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Psychopathology as an outcome of development

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Abstract
When maladaptation is viewed as development rather than as disease, a transformed understanding results and a fundamentally different research agenda emerges. Within a developmental perspective, maladaptation is viewed as evolving through the successive adaptations of persons in their environments. It is not something a person "has" or an ineluctable expression of an endogenous pathogen. It is the complex result of a myriad of risk and protective factors operating over time. Key research questions within this framework center on discovery of factors that place individuals on pathways probabilistically leading to later disturbances and factors and processes which maintain individuals on, or deflect them from, such pathways once enjoined. There is an interest in recognizing patterns of maladaptation which, while not properly considered disorder themselves, commonly are precursors of disorder and also in conditions of risk that lie outside of the individual, as well as any endogenous influences. Likewise, there is a focus on factors and processes that lead individuals away from disorder that has emerged, which goes beyond interest in management of symptoms. Finally, many topics that currently are capturing attention in the field, such as "comorbidity" and "resilience," are seen in new ways from within the perspective of development.

How childhood problems and psychological disturbance are conceptualized has a profound influence on research that is conducted and the interpretation of research findings. More than two decades ago Lazare (1973) introduced the idea of "hidden conceptual models" in psychopathology. By conceptual models he meant the frameworks for understanding psychological disorder, the set of guiding assumptions utilized by clinicians and researchers to make sense out of their observations of disturbed behavior, thought, and affect. By using the term "hidden," he underscored the fact that such assumptions often are not made explicit *as assumptions* and that people often are not aware that such models are being employed, that is, that they are viewing the world from a particular perspective. One major consequence of such hidden models is that investigators may treat assumed meanings of observations as factual and may fail to recognize powerful and compelling alternative interpretations. Another consequence is that limitations of the model, for example how it constrains research questions, may be obscured.

Embracing a particular model of disturbance is analogous to putting on lenses which may bring some issues or questions into focus while distorting others in ways that may not be obvious to the observer. The thesis of this paper is that what will be called a developmental model leads to a unique and at times radically different view than the position Lazare referred to as the "medical model," a view in which disorders often are seen as discrete and as arising from singular, endogenous pathogens. While not always obvious, this medical model remains a dominant influence in the field, even though in its simple form it is outmoded in much of medicine itself (Rutter, 1996).

Within the classic medical model an analogy is made between childhood behavioral

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and emotional problems and organic disease. This principal guiding assumption has sweeping implications. It is reflected in description and conceptualization of disorder itself, in the nature of research questions that are given priority (centered on endogenous factors) and in how research findings are interpreted. One manifestation of this model is the diagnostic classification system of disorders proposed by the American Psychiatric Association (American Psychiatric Association, 1994). Problems shown by children as well as adults are grouped into disorders, considered to be discrete and distinctive, and often given names suggesting that they reflect enduring conditions of the individual. For example, the category *attention deficit hyperactivity disorder* not only provides the important function of summarizing an array of problem behaviors, but it also implies (via *deficit*) an endogenous problem of the child. This is no mere oversight of terminology. It follows directly from using the organic disease analogy for considering behavioral and emotional disturbance. Environmental factors may be viewed as playing a role, as they do of course in many medical conditions, but core aspects of etiology are assumed to lie in neurophysiological pathology, whether due to genetic defect or environmental pathogens. Likewise, medical treatments are emphasized in research and clinical practice. Environmental manipulations may have some role, but they are seen in terms of managing "symptoms," not as efforts to transform the child's adaptation or to alter the larger child–environment system. All of this is despite the fact that there is little empirical evidence that these children have an attention *deficit* at all (Taylor, 1994). Without the unacknowledged disease assumption, the term deficit would have no place in the description of this set of problems.

Within a developmental model, in contrast, organism and context are viewed as inseparable (see Cohen & Stewart, 1994). There is no attempt to explain behavior as merely an expression of underlying, endogenous neurophysiological differences. Behavioral and emotional disturbance is viewed as a developmental construction, reflecting a succession of adaptations that evolve over time in accord with the same principles that govern normal development. Just as personality or the emergence of competence involves a progressive, dynamic unfolding in which prior adaptation interacts with current circumstances in an ongoing way, so too does maladaptation or disorder.

Sroufe and Rutter (1984) presented some of the guiding assumptions of a developmental perspective, including holism and directedness (see Santostefano, 1978). Thus, meaning of behavior is inseparable from its context and the influence of one factor (an experience, a stressor, a genetic variation) is dependent on the other factors. It is the unique combination of risk and protective factors that governs the emergence of maladaptation (see Cicchetti & Tucker, 1994; Gottlieb, 1991; Rutter, 1996; Sameroff & Fiese, 1989). Moreover, with development the individual plays an increasingly active role in adaptation, interpreting and creating experience as well as responding to external and internal changes. From a developmental point of view behavior is not simply the interaction of genes and environment but genes, environment, and the history of adaptation to that point (Sroufe & Egeland, 1991). This neglected third factor, prior adaptation, is of profound importance and deserves a central place on the research agenda.

One may argue that a caricature of the medical model is being presented and that the contrast with the developmental model is overdrawn. Indeed, Rutter (1996) has argued that in internal medicine multifactorial causation is seen as the rule and that risk factors may be dimensional as well as discrete. Rutter provides numerous examples of complex causality in physical ailments, with environmental factors and context playing a powerful role, interacting with endogenous factors. Thus, the classic medical model has been supplanted in much of medical research itself. Moreover, multiple causality is widely embraced by researchers of diverse persuasions in the study of psychopathology, and environmental context can be considered even when organic factors are emphasized. Developmental history also can be considered, as in, for example, the distinction between good and poor premorbid schizophrenia. An emphasis

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on endogenous factors can be integrated with a variety of other positions, using the concept of development (Eisenberg, 1977; Lazare, 1973; Rutter, 1980). It need not be so narrowly construed.

