A Dialectic Integration of Development for the Study of Psychopathology

2

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The field of developmental psychopathology was initially focused on efforts to understand the etiology of adult mental disorders by studying children and their disorders. However, this effort produced unanticipated changes in our understanding of pathology, individual development, and the role of social context. Among these modifications were 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. From another perspective, the history of developmental psychopathology is an example of universal dialectical processes where action in the world, that is, research on mental illness, produces results that contradict the models that inspired that action, that is, linear models of individual psychopathology. Dialectical developmental processes are evident as we trace how patterns of adaptation by researchers, expressed in theoretical models and empirical paradigms, increasingly have come to match the complexities of human mental health and illness.

The attention of philosophers and then scientists to human development has always begun with a concern that children should grow up to be good citizens who would contribute to society through diligent labor, moral family life, and civil obedience, and, more recently, to be happy while making these contributions. The motivation for these concerns was that there were many adults who were not. Although attention was paid to the socialization and education of children, it was ultimately in the service of improving adult performance. The societal concern has always had a lifespan perspective. Without healthy, productive adults no culture could continue to be successful.

With these civic motivations and supports, there have been major advances in our understanding of the intellectual, emotional, and social behavior of children, adolescents, and adults. Moreover these understandings have increasingly involved multilevel processes cutting across disciplinary boundaries in the social and natural sciences. This progress has forced conceptual reorientations as earlier unidirectional views that biological or social circumstance controlled individual behavior have become multidirectional perspectives where individual behavior reciprocally changes both biological and social circumstance.

Understanding continuity was the basis of traditional developmental science. Understanding discontinuity is the basis of contemporary developmental science. Why is it that a biological gene or human trait does not always lead to the same outcome? More complexly, why is it that some children who are doing well end up as adults with many problems, and more hopefully, why is it that some children with many problems end up doing very well as adults? The answer lies in the

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series of development steps where context amplifies or reduces the effects of prior steps. Multidisciplinary efforts in the biological and social sciences continue to demonstrate that successful developmental predictions from prior genetic or psychological measures are highly contingent on the child's environment. For those concerned with improving developmental outcomes, explaining discontinuities has a high priority because they offer opportunities to change the course of development through therapeutic interventions. Understanding such discontinuities requires integrating analyses of individual behavior with constructs from the full range of life and social sciences.

The theoretical history of developmental psychopathology has been characterized by swings between beliefs that determinants of an individual's behavior could be found either in their irreducible fundamental units or in their irreducible fundamental experiences. The growth process between babyhood and adulthood could be explained by appeals either to *intrinsic* properties of the child or to *extrinsic* properties of experience—the nature-nurture dilemma. Current research continues to document how deterministic conceptualizations of either emphasize the limitations of both approaches. In a collaborative study of the genetic determinants of height, one of the most heritable human traits, with a combined sample of 63,000 individuals and assessing 500,000 genetic variations, three genes were found to be related to the outcome (Visscher, 2008). Combined they explained only 3 % of the variance. If 97 % of the variance is left unexplained in this classic quantitative trait, what can we expect for much more complex psychological characteristics? On the environmental side one of the most universal transmitted traits is culture. However, when culture is examined as a predictor, more variation for psychological traits is found within cultures than between them. Similarly more psychological variation is found within neighborhoods than between, within schools than between and within families than between (Furstenburg, Cook, Eccles, Elder, & Sameroff, 1999).

Practically, the nature-nurture question comes into play when a child has a problem, and the question arises, "Who is responsible?" Most parents' first response is to blame the child, and most professionals' first response is to blame the parents. However, most scientists know that it is both. It is both child and parent, but it is also neurons and neighborhoods, synapses and schools, proteins and peers, and genes and governments. But that conclusion does not explain how it is both. Explicating the probabilistic transactions between individual and context will be the topic of this chapter. In what follows I will present a contemporary summary of what developmental models should contain and offer a suggestion for an integrated view of psychopathology that captures much of the variance that needs explaining.

Roots of Developmental Psychopathology

There is a set of unresolvable dialectical contradictions inherent in any discipline, and it is within these contradictions that the sources of progress can be found. 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. One of the basic contradictions in each of these domains is between the labels used to divide and categorize the phenomena of concern and the dynamic reality which comprises the phenomena themselves. Unique to the study of pathology is the contradiction between the abstracted diagnostic schemes used for categorizing individuals and the complex dynamic processes of the individuals themselves.

Another contradiction is the contrast between the study of serious mental disorders and mental health. Whereas clinicians have needed to center their attention on children who are in the greatest therapeutic need, most developmentalists who have entered 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 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).

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

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? This is one aspect of the continuity vs. discontinuity issue, here between one kind of individual and another.

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) characterized 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 as either deterministic or probabilistic.

There are two basic questions that need to be addressed for understanding childhood psychopathology. One is *what does it mean to be disordered,* and the other is *are disordered children different in kind or in degree*. These issues have been best described by Zigler and Hodapp (1986) 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 two 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 will be 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 may be different, 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 lowest part 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 between individuals and their specific contexts?

How Do We Understand Individuals and Their Development?

Is it through a search for stable characteristics of the individual independent of context, or is it the search for patterns of functioning in context? Moreover, when these characteristics change over time, is it the unfolding of some maturational pattern or a reaction to new contextual demands as each individual interacts with an expanding social domain? Again the continuity-discontinuity issue is of central concern.

Progress in the technology of molecular genetics has led to hopes that the etiology of mental disorders will soon be revealed and that their treatment and prevention will follow. 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 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 hypothesized lines of development are those that produce the emotionally disturbed, such as schizophrenics and depressives; the cognitively disturbed, such as the learning disabled and the retarded; and the undisturbed, that is, normal individuals.

But does 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 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. Garmezy, 1985).

The Rochester Longitudinal Study (RLS) that my colleagues, Melvin Zax, Ronald Seifer, Ralph Barocas, and Alfred and Clara Baldwin, have been involved in for 40 years (Sameroff, Seifer, Baldwin, & Baldwin, 1993; Sameroff, Seifer, & Zax, 1982; Sameroff & Zax, 1973) was 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 characterizes the scientific problem for the discipline of developmental psychopathology.

