was a risk factor her good mental health became promotive. We then summed these promotive factors and examined their relation to adolescent outcomes. The results mirrored the effects of multiple risks. Families with many promotive factors did substantially better than families from contexts with few promotive factors. For the youth in this study there did not seem to be much difference between the influence of risk and promotive variables. The more risk factors the worse the outcomes, the more promotive factors the better the outcomes. In short, when taken as part of a constellation of environmental influences on child development, most contextual variables in the parents, the family, the neighborhood, and the culture at large seem to be dimensional, promoting mental health at one end and producing mental disorder at the other.

Interpreting environmental action

Reviews of the environmental risk factors that lead to particular mental health problems have been converging on what at first appeared to be a startling conclusion. The lists of risks associated with depression, conduct disorder, substance abuse, and even schizophrenia look the same. Bad environmental elements that affect one outcome also affect others (Coie et al., 1993). This is certainly unsatisfying when one is searching for causal chains leading from antecedents to consequences.

Developmental psychology has and should thrive on the study of processes. We need to know the details of how all pieces work together. But when we are seeking the cause of pathology, those individuals who are at the low end of the normal distributions, we may not be able to find it in single processes. Development is composed of many part processes, each requiring its own set of experiences. But these processes are integrated into a whole by the developing self. If one or another part of this progression goes awry, there are sufficient compensating processes in the average social environment. However, when the compensating processes are missing as well as the nurturing processes, regulation becomes more and more difficult. It is the accumulation of environmental adversity that combines with unusual needs of the child that produces initial patterns of maladjustment which then spin their way to diagnosable pathology. Multipathology, perhaps a better term than comorbidity, is usually associated with a plenitude of risk factors and a paucity of promotive ones.

Regulatory systems in development

A theory that integrates our understanding of pathology and development must explain how the individual and the context work together to produce patterns of adaptive or maladaptive functioning, and relate how such past or present functioning influences the future. The most basic principle to emerge in such a general theory of development is that individuals can never be removed from their contexts. Whether the goal is understanding causal connections, predicting outcomes, or intervention, it will not be achieved by removing the individual from the conditions that regulate development.

Growing attention is being given to the biological regulators of development. New advances in biological research are forcing more attention to be paid to analyzing environmental influences. At the molecular level we have learned that despite the fact that every cell in an organism has the same genotype, each will have different characteristics and a different history. This differentiation is a function of the differing experiences of each cell; these are environmental effects.

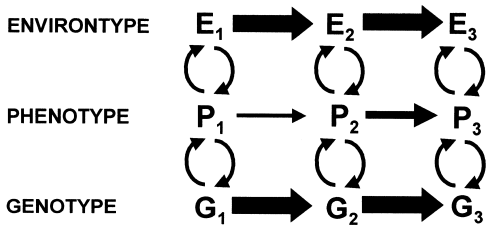

Figure 1. Model of transactional development where the environtype and genotype mutually regulate and are regulated by the phenotype over time (adapted from Sameroff, 1989).

Similarly at the level of behavioral genetics we have learned that each family member has a unique environment of his or her own (Plomin, 1994). The concept of nonshared environments forces us to move beyond SES or family warmth as our primary indicators of the environment to measures of how each individual is experiencing his or her own niche. As the child develops, the number of proximal environments expands from the parents and siblings in the immediate family, to the peer group, and to the school and community, and each has its own set of influences on the course of development.

Environtypes

To alleviate our scientific distress at the multiplying number of documented influences on development, I (Sameroff, 1989) proposed a conceptual simplification that disguises a large measure of complexity (see Figure 1). In this model there is an organization of the environment over time that captures the processes that are relevant to individual development. Just as there is a biological organization, the genotype, that regulates the physical outcome of each individual, there is a social organization that regulates the way human beings fit into their society. This organization operates through family and cultural socialization patterns and has been postulated to compose an "environtype" analogous to the biological genotype.

The child's behavior at any point in time is a product of the transactions between the phenotype (i.e., the child), the environtype (i.e., the source of external experience), and the genotype (i.e., the source of biological organization). Traditional developmental research has emphasized the child's utilization of biological capacities to gain experience and the role of experience in shaping child competencies, but there has been far less attention to how that experience is organized. Indeed, the organization of experience is explicit in the great amount of attention given to curriculum development and behavior modification plans, but far less attention is given to the implicit organization of experience found in the environtype.

From conception to birth interactions with the biological system are most prominent. These processes continue less dramatically after birth with some exceptions (e.g., the initiation of adolescence and possibly senility). The period from birth to adulthood is dominated by interactions with the social system. The result of these regulatory exchanges is the expansion of each individual's ability for biological self-regulation and the development of behavioral self-regulation.

There are clearly individual factors in developmental success, but it is the environtype through the actions of parents, child-care providers, educators, therapists, and other social agents that facilitates adaptation. If a parent or teacher is unresponsive or unadaptive to the unique needs of the child, this should result in a diagnosis of deviancy aimed at the parent or teacher as well as at the child. Successful assessments need to be directed at the environment to the same degree as to the child if we are to have successful interventions and treatments.

What is clear is that there is no emergent simplification on either the environmental or constitutional side that can explain how successful development occurs or how development can be changed. Single factors can be potent in destroying systems. An earthquake can destroy a city, or a gunshot can destroy a child. But single factors cannot create a child or any other living system. At the biological level 100,000 genes are required to transform an egg cell into an adult human body, each gene expressing itself in precise degrees at precise times in precise locations. It may take far more than 100,000 events to produce the

complex psychological functioning of the adult human, integrating a wide variety of environmental experiences with a wide variety of developing capacities.

Contexts and adaptation

The field of developmental psychopathology has introduced an important reorientation to the study of mental health and disorder. The principles of development that apply to the achievement of healthy growth are now seen as the same ones that apply to the achievement of illness (Sroufe & Rutter, 1984). In this view most illnesses are indeed achievements that result from the active strivings of each individual to reach an adaptive relation to his or her environment. The nutrients or poisons that experience provides will flavor that adaptation. No complex human accomplishment has been demonstrated to arise without being influenced by experience. From this dynamic perspective the discussion of developmental psychopathology can be summarized in three aspects: an adaptational process, a linkage between constitution and experience, and a linkage across time.

The study of the adaptational process emphasizes the constructive aspect of development where each individual comes to terms with the opportunities and limitations of experience to produce a uniquely integrated outcome. The study of the linkage between constitution and experience contains the recognition that no individual can be understood apart from the context in which he or she lives. The study of linkages across time is perhaps the most defining of developmental psychopathology in that it contains the basis for continuities and discontinuities.

The perspective taken by developmental psychopathology offers a powerful alternative to nondevelopmental approaches because principles of process are integrated into an understanding of behavioral deviancy. Where traditional views have seen deviancy as inherent in the individual, developmental views place deviancy in the dynamic relation between the individual and internal and external contexts. Similarly, when we view the history of developmental psychopathology as a field we see further evidence of the importance of developmental principles as we trace the progress of researchers and see how their patterns of adaptation, expressed in theoretical models and empirical paradigms, increasingly have come to match the complexities of human mental health and illness.

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