Nonetheless, the position here is that the medical model (henceforth referred to as the *classic* medical model or *disease* model), though outmoded even in medicine, still exercises a dominant influence in the field of child psychopathology. Because the assumptions underlying this classic model are often unreflectively accepted and not explicitly acknowledged, it exercises a pervasive, if often subtle, influence on the conduct and interpretation of research. Claims for the importance of a broad causal net, and for an emphasis on process, may be the mode, but in reality priority is often still given to the search for particular endogenous pathogens of a disorder. Environmental as well as endogenous influences on child psychopathology may be examined. But "environment" often refers to prenatal teratogens or lead-painted walls (which are, of course, surrogates for endogenous influences), as though these are broadly representative of exogenous factors. Also, as discussed in the section on research below, physiological concomitants of disturbance are routinely interpreted as causes, rather than simply as correlates or markers. The disease model takes on the status of *the* description of reality rather than as one point of view, which may in fact often be distorted. Before turning to further examples of the still prevailing influence of the classic medical model at the expense of alternative points of view, an elaboration of a contrasting developmental position will be presented.

The Pathways Framework

Many implications of a developmental model of disturbance can be captured with the concept of developmental pathways, introduced by Waddington (1957) and adapted by Bowlby (1973). Bowlby's preferred metaphoric representation of the pathways concept is the continuous branching of tracks in a railway train yard (see also Loeber, 1991), but it may be pictorially represented as a tree as well (see Fig. 1). Pathology may be thought

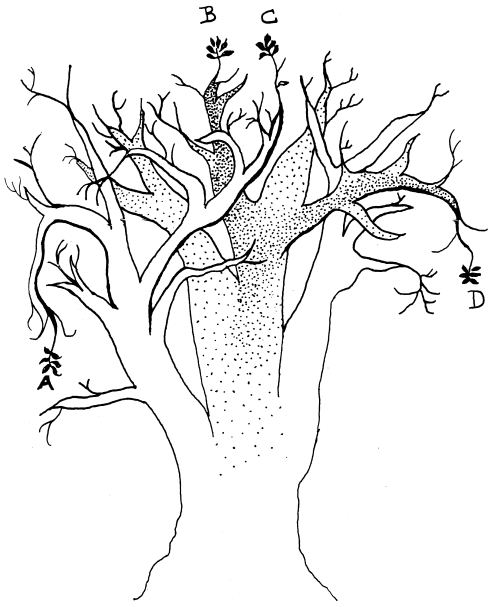


Figure 1. A schematic representation of the developmental pathways concept. (A) Continuity of maladaptation, culminating in disorder. (B) Continuous positive adaptation. (C) Initial maladaptation followed by positive change (resilience). (D) Initial positive adaptation followed by negative change toward pathology.

of as a succession of branchings which take the child away from pathways leading to competent functioning. Five major implications of this model may be summarized as follows (see also Sroufe, 1989; Sroufe & Rutter, 1984; Sroufe, Egeland, & Kreutzer, 1990).

1. *Disorder as deviation over time*: Pathology is viewed in terms of developmental deviation. This requires first an understanding of normative developmental issues (e.g., secure attachment, modulated impulse control, effective entry into the peer group) and the various patterns of positive adaptation with respect to them. A significant deviation in pattern of adaptation represents an increased probability of problems in negotiating subsequent developmental issues. Pathology generally reflects repeated failure of adaptation with respect to these issues. A particular adaptational failure at any point in time is best viewed as placing an individual on a pathway potentially leading to disorder or moving the individual toward such a pathway. Thus, for example, maladaptive patterns of attachment in infancy (anxious

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attachment) are not viewed as psychopathology per se but in terms of developmental risk for disturbance (see below). Pathology involves a succession of deviations away from normative patterns.

2. *Multiple pathways to similar manifest outcomes*: When development is viewed in terms of a succession of branchings, it follows that individuals beginning on different pathways may nonetheless converge toward similar patterns of adaptation. Different influences and different courses may be germain for different individuals (see Cicchetti & Rogosch, 1996). A pattern of maladaptation with many features in common (e.g., lack of social engagement, depressed mood, low self-esteem) may be the result of distinctly different developmental pathways, one rooted in alienation, and one rooted in anxiety and helplessness (see also Blatt, 1995). If so, quite different interventions may prove helpful to members of these two groups, and it may be inappropriate to employ the same label to describe them, despite similar manifest behavior. Whether such phenotypically similar individuals differ in terms of "prognosis," subsequent outcome, or effective intervention become key research questions (e.g., Moffitt, 1993).

3. *Different outcomes of the same pathway*: Similarly, the concept of successive branchings suggests that individuals beginning on a similar pathway may diverge, ultimately showing different patterns of pathology (or positive adaptation) (cf. Cicchetti & Rogosch, 1996). Despite the phenotypic dissimilarity of such outcomes, it remains possible that they will represent a coherent family. The study of branching pathways over time may suggest radically different approaches to classification, based on developmental trajectory rather than final manifest behavior alone (Loeber, 1991; Thelen, 1990).

4. *Change is possible at many points*: Despite early deviation, changes in developmental challenges or other aspects of context may lead the individual back toward a more serviceable pathway. Not only is pathology typically not simply an endogenous given, but even when a maladaptive pathway is enjoined, return to positive functioning often remains possible. It is generally inappropriate to think of maladaptation or disturbance as something a child either "has" or "does not have" in the sense of a permanent condition. Within this perspective, extremely stable conditions such as early emerging conduct disturbance call for research on supports for such problems, centering on positive feedback cycles between child maladaptation and environmental reaction (Patterson, DeBarysh, & Ramsey, 1989; Richters & Cicchetti, 1993).