In 1968, we (Sameroff & Zax, 1973) initiated a study using the high-risk approach to examine the early development of children of parents who had a variety of psychiatric diagnoses with special attention to schizophrenia. At the outset we considered three major hypotheses: (1) that deviant behavior in the child would be attributed to variables associated with a specific maternal diagnosis, e.g., schizophrenia; (2) 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 (3) that deviant behavior would be associated with social circumstances, exclusive of parental psychopathology.

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 and/or chronicity of the illness. In addition, there were a large number of developmental effects produced by severity and/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 risk than their specific diagnoses, but even stronger effects on development were found from social status variables. At Rochester 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 the child at risk for schizophrenia brought to the fore information that it may be the nature of the environment that was as important as any biological heritage for their future mental health.

How Do We Conceptualize the Environment?

Is it a passive set of additive experiences that maximizes or minimizes innate individual potential as in the concept of genetic ranges of reaction, or does experience have nonlinear transformative effects as it interacts and transacts with dynamic individual developmental processes? This issue will be fully explored in the following description of a unified theory of development.

A Unified Theory of Development

In tune with the advanced understanding of molecular genetics, there is a contemporary zeitgeist emphasizing dynamic conceptualizations within most scientific disciplines. In his spirit, I recently proposed that contemporary theories of development require at least four models for understanding human psychological change: a *personal* one, a *contextual* one, a *regulation* one, and a *representational* one (Sameroff, 2010). However, a fifth model for *evolutionary* change has become essential. The *personal model* is necessary for understanding the progression of competencies from infancy on. It requires unpacking the changing complexity of the individual as he or she moves from the sensorimotor functioning

of infancy to increasingly intricate levels of cognition, from early attachments with a few caregivers to relationships with many peers, teachers, and others in the world beyond home and school, and from the early differentiation of self and other to the multifaceted personal and cultural identities of adolescence and adulthood. The *contextual model* is necessary to delineate the multiple sources of experience that augment or constrain individual development. The growing child is increasingly involved with a variety of social settings and institutions that have direct or indirect impact as exemplified in Bronfenbrenner's (1977) view of the social ecology. The *regulation model* adds a dynamic systems perspective to the relation between person and context. During early development, human regulation moves from the primarily biological to the psychological and social. What begins as the regulation of temperature, hunger, and arousal soon turns to regulation of attention, behavior, and social interactions. The *representational model* is where an individual's here and now experiences in the world are given a longer term existence in thought. These representations are the cognitive structures where experience is encoded at abstracted levels that provide an interpretive structure for new experiences, as well as a sense of self and other. Finally the *evolutionary* model is necessary to explain the codevelopment of genetic polymorphisms, psychological, and social functioning. Combining these five models offers a comprehensive view of the multiple parts, wholes, and interconnecting processes that comprise human development, especially as they are related to psychopathology. Moreover, within each model there is evidence of discontinuities that can expand or contract the developmental success of children.

Personal Model

Because psychology's central focus is on individuals, developmental psychopathology's main concerns have been on how children change over time, especially how early characteristics lead to mental health problems. How one thinks about change will have a clear influence on research objectives. Three ways of conceptualizing change are notions about trait, growth, and development. If one believes that an individual consists of a set of unchanging traits, then there is no need for developmental research. Thinking about change as a growth process allows for change but only on quantitative dimensions, more words, more numbers, more ideas. Viewing personal change as development implies qualitative changes where there is a period of stability of functioning followed by a transition to a structurally different period of stability presumed to reflect more encompassing cognitive and social functioning. The classic examples of stages are in the writings of Freud and Piaget. Although there have been major revisions or rejections of their specific formulations, there are some generally accepted notions that within many domains individuals move in steps from novices, to experts, to masters where they do not just do things better, they do things differently (Ericsson & Charness, 1994). Qualitative or structural reorganizations of the individual are the points of discontinuity where children can enter different trajectories for better or worse. The study of depression and conduct disorder in children are examples of empirical complexities in attempts to use specific diagnoses as continuing individual characteristics.

Depression

The criteria for identifying children with depression vary from high scores on a parent checklist to careful diagnostic interviews. Compas and Hammen (1994) did an extensive analysis of the meaning of such scores, and they raised three questions overlapping with our present concerns. The questions were 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 whether depression is a unitary construct that can be separated from the symptoms of other disorders—the comorbidity question.

Their conclusions 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 an 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 be 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-occurrence with externalizing 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 non-affective 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, 1974). 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 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 than internalizing problems into the lives of those around affected children. 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 one-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 has the highest levels of comorbidity. Boys who were only aggressive were less likely to become persistent offenders than boys who were aggressive and hyperactive, for example. They are also more likely to have a variety of nondiagnostic problems including academic deficiencies, poor interpersonal relationships, and deficiencies in social problem solving skills.

Developmental pathways associated with conduct disorder have been increasingly studied (cf. NICHD Early Child Care Research Network, 2004). An interesting example is a developmental analysis of boys from childhood to adolescence by Rolf Loeber and his colleagues (Loeber et al., 1993). They were able to distinguish three 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 through these

behavior patterns, 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 pathway, with the highest rates for youth who were in all three pathways. As in other such studies, comorbidity is rampant in this sample with attention-deficit hyperactivity and substance abuse especially associated with the overt pathway. The result is that not only the more serious the disturbance the more comorbidity between disorders but also the more deviant pathways within a disorder.

What we have learned from this discussion of individual behavior is that children are integrated wholes rather than collections of diagnostic traits. When they show evidence of serious dysfunction, it is not restricted to single domains unless the study only measures single domains of dysfunction. The worse the problems, the more likely it is that more than one behavioral area is involved. This conclusion is in keeping with one of the more articulate redefinitions of psychopathology in developmental terms provided by Sroufe and Rutter (1984) who saw the discipline as the study of patterns of adaptation rather than individual traits.

Contextual Model

Although developmental psychopathology is focused on individuals, it has become clear that understanding change requires an analysis of an individual's experience. Behavior, in general, and development, in particular, cannot be separated from the social context. Our understanding of experience has moved from a focus on primary caregivers to multiple other sources of socialization. There were many predecessors who felt that families, schools, neighborhoods, and culture had influences on development, but Bronfenbrenner (1977) turned these ideas into a comprehensive framework with predictions of how these settings affect the child but also how they affect each other. Although his terminology of microsystems, mesosystems, macrosystems, exosystems, and chronosystems may not be universally accepted, his principle that the family, school, and community are all intertwined in explaining any particular child's progress is now universally acknowledged.