5. *Change is constrained by prior adaptation*: This final implication somewhat counterbalances the fourth. It suggests that the longer a maladaptive pathway has been followed (especially in the sense of going across phases of development), the less likely it is that the person will reclaim positive adaptation. (Bowlby implied that adolescence might mark the end of relative flexibility.) This is consistent with the "active child" principle. By creating negative experiences in an ongoing way, failing to engage positive opportunities, and interpreting even benign experience as malevolent (which often are core features of maladaptation), the child's adaptation may make positive change less likely. Dodge's work on attribution no longer is interpreted in terms of inherent cognitive deficit, but does suggest that negative experiences of some children lead to interpretive frameworks (and congruent behavior) that further lead to negative experiences and so on (Crick & Dodge, 1994; Rieder & Cicchetti, 1989; Rogosch, Cicchetti, & Aber, 1995; Suess, Grossmann, & Sroufe, 1992). This proposition also is in accord with abundant empirical data, including the finding that children who enjoin early, and consistently pursue, the path from defiance to aggression to antisocial behavior are highly likely to persist toward criminality (Loeber et al., 1993; Moffitt, 1993).

Like any metaphor, this branching pathway model has its limitations, implying for example that certain sorts of outcomes would be absolutely impossible for some individuals, whereas at this stage of our knowledge "improbable" would seem more likely. Still, it does provide a useful framework for summarizing a great deal of information about development. It also provides a distinctive alternative for interpreting findings from research on

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childhood disturbance and suggests an important research agenda which hitherto has been largely neglected, namely, processes of initiation, continuity, and change in maladaptation.

Some distinctions between this presentation of the developmental pathways concept and other recent discussions should be pointed out. Loeber (1991), for example, has nicely summarized a set of ideas somewhat parallel to points 3–5 above. However, for him the starting point of a "pathway" is the presence of disordered behavior patterns. The pathway is defined by the problem behaviors, and the focus is on persistence and desistance of these. Other problem behaviors that are present or "emerge" are subsumed within the concept of "comorbidity" (discussed below) and viewed as influences on subsequent development (p. 107). An important contribution of the present pathways model is emphasis on patterns of adaptation (with respect to normative developmental issues) that precede the emergence of frank disturbance, that include strengths as well as weaknesses, and that are viewed as an important part of a causal network of influences. Early patterns of adaptation are viewed as prototypes—root forms with diverse potential—that are linked to later outcomes as part of a multidetermined process. Some individuals on a pathway may, in fact, never go on to disorder. The work of Loeber, and other work that he discusses, is important in indicating that early onset, duration, and number of problems are of great prognostic significance. However, to provide a fully viable and distinctive alternative to the classic medical model, developmental pathways must be traced from a point prior to the onset of disturbance.

Tracing pathways from a point prior to the emergence of disturbance allows one to discover heterogeneity in disorder. Individuals showing similar "symptoms" may in fact be on different pathways if examined longitudinally and may have predictably different outcomes. From the viewpoint of development, they may not be manifesting the same disturbance. This has been illustrated by Moffitt (1993) in work on "adolescent limited" and early emerging antisocial behavior. Those whose problem behavior emerged first in adolescence are not on the same pathway as those whose behavior arises in, and persists from, early childhood. Only the latter are likely to show criminality in adulthood. Thus, despite the overlapping conduct problems in adolescence, these two groups should not be treated as members of one disease entity group. Likewise, those showing the problem cluster in question, plus particular patterns of other problems, may again be distinguished by antecedent and subsequent development. They may not be on the same pathway. (As a matter of internal consistency with regard to the pathway metaphor, it certainly makes no sense to say that individuals may be on two separate pathways at once, one in common with another group and one distinctive.) Starting with symptoms to define a pathway simply accepts that the medical model-based classification system is valid, leading inevitably to additional problems being interpreted as the cooccurrence of second diseases and differential persistence as due to varying time courses of the disease. The critical research questions regarding developmental process that arise from these observations are simply swept aside. The existing disease-based classification system, and the classic medical model of psychopathology in general, need to be tested, not simply assumed as the starting point for studying problem behaviors over time.

Conceptualizing Competence and Disturbance

Varying conceptualizations of basic phenomena in the field reveal the operation of different models. Two examples are considered here, one from the domain of competence and one from the domain of disturbance. The first phenomenon to be discussed has been termed "resilience" and the second comorbidity. That the medical model is frequently in operation in discussions of the former, as well as the latter, is testimony to its pervasiveness.

Resilience

The concept of resilience can be used to illustrate the distinctiveness of the developmental

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perspective. Resilience simply refers to the fact that some children facing adversity nonetheless do well (or return to positive functioning following a period of maladaptation; e.g., Masten & Coatsworth, 1995). Such an observation is open to a variety of interpretations. This observation often is explained in terms of endogenous traits (an inherent robustness or other such characteristic of the child). As is often done in discussing disorders, the term "resilience" is therefore made the explanation for the observed phenomenon. Why do some children do well in the face of adversity? Because they are resilient. (Why do some children manifest attention problems? Because they have Attention Deficit Hyperactivity Disorder, ADHD.) Thus, in this perspective, resilience is treated as a trait rather than as a process. So powerful is such a preconception that ambiguous data is often interpreted as implying such an endogenous trait. The well known work of Werner and Smith (1992) is frequently cited as demonstrating that positive "temperament" is a determinant of resilience. The significant variable underlying this interpretation, which shows up only late in infancy, actually is a parental report of the degree to which the child is "lovable." Not only could this variable readily be interpreted as a caregiving variable (caregiver *perception* of loveableness), but also the idea that loveableness itself is a developmental product is not considered. Only when this finding is uncritically interpreted within a classic medical model framework would a trait interpretation automatically follow from this finding.

Within a developmental perspective, in contrast, resilience is not something some children simply "have a lot of." It develops. A capacity to rebound following periods of maladaptation (or to do well in the face of stress) evolves over time within the total context of developmental influences. The capacities for staying organized in the face of challenge, for active coping and for maintaining positive expectations during periods of stress are evolved by the person in interaction with the environment across successive periods of adaptation. And even as an acquired capacity it is not static but is continually influenced by ongoing changes in context. Prospective, longitudinal data (e.g., Egeland, Carlson, & Sroufe, 1993) reveal that manifestation of resilience is associated either with a history of positive experience and positive adaptation (prior to the period of stress or maladaptation) and/or positive experience between the period of stress and recovery. For example, groups of malfunctioning 4-year-olds who later were free of behavior problems at school more often had histories of early secure attachment and stable emotional support in the toddler period than did 4-year-olds who showed continuity of malfunctioning (Sroufe et al., 1990). Had the research started at age 4, the resilience would have appeared mysterious and may have been attributed to some children simply having the "right stuff." From within the classic medical model the search for antecedents of resilience (with the exception of IQ or temperamental traits) has had low priority.

Other research from our project shows that changes in parental stress and social support also account for differential improvement in children's functioning over time (e.g., Egeland et al., 1993). This graphically illustrates that resilience resides more in the developmental system (which, of course, includes the child's history of adaptation) than in the child alone.

In a completely parallel manner, understanding desistance of problem behavior will be enhanced employing a developmental model. Both adaptational history and current supports and challenges are needed to explain those whose disordered behavior improves. "Spontaneous recovery" is just a term for current ignorance, left unexamined because of weddedness to a particular disease model of disorder.

Comorbidity

The phenomenon to which comorbidity refers is the simple fact that children (especially) often show behaviors that fit two or more of the currently designated diagnostic categories (see Caron & Rutter, 1991, for a discussion). Saying that such joint occurrences are a reflection of comorbidity is no explanation. Nothing in this observed fact suggests that children therefore often have concurrently

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two or more discrete disorders (read, *diseases*). But the term comorbidity, based in the hidden assumptive network of the disease model, suggests just that. If a child manifests problems that fit two current categories, it is concluded (assumed) that he or she has two conditions, rather than even considering the alternative that there is a failure of syndromic integrity for one or both categories. The power of the hidden assumptions leads investigators to skip right over the challenge the basic observation poses to the classic medical model.

The disease model requires syndromic integrity. If the disease model is apt for children's behavioral and emotional problems, children generally should manifest tight clusters of symptoms, with unique indicators of other syndromes being absent. But in reality children commonly manifest problems that cut across established categories. To be sure, one disorder may potentiate another in medicine as well (Rutter, 1996), but not nearly to the extent implied by the prevalence of comorbidity of childhood disturbances. Descriptions of disorders in the literature frequently begin by noting large percentages of overlap with other conditions (e.g., Rutter, Taylor, & Hersov, 1994). For example, Harrington (1994) reports that most children who meet criteria for depression also have been given another primary diagnosis. Citing other work (Anderson, Williams, McGee, & Silva, 1987), he also reports that of 14 11-year-olds with depression, 11 qualified for at least one other diagnosis, 8 of the 14 qualifying for anxiety disorder, attention deficit disorder, *and* conduct disorder. Conduct problems and activity/attention problems have been found to correlate quite highly (e.g., .77; August, MacDonald, Realmuto, & Skare, 1996). Comorbidity is the rule, not the exception. Moreover, broad classes of problems such as externalizing behaviors are predictive of a myriad of later conditions, including depression and other conditions not typified by aggression or other hallmarks of externalizing (Robins & Price, 1991). Number of problems rivals clustering of problems in predicting later dysfunction.

One might think the discovery that children's problems often cut across the working categories would have led to a questioning of the entire system, not to a new medical term in our reference books. Because of the power of the medical models, the literature contains almost no discussion of concerns about basic tenets underlying the DSM classification system itself (for recent exceptions, see Richters & Cicchetti, 1993; Wakefield, 1992a, 1992b). Rather, discussion focuses on category changes alone (Rutter et al., 1994). If taken seriously, the data on comorbidity could lead to revolutionary changes in classification of childhood psychological problems and perhaps quite distinctive views of disturbance itself. The way would be cleared for evolving classification schemes centered on patterns of adaptation and developmental trajectories.

Designing and Interpreting Research

The pervasiveness of the medical model not only has implications for conceptualization (and treatment) of childhood problems but also has a major impact on research. It powerfully guides the questions that are asked as well as how obtained findings are interpreted. Much current research is focused on finding *the* pathogen for a given problem—the gene or the particular neuropathology assumed to underly all instances of a disturbance. It follows that this search commonly is localized in the person, and the assumption is made that the pathogen by itself accounts for the origin, onset, and course of the problem ("disorder"). Nothing, of course, is wrong with neurophysiologically oriented research. At this stage of our knowledge of maladaptation, research on numerous fronts is vital. However, singular devotion to the disease model, with its hidden assumptions, has the unfortunate consequence of limiting and narrowing the research endeavor. When it is taken as a given that *disorder* derives from *pathogens* that are *endogenous* to the individual there will be limited efforts to discover etiological factors lying outside of the child (and to understand how these interact with endogenous factors) or, perhaps especially, to understand what factors may bring the child back toward normal adaptation away from a disturbed pattern. Examining factors that lead a child into or away from

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maladaptation is not even a very meaningful issue if disorder is considered something an individual either has or does not have.