The analysis of social ecologies proposed by Bronfenbrenner 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. The ecological model emphasizes the contributions of multiple environmental variables at multiple levels of social organization to multiple domains of child development.

Traditionally, social contacts were considered to expand from participation wholly in the family microsystem into later contact with the peer group and school system. Today, however, many infants are placed in out-of-home group childcare in the first months of life. Each of these settings has its own system properties such that their contributions to the development of the child are only one of many institutional functions. For example, the administration of a school setting needs attention to financing, hiring, training of staff, and building maintenance before it can perform its putative function of caring for or educating children (Maxwell, 2009). Thus, a sociological analysis of such settings provides information about its ability to impact children.

Attention to the effects on children of changing settings over time must be augmented by attention to changing characteristics of individuals within a setting. Contemporary social models take a life course perspective that includes the interlinked life trajectories of not only the child

but other family members (Elder, Johnson & Crosnoe, 2003). For example, experience for the child may be quite different if the mother is in her teens with limited education or in her 30s after completing professional training and entry into the job force.

For the purposes of this discussion of issues in developmental psychopathology, I will restrict this review to two environmental issues, the multiple risk model and the contrast among risk, protective, and promotive factors. Although a central role of epidemiology is the identification of the causes of poor health, Costello and Angold (1996) point out that in the study of complex physical disorders, the preponderance of studies have identified risk factors rather than causes. Moreover, such comprehensive efforts as the Framingham Study of heart disease have 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 noted 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.

Capturing the complex effects of multiple environmental situations has been a daunting enterprise requiring vast sample sizes to capture the unique contributions of each setting. An alternative methodology to dimensionalize the negative or positive quality of a child's experience has been the use of multiple or cumulative risk or promotive factor scores. In the Rochester Longitudinal Study, we combined ten environmental risk variables to calculate a multiple risk score for each child when they were 4 years old. These included (1) a history of maternal mental illness; (2) high maternal anxiety; (3) parental perspectives that reflected rigidity in the attitudes, beliefs, and values that mothers had in regard to their child's development; (4) few positive maternal interactions with the child observed during infancy; (5) head of household in unskilled occupations; (6) minimal maternal education; (7) disadvantaged minority status; (8) single parenthood; (9) stressful life events; and (10) large family size. The resulting score was highly correlated with child mental health; there was a significant linear function. 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 et al., 1999). We took a more conceptual approach in designing the project so that there were 20 environmental measures spread among six ecological levels. These were *family processes* that included support for autonomy, behavior control, parental involvement, and family climate; *parent characteristics* that included mental health, sense of efficacy, resourcefulness, and level of education; *family structure* that included the parents' marital status and socioeconomic indicators of household crowding and welfare status; *family management* comprised of variables of institutional involvement, informal networks, social resources, and adjustments to economic pressure; *peers* that included indicators of association with prosocial and antisocial peers; and *community* that included census tract information on average income and educational level of the neighborhood, a parent report of neighborhood problems, and measures of the adolescent's school climate. In addition to the large number of ecological variables, we used a wide array of youth developmental outcomes in five domains: *psychological adjustment*, *self-competence*, *conduct problems*, *extracurricular involvement*, and *academic performance*.

For the environmental risk analyses, each of the 20 variables was dichotomized with approximately a quarter of the families in the high-risk group and then the number of high-risk conditions summed. When we examined the relation between the multiple risk factor score and the five adolescent outcomes, there were large declines in outcome with increasing risk and a substantial overlap in slope for each (Sameroff, 2006). Although this kind of epidemiological research does not unpack

the processes by which each individual is impacted by contextual experience, it does document the multiple factors in the environment that are candidates for more specific analyses.

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).

Some have argued that protective factors can only have meaning in the face of adversity (Rutter, 1987), that is, much reduced effects for advantaged children. But in most cases protective factors appear to be simply the positive pole of risk factors (Stouthamer-Loeber et al., 1993), that is, they help everybody (Guttman, Sameroff, & Eccles, 2002). 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 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, aiding in general child development at one end and inhibiting it at the other.

Of interest here is how the ecological model affects our understanding of continuity and discontinuity. What one would expect is that good families, good schools, and good neighborhoods go together, and conversely bad families, bad schools, and bad neighborhoods are highly correlated. But it turns out this is only true at the aggregate level from one community to another. When one uses individual children as the level of analysis, then the correlations between the quality of the family, peer group, school, and neighborhood become quite modest. Each child can have a quite different experience with a different set of positive or negative contextual features influencing his or her development, but the conclusion does not change in that the more good things in a children's lives, the better their outcomes.

Of great significance for the life course, these effects play out over time as a manifestation of the Matthew effect, "To the man who has, more will be given until he grows rich; the man who has not will lose what little he has" (Matthew 13:12). In a study of high- and low-IQ 4-year-olds, we tracked their academic achievement through high school (Gutman, Sameroff, & Cole, 2003). The low-IQ group living in low contextual risk conditions consistently did better than the high-IQ group living in high-risk conditions. Over time promotive or risky contextual effects either fostered or wiped out prior individual competence.

Regulation Model

The third component of the unified theory is the *regulation model* reflecting the dynamic systems orientation of modern science (Sameroff, 1995). The developmental approach expands upon traditional views 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 imbalance between coping skills and stresses in each individual's life history. Continuities in competence or incompetence from childhood into adulthood cannot be simply related to continuities in underlying pathology or health.

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 and regulate experience and, consequently, the child's level of adaptive functioning.

Growing attention is being given to the biological regulators of development not only at the somatic level but also at the genetic. 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.

The idea that the child is in a dynamic rather than passive relationship with experience has become a basic tenet of contemporary developmental psychology. However, most of the rhetoric is about "self"-regulation. Whether it is Piaget's assimilation-accommodation model in cognition or Rothbart's (1981) reactivity and self-regulatory view of temperament, equilibration is primarily a characteristic native to the child. The context is necessary as a source of passive experiences that stimulate individual adaptation, but has no active role in shaping that adaptation. These views promote a belief that regulation is a property of the person. However, self-regulation mainly occurs in a social surround that is actively engaged in "other"-regulation. At the biological level the self-regulatory activity of genes is intimately connected to the other-regulatory activity of the epigenome and the surrounding cell cytoplasm.