Within a developmental perspective the research agenda changes dramatically from that inspired by the disease model, and existing data frequently are seen in different light. One moves away from the search for single pathogens, conceptualized as linear causes ineluctably producing their outcome, toward the search for a complex of influences that initiate a developmental pathway which only probabilistically is associated with disturbance (Cicchetti & Tucker, 1994; Sameroff & Fiese, 1989; Sroufe, 1989). The etiology of disturbance is conceptualized in terms of a combination of risk factors and protective factors of diverse sorts. Moreover, the possibility or even probability of later disturbance may be seen in early patterns of maladaptation that in and of themselves are not pathological and in aspects of the developmental context even prior to the appearance of child maladaptation. Second, and equally important from this perspective, is research on factors influencing continuity and change, that is, processes and mechanisms that maintain individuals on pathways once enjoined or deflect them toward others. This includes the search for factors that lead individuals away from disturbance following its manifestation. Disturbance is not a given; it is supported. Pathology is not something a child "has"; it is a pattern of adaptation reflecting the totality of the developmental context to that point.

When disturbance is viewed as development one asks numerous questions. How do individual children get off track? When going off track, what deviating track is a particular child likely to take? What influences (in their pattern of adaptation and in the total developmental context) tend to maintain them on the track they are on, and what would be required to bring them back to a more serviceable developmental pathway? These are very different than questions about which gene causes, or what are the physiological correlates of, a particular disorder, which are inspired by the classic medical model and shed limited light on most childhood problems.

Two illustrations from the Minnesota longitudinal study of development from birth through adolescence (e.g., Egeland et al., 1993; Carlson, Jacobvitz, & Sroufe, 1995; Sroufe, Carlson, & Shulman, 1993) can illustrate the heuristic value of this viewpoint and its distinctiveness from the disease model. The first comes from a prospective, longitudinal investigation of children's attention and activity problems, using criteria of ADHD in DSM III–R. The second is based on an adolescent outcome study of infant–caregiver attachment problems.

A developmental view of attention and activity problems

The starting point for a developmental approach to psychopathology is always a consideration of normal development. Thus, we began our investigation of attention/activity problems by considering factors that normally support the development of the capacities to modulate arousal, regulate affect, control impulses, and direct attention. Basically, a process unfolds wherein what begins as caregiver-orchestrated regulation becomes dyadic regulation, with increasingly active participation by the infant. Then, progressively, transfer of the regulatory responsibility to the child occurs over the course of early childhood through a series of phases. At each phase, beginning in the early months of life, patterns of affective, attentional, and behavioral regulation are constructed within the caregiving system. Such developing patterns or *prototypes* are carried forward and interact with subsequent challenges to regulation as development continues (see Sroufe, 1989, for more detail). Given this understanding, we then asked what factors would be liabilities with respect to pursuing the normative pathway toward effective self-regulation. What might lead some children to get off track?

The data set was comprehensive, and a range of factors were considered. We examined a number of early "child" variables, that is, variables commonly thought of as residing in the child. These included premature birth, nonoptimal newborn neurological status, nurses ratings of fussiness, soothability, and other behaviors in the newborn nursery; ob-

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servational measures of infant activity level and irritability; and parent-based temperament questionnaire data in infancy and at age 2½ years. While we believe that each of these variables is best thought of as reflecting a developmental process, it is the case that most of them (the exception being the parental reports) can be defined as child characteristics. They are manifest in child behavior, observable even when the child is apart from the caregiver.

But in addition to these child characteristics, which along with environmental toxins often would exhaust variables in a study guided by a disease model of disorder, we also examined aspects of the developmental context. This included the immediate context of parenting behaviors (patterns of stimulating and regulating the child), the broader context in which parenting was nested (the stress, support, and general degree of stability in the parent's life), and more distal contextual factors, such as marital status at birth. No prior study had explored the origins of attention and activity problems in this way, though from a developmental perspective it is obvious to do so.

The results of this research, based on following some 180 children from birth through sixth grade and using teacher Behavior Problem Checklist data as the outcome, strongly supported the heuristic value of a developmental perspective (Jacobvitz & Sroufe, 1987; Carlson, Jacobvitz, & Sroufe, 1995). The more than 40 early child variables were consistently weak in terms of predictive power. One variable from the Brazelton Neonatal Exam (Motor Immaturity) showed modest prediction of ADHD criteria in kindergarten, but not thereafter. Observed or parent-reported activity level or other dimensions of infant temperament were never significantly related to subsequent attention or activity problems. In contrast, measures of parental intrusiveness and overstimulation, including the single measure of such parenting obtained when the infant was 6 months old, were more predictive, with some consistency across ages. It is important to note that parental intrusiveness at 6 months was not predicted by any antecedent or concurrent child variable. Thus, we view this influence as initially lying outside of the child. Moreover, the single best predictor of attention problems was mother's relationship status at birth; children later showing attention and activity problems had single mothers. Such a contextual feature cannot be attributed to the child and shows the importance of casting a broad net in defining factors that place children on pathways to disorder. With regard to the prediction of attentional and hyperactivity problems in kindergarten, we found that there was almost no overlap between those few cases that were predictable from newborn motor immaturity and the others that were predicted from the parenting and other contextual variables (Jacobvitz & Sroufe, 1987). Thus, multiple pathways to the same disturbed behavior are suggested.