This issue of the developmental expansion of self-regulation to include other-regulation is captured by the *ice-cream-cone-in-a-can* model of development (Sameroff & Fiese, 2000), depicted in Fig. 2.1. The developmental changes in the relationship between individual and context are represented as an expanding cone within a cylinder. The balance between other-regulation and self-regulation shifts as the child is able to take on more and more responsibility for his or her own well-being. The infant, who at birth could not survive without the caregiving environment, eventually reaches adulthood and can become part of the other-regulation of a new infant, beginning the next generation.

It is parents who keep children warm, feed them, and cuddle them when they cry; peers who provide children with knowledge about the range and limits of their social behavior; and teachers who socialize children into group behavior as well as regulate cognition into socially constructed domains of knowledge. Although these other-regulators can be considered background to the emergence of inherent individual differences in regulatory capacities, there has been much evidence from longitudinal research among humans and cross-fostering studies in other animals that "self"-regulatory capacities are heavily influenced by the experience of regulation provided by caregivers. The capacity for self-regulation arises through the actions of others. This regulation by others provides the increasingly complex social, emotional, and cognitive experiences to which the child must self-regulate and the safety net when self-regulation fails. Moreover, these regulations are embedded not only in the relation between child and context but also in the additional relations between family and their cultural and economic situations (Raver, 2004). These regulatory systems range from the here-and-now experiences of parent–child interactions to governmental concern with the burden of national debt that will be passed on the next generation and to conservationists' concerns with the fate of the planet as a viable environment for future generations of humans.

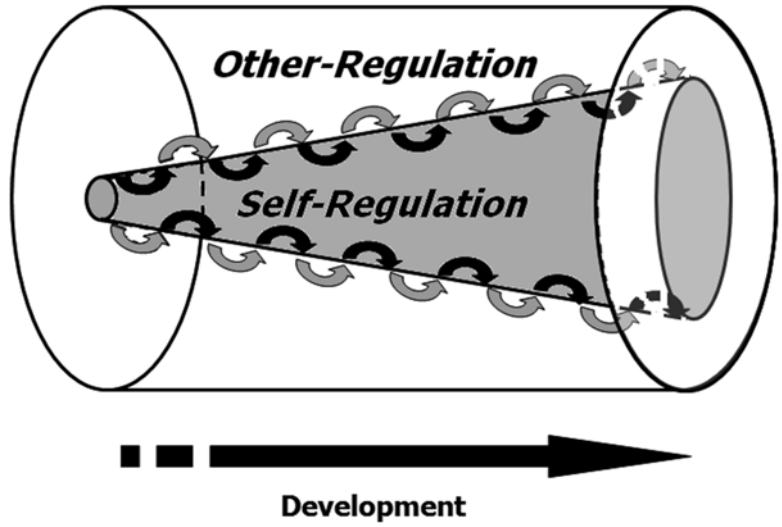

Fig. 2.1 Transactional relations between self-regulation and other-regulation

Early functional physiological self-regulation of sleep, crying, and attention is augmented by caregiving that provides children with regulatory experiences to help them quiet down on the one hand and become more attentive on the other. Sleep is an interesting example where biological regulation becomes psychological regulation through social regulation. As wakefulness begins to emerge as a distinct state, it is expanded and contracted by interactions with caregivers who stimulate alertness and facilitate sleepiness. Although it remains an essential biological process, eventually it takes on a large degree of self-regulation as the child increasingly makes active decisions about waking time and sleeping time. But this agentic decision-making remains intimately connected with other-regulation in terms of the demands of school and work for specific periods of wakefulness.

The relation between self- and other-regulation has implications for diagnostic systems for the psychopathology of children. In an attempt to define mental health diagnoses for infants, Sameroff and Emde (1989) argued for a position that infant diagnoses could not be separated from relationship diagnoses. Our point was that in early development life is a "we-ness" rather than an "I-ness." The developmental and clinical question in this case is when does a diagnosis become individualized, at what stage does a child have a self-regulation problem instead of an other-regulation problem? One answer is to identify the point in development when areas of self-regulation become independent of initial regulatory contexts and are carried into new relationships.

The previous discussion of the need for a construct of other-regulation to complete an understanding of self-regulation leads now to how the relation between self and other operates developmentally, and for this we turn to the transactional model (Sameroff & Chandler, 1975). Transactions are omnipresent. Everything in the universe is affecting something else or is being affected by something else. In the transactional model the development of the child is a product of the continuous dynamic interactions of the child and the experience provided by his or her social settings. What is core to the transactional model is the analytic emphasis placed on the interdependent effects of the child and environment and is depicted in the bidirectional arrows between self and other in Fig. 2.1. The transactional model helps to explain many of the continuities and discontinuities in development. Interactions are typified by continuity where there may be a mutual dependence between one's behavior and another's, but there is not restructuring—there is a stable pattern of correlations. Transactions

occur when one partner changes their behavior such that there is a new pattern of interaction—a discontinuity—that can move in a positive or negative direction. Transactions are opportunities for interventionists to aim for the more positive outcome.

In a recent book on the topic (Sameroff, 2009), a number of researchers documented transactional processes in cognitive and social-emotional domains where agents in the family, school, and cultural contexts altered the course of children's development in both positive and negative directions. Transactional examples have been typically in the behavioral domain with an emphasis on parent–child mutual exacerbations producing problem behavior in both partners (Patterson, 1986). More recently, transactions have been recognized in teacher–student relationships where the effects of the teacher on the child in one grade will change the reaction of the teacher in the next moving the student to higher or lower levels of competence (Morrison & Connor, 2009). Multilevel transactions have also been documented where not only the parent and child are transacting with each other but both are also transacting with cultural practices (Bornstein, 2009).

Representational Model

Since the beginnings of psychodynamic thinking, representations have been used to explain psychopathology and as targets for psychotherapeutic interventions. Representations are encodings of experience that are more or less elaborated internal summaries of the external world. They include the cognitive representations where the external world is internalized, the social representations where relationships become working models, the cultural representations of different ethnicities or social classes, and even the developmental theories discussed here. Representations are obviously not the same as what they represent. They have the function of bringing order to a variable world, producing a set of expectations of how things should fit together that are generally adaptive but in the case of psychopathology tend more toward the maladaptive.

We have long been familiar with such representations as perceptual constancy in which objects are perceived as being a certain size even when the sensory size is manipulated. In such a summation certain aspects are selected and others ignored. In the representation of a square, for example, the size, color, and texture of the square object may be ignored. Analogously, when representations are made of a social object such as a parent, certain features are included in the representation and others are ignored. Research using the adult attachment interview (Main & Goldwyn, 1984) has emphasized that representations of parents are often idealized, where only positive aspects are included in the mental model. Although the links between the quality of representations of child–parent relationships during infancy and those during adulthood are far from direct, early working models of attachment do seem to have long-term consequences for adult development (Sroufe, Egeland, Carlson, & Collins, 2005).

Similarly, parents create representations of their children that emphasize certain aspects, deemphasize others, and have stability over time independent of the child's actual characteristics. We had parents rate their infants' temperament during the first year of life following a structured interaction sequence (Seifer, Sameroff, Barrett, & Krafchuk, 1994). We also had them rate the temperament of six unfamiliar infants engaged in the same interaction sequence. The average correlation in temperament ratings of the unfamiliar infants between mothers and trained observers was 0.84 with none below 0.60. The average correlation in temperament ratings between mothers and trained observers for their own children was 0.35 with a range down to −0.40. Mothers were very good raters of other people's children, but very poor raters of their own due to the personal representations that they imposed on their observations. Documenting such differences in parent representations would be of no more than intellectual interest, if there were not consequences for the later development of the child. For example, infants whose mothers perceived them as problematic criers during infancy increased their crying during toddlerhood and had higher problem behavior scores when they were preschoolers

(McKenzie & McDonough, 2009). Representations are further examples of the ubiquity of discontinuities in development. Individuals, parents in this case, interpret the same reality in quite different ways leading to quite different outcomes from the same initial child conditions.

Individual well-being is also a result of meaningful cultural engagement with desirable everyday routines that have a script, goals, and values (Weisner, 2002). Meaningfulness, a key component of cultural analyses, is primarily found in coherent representations. Meaning systems can have a positive influence as where family routines provide a narrative representation for the family members that allows the whole to continue adaptive functioning despite the variability in the behavior of the parts (Fiese and Winter, 2009), for example, an alcoholic parent or an ill child. The negative effect of a lack of meaningfulness was found in a study of native Canadian youth who showed much higher levels of suicide and other problem behavior when there were large inconsistencies in cultural continuity from one generation to another (Chandler, Lalonde, Sokol, & Hallett, 2003). The order or disorder in a family or society's representation of itself affects the adaptive functioning of its members.

Evolutionary Model

Historically, evolutionary psychologists have tended toward reductionism, explaining current psychological and social organization as the result of Darwinian selective processes on the genome during the history of the species. More recent formulations have added more dynamic conceptualizations to our understanding of both historic evolutionary forces and contemporary gene expression. In each case there is an intimate relationship between the evolving or developing organism and its experiential surround. Of empirical interest are the reformulations of gene–environment interactions in terms of differential susceptibility theory and epigenetics.

The original descriptions of gene–environment interactions (cf. Caspi et al., 2003) found that certain gene alleles produced a greater mental health vulnerability to abusive environments and described these polymorphisms categorically as vulnerability genes. Further research has enlarged the concept of gene–environment interaction into a U-shaped function labeled as differential susceptibility (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011), such that the same allele can produce worse mental health in stressful contexts but better mental health in more supportive social contexts. These opposite effects where the same polymorphism can express itself as either a risk factor or a promotive factor depending on social experience emphasize the lack of determinism in even the most basic individual biological characteristics.

Advances in epigenetics have reframed what we consider to be the basic biological units, from the unchanging genome to the epigenome where experiences are dynamically coded (Meaney, 2010). A fundamental premise of the transmission of genes from one generation to another was that the genotype is not influenced by the experience of the phenotype. This is no longer the case when the more inclusive epigenome is taken into consideration. Not only is the activation of the genome influenced by the experience of the individual, but such changes are transmitted from one generation to another. Researches in both differential susceptibility and epigenetics are further demonstrations of how discontinuities can be found at every level of functioning. Initial conditions alone are not predictive of future development.

From the systems perspective evolutionary theory has provided a fruitful analog for understanding the transitions that lead from one developmental stage to another in the personal model described above. As opposed to the gradualist understanding of evolutionary changes originally proposed by Darwin that would look like the growth model of individual change, Eldredge and Gould (1972) argued that evolution was characterized by continuity evidenced in long periods of stasis where there were only modest changes, alternating with discontinuity, where there were short periods of rapid change, which they labeled *punctuated equilibrium*. The implication was that there was a balance between species and their ecosystems until it was interrupted by either large

changes in the species or large changes in the environment that required a new equilibration. In terms of understanding developmental discontinuities in the individual, we would need to search for such changes in the child or the context that create pressures for a new equilibration leading to future mental order or disorder.

Unifying the Theory of Development

Now that the five models necessary for a theory of development have been described, we can attempt to integrate them into a comprehensive view that contains most known influences on developmental psychopathology using both a structural model that describes all the pieces and then a functional model that shows how their interactions and transactions unfold over time.

The self has often been described as a set of interacting psychological and biological processes as depicted in Fig. 2.2. The psychological domains overlap in cognitive and emotional realms of intelligence, mental health, social competence, and identity, among others. Here they are depicted as the set of grey, overlapping circles comprising the psychological part of the self. Each of these psychological domains is subserved by and interacts with a set of biological processes, including neurophysiology, neuroendocrinology, proteomics, epigenomics, and genomics that are depicted as a set of black, overlapping circles. Together the grey and black circles comprise the *biopsychological* self-system. This self-regulation system transacts with the other-regulation system, depicted by the surrounding white circles, representing the many settings of the social ecology, including family, school, neighborhood, community, and overarching geopolitical influences. Taken together the three sets of overlapping circles comprise the *biopsychosocial* aspects of an individual in context.

Next, the developmental model of personal change is added to the biopsychosocial model, where there are qualitative shifts in organization reflecting changing relationships among the biopsychosocial aspects as seen in Fig. 2.3. These periods of changing organization are analogous to the evolutionary shifts described in the theory of punctuated equilibria. The leading edge for these changes can originate in the individual, represented by the arrows pushing outward in the figure, or from the context, as represented by the arrows pushing inward, resulting in points of inflection, that is, developmental change. It is the relation between shifts in the child and shifts in the context that mark new stages. Such individual shifts can be tied to personal changes as mundane as beginning to walk or as complicated as adolescence.