While nothing observable in the child during the infancy period was found to predict later attentional and hyperactivity problems, by age 3½ this no longer was so. Consistent with other literature (e.g., Campbell, 1990), our observation-based rating of distractibility was modestly related to ADHD criteria behaviors in early elementary school (accounting for about 6% of the variance). By the preschool period, then, one might say that some children are on the attentional problem/hyperactivity pathway, even though enjoining this pathway (the 3½ year distractibility measure) is predictable from contextual variables well before this time, as is later criterial ADHD behavior itself. Moreover, a combination of distractibility and early and later contextual variables predicted elementary attention problems far more strongly than early distractibility alone (up to 28% of the variance in Grade 1–3 problem behavior).

In the second phase of the research we showed that contextual variables accounted for change in ADHD criterial behaviors over time. Changing support for caregivers and changing caregiver relationship status were the most consistent predictors of change in child problem behaviors. As the primary caregiver's relationship stabilized or destabilized, the child's manifestation of attentional and hyperactivity problems changed. Thus, in the current developmental terminology, some children who were on the ADHD pathway at

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ages 3½, 5, or 6 were apparently not on this pathway at a later age, whereas others not manifesting such problems early had enjoined this pathway at a later age. More detailed, process data will be required to determine whether such change is mediated primarily by change in the caregiver's behavior toward the child, as we would hypothesize.

A final result is relevant to the pathways model. When cumulative attentional and hyperactivity problems up through third grade are considered, very little change can be accounted for thereafter. This suggests that, at least for these types of externalizing problems, change becomes increasingly difficult the longer the pathway is followed. This also seems to be true for aggression (Gottesman, 1995; Loeber et al. 1993; Moffitt, 1993).

Reactions to this work during conference discussions and in editorial review were interesting with respect to the role of models in research evaluation. The first reaction typically has been to ask how many of our subjects "really had" ADHD? This question, steeped in the disease model, presumes the distinct entity, organically based nature of such problems. Taken to extreme this would preclude scientific investigation. If an organic variable is not predictive (or if medication is ineffective long term), then this is taken as evidence that the children in question did not have ADHD. The alternative of a continuum of problem behaviors is simply not taken as a viable position. We found no evidence for discontinuity in the distribution (univariate or bivariate) of our variables, nor is there a body of evidence suggesting the 8 (DSM III–R) or 6 (DSM–IV) "symptoms" represent a qualitative break point (Jacobvitz, Sroufe, Stewart, & Leffert, 1990). We obtained results parallel to those above looking at extreme cases and at the 12 children placed on stimulant medication (which itself bore little relation to our objective assessment of behavior across time—a sad commentary on at least some clinical practice).

Other reactions suggested that the questions addressed by our work did not make sense or dealt with resolved or superfluous issues. Environmental factors, and even family factors in particular, were said to require no further attention, and citations were provided. However, "environmental factors" often referred to toxins such as lead. Within a medical model, of course, these are the kind of environmental factors that command attention, rather than psychosocial stressors and other aspects of developmental context which might also be considered. Moreover, "family" variables, it was argued, had been shown to be irrelevant or to be effects, not causes. A cited example of the former was Goodman and Stevenson's (1989) twin study. But their family data were based on contemporaneous parent interviews which not surprisingly yielded no predictability; there was no observation of caregiver behavior, antecedent or contemporary. Weak measures are quickly accepted when the null hypothesis follows from tacit assumptions about factors that are irrelevant. Another example is a study by Schachar and Wachsmuth (1990), which could be cited as showing a lack of family influence on ADHD (Taylor, 1994). Schachar and Wachsmuth simply examined DSM diagnoses of parents, finding no increment in disorders among parents of ADHD cases compared to parents of controls (though there was an increase for conduct disorder cases). Such a family variable follows from a medical model, given a preoccupation with genetic causality. However, it is not parental psychiatric diagnosis, but patterns of stimulation, control, and dyadic regulation that are critical within a developmental perspective. These were not assessed. A study by Hinshaw and McHale (1991) was cited as an example of research showing that parenting differences are effects and not causes. These authors reported that parent controllingness decreased when children with attention problems were given stimulant medication, which, as we will discuss below, is not relevant to the question of etiology. Finally, many reviewers and discussants of this work, and researchers in general, have argued that it has already been proven that attentional problems are "largely the result of neurological dysfunction" (e.g., Frick & Lahey, 1991). Such a conclusion has been based on descriptions of the syndrome, the presumed "lack" of evidence for parenting and other contextual influences, short-term re-

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sponsiveness of the disorder to stimulant medication, and occasional neurophysiological correlates. One highly acclaimed example of the latter is Zametkin's (1993) report of frontal lobe blood flow differences in adults presumed to have been ADHD as children, compared to control subjects. As will be discussed below, such a correlation cannot be interpreted as causal. All of this reflects interpretation of information within a medical model, without consideration of compelling alternatives.

When the medical model lens is removed and the literature on etiological factors in attention problems is considered from within a developmental perspective, very different interpretations result. With respect to parent behavior, for example, cause is not looked at in simple, linear terms. Of course, managing a child with attention problems is extraordinarily difficult, regardless of what is ultimately understood regarding etiology. It also would seem natural for parents to be controlling and even critical of a child having such problems, and the literature contains such findings (Taylor, 1994). If with intervention child attention problems decrease, one would expect controllingness to decline. Ongoing, mutual influence is the basic expectation within a developmental process model. However, such a finding in no way suggests that caregiving factors are irrelevant to etiology. It would not be hypothesized that parental overcontrol would lead to hyperactivity, so the fact that overcontrol declines with diminished child problems is not germain to this issue. Our prospective, longitudinal assessments revealed no infant predictors of parental intrusiveness or overstimulation. At the same time, *these* parenting patterns predicted later attention and activity problems. How child factors interact with such parenting variables has as yet been little explored.

Central nervous system correlates also would be expected within an integrative developmental framework. First of all, many of the data utilizing brain physiology or blood flow, such as the findings of Zametkin (1993), are gathered during attentional tasks. The measures therefore simply corroborate the attention problem. That lifelong attention problems would in adulthood be manifest both in behavior and in CNS functioning is no surprise, given the integrated nature of human functioning. However, this in no way allows the conclusion of innate damage or even "dysfunction" in the sense of aberrant behavior–brain linkages. Such measures thus have the status of markers but not necessarily causes. The leap to equate correlation with cause is a reflection of a commitment to the medical model. Moreover, even were dysfunction in brain functioning shown to be antecedent to the emergence of attention and activity problems, which certainly has not been done yet, this still would be best interpreted within a broader causal framework. The recent outpouring of evidence concerning experience dependent brain development (Cicchetti & Tucker, 1994; Greenough, Black, & Wallace, 1987; Kraemer, 1992; Schore, 1994) makes it clear that there are massive experiential influences on the development of the central nervous system, including the tuning of systems concerned with activation and regulation of affect and behavior. Eclectic investigators will look for ongoing parenting influences on endogenous factors as well as endogenous influences on parenting.

Changes in behavior in response to stimulant medication likewise do not allow etiological interpretations. Models of etiology and models of treatment bear no necessary relation to one another. Frankly retarded youngsters may be trained to perform certain cognitive tasks, but no one currently argues that their retardation was the result of insufficient reinforcement. Those who argue that drug studies have etiological significance overlook the fact that stimulants also enhance the performance of normal children and adults and show little evidence of improving the functioning of attention disordered children in the long term (see Jacobvitz et al., 1990). Even were such results ever demonstrated, and even if the effective drugs were those with more specific neurotransmitter actions (in contrast to broadly acting methylphenidate), this still would not prove inherent deficit, due to the complex, systemic nature of development. Central nervous system dysfunction also is best viewed as developing within a complex causal framework.

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A developmental view of infant attachment problems and later disturbance

A second illustration of developmental research on maladaptation concerns the relation of early anxious attachment relationships to later psychiatric problems. Attachment research provides an interesting case for developmental psychopathology, because attachment is a relationship construct, not an individual trait construct. Established assessments of infant caregiver attachment (e.g., the Strange Situation Procedure; Ainsworth, Blehar, Waters, & Wall, 1978) are assessments of relationships, not individuals. This has been supported by ample research, including the findings that attachment pattern with each parent often is different, with concordance barely significant (Fox, Kimmerly, & Schafer, 1991) and that attachment security with a given parent changes as a function of that parent's changing life stress (e.g., Vaughn, Egeland, Waters, & Sroufe, 1979). Clearly, attachment security is not an endogenous infant trait.

Still, patterns of anxious attachment in infancy are proposed to be risk factors for psychopathology. The quality of a particular attachment relationship, whether secure or anxious, is based on the history of interaction within the pair. When the caregiver is routinely responsive to the infant's signals, the infant develops a confidence that reassurance, tending, assistance, and other care will be available when needed. Such confidence in support is precisely what is meant by secure attachment. In contrast, routinely unresponsive or inconsistent care undermines security.

The patterning of the early primary attachment relationship is a prototype for subsequent development, operating on numerous levels (Sroufe, Egeland, & Carlson, in press). In the secure attachment case, having experienced responsive care, the child generalizes the expectation that others will be responsive and available; that is, the child develops generally positive and trusting attitudes toward others. Along with this, the child takes forward a sense of his or her own effectance and personal worth. Being able to effectively elicit responsiveness and care from the parent, they expect to master challenges and to have power in the world. They believe in themselves. Likewise, they value relating and have an internalized template for empathy and reciprocity in relationships.

Patterns evolved in the attachment relationship are taken forward at the behavioral level as well. The child has been entrained into particular patterns of reciprocity and affective sharing, as well as having evolved a sense of curiosity and a skill in exploration, supported by the secure attachment.

Supporting the behavioral level are patterns of arousal regulation, which allow the full range of emotional expression with sufficient modulation, such that organized behavior can be maintained. Such patterns are readily established in the context of responsive care, because responsiveness entails appropriate affective stimulation and interventions to keep arousal within reasonable bounds. Moreover, recent evidence (e.g., Schore, 1994) suggests that a history of patterned, responsive care actually is central in tuning and balancing excitatory and inhibitory systems in the central nervous system itself, which would support emotional regulation and behavioral flexibility.

In addition to those attachment relationships judged to be secure (the clear majority in most samples), there are three patterns of anxious attachment, each of which would compromise the developing capacities for self-regulation and social behavior (Ainsworth et al., 1978; Main & Hesse, 1990; Sroufe, 1988). *Anxious/resistant* attachment is characterized by difficulty settling with the caregiver when distressed, often tinged with anger. Such a pattern is associated with a history of inconsistent care and/or neglect, leaving infants hyperaroused, hypervigilant, and uncertain regarding caregiver availability and their own effectiveness. *Anxious/avoidant* attachment involves explicitly failing to seek contact with the caregiver under conditions of stress (e.g., following brief laboratory separations). This pattern is associated with a history of chronic rebuff, especially when the infant sought physical contact with the caregiver. Such infants learn to cut off or truncate emotional responses, especially when tender needs

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are aroused. Finally, *disorganized/disoriented* attachment reflects confusion about or even fear of caregivers, who themselves have behaved in confused, alarming, or dissociated ways. Such infants face an unresolveable paradox of having caregiver be both the source of alarm and the (biologically) expected source or reassurance. Lapses in orientation and failures of integration of emotions, cognitions, and behavior result.