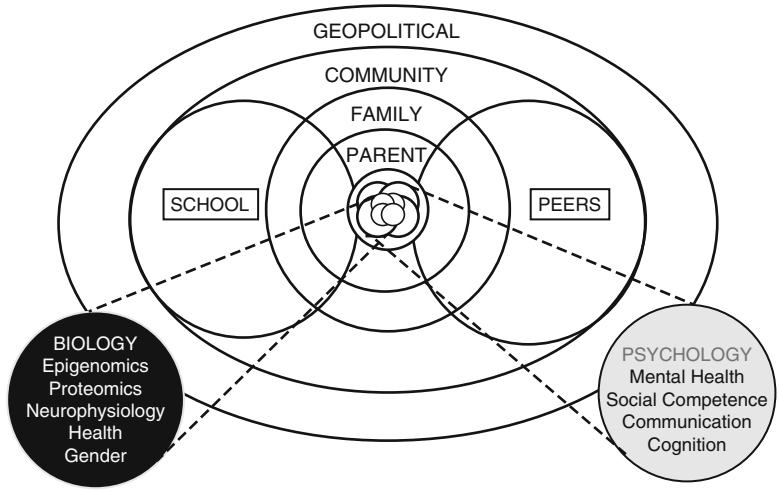

Fig. 2.2 Biopsychosocial ecological system

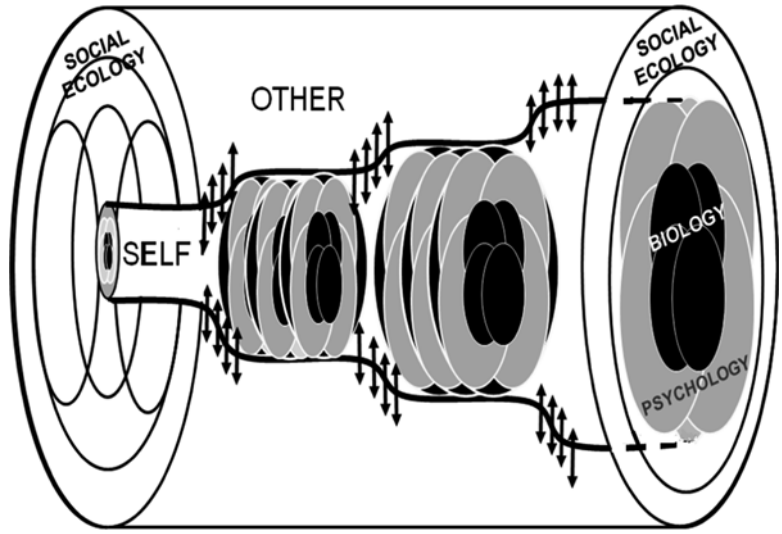

Fig. 2.3 Unified theory of development including the personal change, context, and regulation models

Puberty is a biological achievement of the child, but adolescence is a socially designated phase between childhood and adulthood (Worthman, 1993). Puberty is universal but adolescence is not, in either historical or cross-cultural perspective. In many cultures adolescence is directly tied to biological changes, but in modernizing cultures it is more closely tied to age-based transitions into middle and high schools. Depending on the culture sexual participation can be encouraged at an early age before biological maturity or discouraged until individuals are well into adulthood. These pressures from changes in the child and the context are represented by the up and down arrows around the adolescent transition in Fig. 2.3. In western societies, adolescence is generally recognized, but the quality of the adolescent experience is quite variable and may be heavily dependent on stage-environment fit. Depending on the particular family or school system, desires for autonomy and intimacy can be fostered or thwarted moving the adolescent into better or worse future functioning. Negative psychological changes associated with adolescent development often result from a mismatch between the needs of developing adolescents and the opportunities afforded them by their social environments (Eccles et al., 1993).

The recent emphasis on identifying developmental cascades in psychopathology offers many empirical examples of the interplay between individual and contextual shifts over time (cf. Masten & Cicchetti, 2010). An informative example is the work of Dodge et al. (2009) explaining the predictive cascade between child, parents, and peer group leading from a difficult temperament in infancy to substance abuse in adolescence. The direct correlation between infant and adolescent characteristics is negligible, but becomes amplified as infant problems lead to parent problems that lead to peer problems and back again to later parenting and peer problems and finally to the adverse adolescent outcome.

The unified theory depicted in Fig. 2.3 combines the personal change, contextual, and regulation model, but it would become overly complex to add the representational model to the figure as well. Suffice it to say that representation suffuses every aspect of the model in the interacting identities, attitudes, beliefs, and attributions of the child, the family, the culture, and the organizational structure of social institutions. Moreover, the way developmental science conceptualizes the child may be only one of a number of possible cultural inventions (Kessen, 1979). The most important representation for current purposes is captured in the depiction of a unified theory of development. Like most theories the unified view does not make specific predictions, but does specify what will be necessary for explaining the developmental

phenomena in psychopathology. It is a reversal of the usual bottom-up empirical stance where the researcher maintains as narrow focus as possible unless forced to enlarge the scope by some contradictory findings. The top-down theoretical stance is that researchers need to be aware that they are examining only a part of a larger whole consisting of multiple interacting dynamic systems where each influences the outcome of interest. Over time the body changes, the brain changes, the mind changes, and the environment changes along courses that may be somewhat independent of each other and somewhat a consequence of experience with each other. It should be a very exciting enterprise to fill in the details of how biological, psychological, and social experiences foster and transform each other to explain both adaptive and maladaptive functioning across the life course.

Development and Psychopathology

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. 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 the internal and external context.

I have summarized a universal theory of development that can be used to explain both ordered and disordered adaptive processes using the same models. Within this framework are answers to the questions of defining pathology, understanding individuals and their development, and conceptualizing the environment. All children are constantly adapting to and requiring adaptations from their caregiving environment. Individual differences from the genome on have the potential to lead to more positive mental health outcomes. Which path will be taken is the result of a continuous dynamic with the ability of the context to support or subvert developmental achievements. The extent of our understanding of the elements of this dynamic will limit or increase our ability to plan intervention efforts to move children toward adaptive solutions.

References

Berkson, G. (1978). Social ecology and ethology of mental retardation. In G. P. Sackett (Ed.), *Observing behavior, Vol. I: Theory and applications in mental retardation* (pp. 403–409). Baltimore, MD: University Park Press.

Bornstein, M. H. (2009). Toward a model of culture-parent–child transactions. In A. Sameroff (Ed.), *The transactional model of development: How children and contexts shape each other* (pp. 139–161). Washington, DC: American Psychological Association.

Bronfenbrenner, U. (1977). Toward an experimental ecology of human development. *American Psychologist, 32*, 513–531.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science, 301*, 386–389.

Chandler, M. J., Lalonde, C. E., Sokol, B. W., & Hallett, D. (2003). Personal persistence, identity development, and suicide: A study of native and non-native North American adolescents. *Monographs of the Society for Research in Child Development*, *68*(2, Series No. 273)

Cicchetti, D. (1989). Developmental psychopathology: Some thought on its evolution. *Development & Psychopathology, 1*, 1–4.

Compas, B. E., & Hammen, C. L. (1994). Child and adolescent depression: Covariation and comorbidity in development. In R. J. Haggerty, L. R. Sherrod, N. Garmezy, & M. Rutter (Eds.), *Stress, risk, and resilience in children and adolescents: Processes, mechanisms, and interventions* (pp. 225–267). New York: Cambridge University Press.

Costello, E. J., & Angold, A. (1996). Developmental psychopathology. In R. B. Cairns, G. H. Elder Jr., & E. J. Costello (Eds.), *Developmental science* (pp. 168–189). New York: Cambridge University Press.

Cytryn, L., & McKnew, D. H. (1974). Factors influencing the changing clinical expression of the depressive process in children. *American Journal of Psychiatry, 131*, 879–881.

Dodge, K. A., Malone, P. S., Lansford, J. E., Miller, S., Pettit, G. S., & Bates, J. E. (2009). A dynamic cascade model of the development of substance-use onset. *Monographs of the Society for Research in Child Development, 74*(3), 1–134.

Eccles, J. S., Midgley, C., Wigfield, A., Buchanan, C. M., Reuman, D., Flanagan, C., et al. (1993). Development during adolescence: The impact of stage-environment fit on young adolescents' experiences in schools and in families. *American Psychologist, 48*, 90–101.

Elder, G. H., Jr., Johnson, M. K., & Crosnoe, R. (2003). The emergence and development of life course theory. In J. T. Mortimer & M. J. Shanahan (Eds.), *Handbook of the life course* (pp. 3–19). New York: Kluwer Academic/Plenum.

Eldredge, N., & Gould, S. J. (1972). Punctuated equilibria: An alternative to phyletic gradualism. In T. J. M. Schopf (Ed.), *Models in paleobiology*. San Francisco, CA: Freeman Cooper.

Elliott, D. S., Huizinga, D., & Ageton, S. S. (1985). *Explaining delinquency and drug use*. Beverly Hills, CA: Sage.

Ellis, J. E., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary–neurodevelopmental theory. *Development and Psychopathology, 23*, 7–28.

Ericsson, K. A., & Charness, N. (1994). Expert performance: Its structure and acquisition. *American Psychologist, 49*(8), 725–747.

Fiese, B. H., & Winter, M. A. (2009). The dynamics of family chaos and its relation to children's socio-emotional well being. In G. W. Evans & T. D. Wachs (Eds.), *Chaos and its influence on children's development: An ecological perspective* (pp. 55–76). Washington, DC: APA Books.

Flemming, J. E., & Offord, D. R. (1990). Epidemiology of childhood depressive disorders: A critical review. *Journal of American Academy of Child and Adolescent Psychiatry, 29*, 571–580.

Furstenberg, F. F., Jr., Cook, T. D., Eccles, J., Elder, G. H., Jr., & Sameroff, A. (1999). *Managing to make it: Urban families and adolescent success*. Chicago: University of Chicago Press.

Garmezy, N. (1985). Stress-resistant children: The search for protective factors. In J. E. Stevenson (Ed.), *Recent research in developmental psychopathology* (pp. 213–233). Oxfort: Pergamon Press.

Gutman, L. M., Sameroff, A. J., & Cole, R. (2003). Academic growth curve trajectories from first to twelfth grades: Effects of multiple social risk and preschool child factors. *Developmental Psychology, 39*, 777–790.

Gutman, L. M., Sameroff, A. J., & Eccles, J. S. (2002). The academic achievement of African American students during early adolescence: An examination of multiple risk, promotive, and protective factors. *American Journal of Community Psychology, 30*(3), 367–399.

Keller, M. B., Beardslee, W., Lavori, P. W., Wunder, J., Dils, D. L., & Samuelson, H. (1988). Course of major depression in non-referred adolescents: A retrospective study. *Journal of Affective Disorders, 15*, 235–243.

Kessen, W. (1979). The American child and other cultural inventions. *American Psychologist, 34*, 815–820.

Loeber, R., Wung, P., Keenan, K., Giroux, B., Stouthamer-Loeber, M., Van Kammen, W. B., et al. (1993). Developmental pathways in disruptive child behavior. *Development and Psychopathology, 5*, 103–133.

Luthar, S. S., & Zigler, E. (1991). Vulnerability and competence: A review of research on resilience in childhood. *American Journal of Orthopsychiatry, 61*, 6–22.

Main, M., & Goldwyn, R. (1984). Predicting rejection of their infant from mother's representation of her own experience: Implications for the abused and abusing intergenerational cycle. *Child Abuse and Neglect, 8*, 203–217.

Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology, 22*, 491–495.

Masten, A. S., & Garmezy, N. (1985). Risk, vulnerability, and protective factors in developmental psychopathology. In B. B. Lahey & A. E. Kazdin (Eds.), *Advances in clinical child psychology* (Vol. 8, pp. 1–52). New York: Plenum.

Maxwell, L. E. (2009). Chaos outside the home: The school environment. In G. W. Evans & T. D. Wachs (Eds.), *Chaos and its influence on children's development: An ecological perspective* (pp. 117–136). Washington, DC: APA Books.

McKenzie, M. J., & McDonough, S. C. (2009). Transactions between perception and reality: Maternal beliefs and infant regulatory behavior. In A. Sameroff (Ed.), *The transactional model of development: How children and contexts shape each other* (pp. 35–54). Washington, DC: American Psychological Association.

Meaney, M. J. (2010). Epigenetics and the biological definition of gene X environment interactions. *Child Development, 81*(1), 41–79.

Morrison, F. J., & Connor, C. M. (2009). The transition to school: Child instruction transactions in learning to read. In A. Sameroff (Ed.), *The transactional model of development: How children and contexts shape each other* (pp. 183–201). Washington, DC: American Psychological Association.

NICHD Early Child Care Research Network. (2004). Trajectories of physical aggression from toddlerhood to middle childhood. *Monographs of the Society for Research in Child Development*, *69*(4), Whole No. 278.

Nichols, P. (1984). Familial mental retardation. *Behavior Genetics, 14*, 161–170.

Patterson, G. R. (1986). Performance models for antisocial boys. *American Psychologist, 41*, 432–444.

Raver, C. C. (2004). Placing emotional self-regulation in sociocultural and socioeconomic contexts. *Child Development, 75*, 346–353.

Robins, L. (1978). Sturdy childhood predictors of adult antisocial behaviour: Replications from longitudinal studies. *Psychological Medicine, 8*, 611–622.

Rothbart, M. K. (1981). Measurement of temperament in infancy. *Child Development, 52*, 569–578.

Rutter, M. (1987). Continuities and discontinuities from infancy. In J. Osofsky (Ed.), *Handbook of infant development* (2nd ed., pp. 1256–1296). New York: Wiley.

Rutter, M., & Garmezy, N. (1983). Development psychopathology. In E. M. Hetherington (Ed.), *Carmichael's manual of child psychology: Vol. 4. Social and personality development* (pp. 775–911). New York: Wiley.

Rutter's, M. (1979). Protective factors in children's responses to stress and disadvantage. In M. W. Kent & J. E. Rolf (Eds.), *Primary prevention of psychopathology Vol. 3: Social competence in children* (pp. 49–74). Hanover, NH: University of New England Press.

Sameroff, A. J. (1995). General systems theories and developmental psychopathology. In D. Cicchetti & D. Cohen (Eds.), *Manual of developmental psychopathology* (Vol. 1, pp. 659–695). New York: Wiley.

Sameroff, A. J. (2006). Identifying risk and protective factors for healthy youth development. In A. Clarke-Stewart & J. Dunn, J. *Families count: Effects on child and adolescent development* (pps. 53–76). Cambridge, UK: Cambridge University Press.

Sameroff, A. J. (Ed.). (2009). *The transactional model of development: How children and contexts shape each other*. Washington, DC: American Psychological Association.

Sameroff, A. J. (2010). A unified theory of development: A dialectic integration of nature and nurture. *Child Development, 81*, 6–22.

Sameroff, A. J., Bartko, W. T., Baldwin, A., Baldwin, C., & Seifer, R. (1998). Family and social influences on the development of child competence. In M. Lewis & C. Feiring (Eds.), *Families, risk, and competence* (pp. 1161–1183). Mahwah, NJ: Lawrence Erlbaum Associates.

Sameroff, A. J., & Chandler, M. J. (1975). Reproductive risk and the continuum of caretaking casualty. In F. D. Horowitz, M. Hetherington, S. Scarr-Salapatek, & G. Siegel (Eds.), *Review of child development research* (Vol. 4, pp. 187–244). Chicago: University of Chicago.

Sameroff, A. J., & Emde, R. N. (Eds.). (1989). *Relationship disturbances in early childhood: A developmental approach*. New York: Basic Books.

Sameroff, A. J., & Fiese, B. H. (2000). Transactional regulation: The developmental ecology of early intervention. In J. P. Shonkoff & S. J. Meisels (Eds.), *Early intervention: A handbook of theory, practice, and analysis* (2nd ed., pp. 135–159). New York: Cambridge University Press.

Sameroff, A. J., Seifer, R., Baldwin, A. L., & Baldwin, C. A. (1993). Stability of intelligence from preschool to adolescence: The influence of social and family risk factors. *Child Development, 64*, 80–97.

Sameroff, A. J., Seifer, R., & Bartko, W. T. (1997). Environmental perspectives on adaptation during childhood and adolescence. In S. S. Luthar, J. A. Barack, D. Cicchetti, & J. Weisz (Eds.), *Developmental psychopathology: Perspectives on risk and disorder*. Cambridge, MA: Cambridge University Press.

Sameroff, A. J., Seifer, R., & Zax, M. (1982). Early development of children at risk for emotional disorder. *Monographs of the Society for Research in Child Development*, *47*(7, Serial No. 199).

Sameroff, A. J., Seifer, R., Zax, M., & Barocas, R. (1987). Early indicators of developmental risk: The Rochester longitudinal study. *Schizophrenia Bulletin, 13*, 383–393.

Sameroff, A. J., & Zax, M. (1973). Neonatal characteristics of offspring of schizophrenic and neurotically-depressed mothers. *Journal of Nervous and Mental Diseases, 157*, 191–199.

Seifer, R., Sameroff, A. J., Barrett, L. C., & Krafchuk, E. (1994). Infant temperament measured by multiple observations and mother report. *Child Development, 65*, 1478–1490.

Sroufe, L. A. (1990). Considering the normal and abnormal together: Te essence of developmental psychopathology. *Development and Psychopathology, 2*, 335–347.

Sroufe, L. A., Egeland, B., Carlson, E. A., & Collins, W. A. (2005). *The development of the person: The Minnesota study of risk and adaptation from birth to adulthood*. New York: Guilford.

Sroufe, L. A., & Rutter, M. (1984). The domain of developmental psychopathology. *Child Development, 55*, 17–29.

Stattin, H., & Magnusson, D. (1991). Stability and change in criminal behaviour up to age 30. *British Journal of Criminology, 31*(4), 327–346.

Stouthamer-Loeber, M., Loeber, R., Farrington, D. P., Zhang, Q., van Kammen, W., & Maguin, E. (1993). The double edge of protective and risk factors for delinquency: Interrelations and developmental patterns. *Development and Psychopathology, 5*, 683–701.

Visscher, P. M. (2008). Sizing up human height variation. *Nature Genetics, 40*(5), 489–490.

Weisner, T. S. (2002). Ecocultural understanding of children's developmental pathways. *Human Development, 45*, 275–281.

Worthman, C. M. (1993). Biocultural interactions in human development. In M. E. Pereira & L. A. Banks (Eds.), *Juvenile primates: Life history, development, and behavior* (pp. 339–358). New York: Oxford University Press.

Zigler, E., & Hodapp, R. M. (1986). *Understanding mental retardation*. New York: Cambridge University Press.