We recently tested the hypothesis that patterns of anxious attachment represent risk factors for psychopathology across childhood and adolescence. For example, at age 17½ we created an overall index of pathology, based on the number, duration, and seriousness of diagnoses derived from the Schedule of Affective Disorders and Schizophrenia (Child Form) clinical interview, which was conducted and coded completely independent of attachment history or other knowledge of the child. The simple correlation of avoidant attachment at 12–18 months and the pathology index was .24. This is significant with 170 subjects though small in absolute terms. The combination of "disorganized" attachment (Main & Hesse, 1990) and avoidant attachment raised the correlation to .41, modest but impressive over these many phases of development and given the challenges of assessing such constructs. The correlation increased still further (into the high .50s) when we also added measures of parenting and adaptation from the preschool and early adolescent periods (see Carlson, submitted). The disorganized attachment pattern was specifically related to dissociative symptoms in childhood and adolescence (.40), as predicted by theory.

In accord with the developmental model, avoidant and disorganized patterns of attachment may be thought of as initial developmental variations, probabilistically associated with later disturbance. Such patterns of anxious attachment are not thought of as psychiatric disorders themselves (Sroufe, 1988). Again, they are viewed as assessments of relationship qualities with a particular caregiver. Avoidance and disorganized/disoriented attachment, which show little concordance across parenting partners, both are predictable from earlier patterns of care by the particular parent and show stability with each parent (Carlson, submitted; Main & Hesse, 1990). Thus, anxious attachment in infancy is better viewed as an initiating condition than as a characteristic of the infant. Still, as a maladaptive relationships pattern it is probablistically linked to later psychological disorder.

Nor is anxious attachment viewed as causal of later disturbance in a simple sense. After all, as is true of most singular risk factors, the majority of individuals showing early anxious attachment do *not* show serious disturbance later. Whether disturbance results depends on the successive combination of liabilities and supports that maintain the individual on a pathway to pathology or bring them back toward positive adaptation. Of course, one of the liabilities (or supports in the case of secure attachment) is the prior adaptation, including prototypical pattern of coping and affect regulation and expectations concerning self, other, and relationships, within which the person negotiates subsequence developmental phases.

The manner in which attachment theory and research have been utilized within the dominant medical model was predictable. "Attachment disorders" were added to the DSM (APA, 1987, 1994). While the criterion of pathological care as the source of these problems marks a break from the traditional medical model approach, and while the cases so designated may indeed have attachment problems (Zeenah, Mammen. & Lieberman, 1994), the circumscribing of attachment problems to specific disorders reveals a failure to grasp the developmental significance of attachment history and the potential power of a developmental approach to psychopathology in general. What could become a model for approaching childhood disturbance of all kinds is sequestered into a circumscribed set of categories.

Various attachment problems seem to have implications for a range of disturbances, certainly not all phenotypically similar to dyadic behavioral patterns shown in infancy (Blatt, 1995). For example, given the tendency of those in avoidant attachment relationships to turn from their caregivers when in need, later social withdrawal and superficial relation-

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ships might be all that were expected based on linear predictions. But the lack of empathic connection and the alienation inherent in these prototypic avoidant attachments also has been viewed as the basis for aggressiveness, bullying, and conduct disorders, predictions which have been confirmed (Renken et al., 1989; Troy & Sroufe, 1987). On the other hand, resistant attachment often is manifest in angry rejection of the caregiver when comfort is offered to the distressed infant, a pattern which may be shown in batting away toys, pushing away from contact, squirming, and/or tantruming. Yet this pattern is not associated with later Oppositional Defiant Disorder or other externalizing problems. It is uniquely related to Anxiety Disorder classifications, as predicted from the chronic vigilance required to monitor an inconsistent caregiver (Warren et al., submitted).

Conclusion: Future Directions

The classic medical model as a framework for approaching behavioral and emotional problems in childhood has inherent limitations. Childhood problems generally are not like diseases. They show little evidence of a bounded, discrete, syndromic nature. Often children qualifying for diagnosis are quantitatively, not qualitatively, different from other children. Research often shows number of problems rather than tight, syndromic coherence, to be predictive of later disorder. Furthermore, most childhood problems are context malleable to a degree that surpasses typical medical conditions (especially during the years of onset). All of this is much more consistent with the idea of development than the idea of disease.

Exceptions to this general case, such as childhood autism, actually further underscore the importance of a developmental viewpoint. Autism is now classed as a "Pervasive Developmental Disorder," and properly so. Such children are profoundly disturbed in all arenas of functioning—cognitive, affective, and social (Hobson & Patrick, 1995). They are qualitatively different from other children, including those with behavior and emotional problems. Children assigned diagnoses of "attention deficit hyperactivity disorder," "anxiety disorder," and so forth and children not fitting any DSM category are far more similar to each other than they are to children diagnosed as autistic. The manifestation of most childhood disturbances, but not autism, is profoundly influenced by context. For example, in our emotionally supportive, well-staffed (one adult to four children), activity-oriented summer camps, which included a good mix of competent 10-year-olds and children with serious conduct problems, aggression was almost nonexistent (e.g., Elicker, Englund, & Sroufe, 1992). This is despite the fact that in school settings the troubled children were reliably reported by both teachers and observers to engage in frequent bullying and other aggressive behaviors. Such contextual variation, across time as well as situations, is a hallmark of most childhood problems but not most medical conditions.

It is sometimes difficult to recognize that the medical model with its assumptive base is being applied broadly to problems of children and youth. Its wide use partly derives from successes of the model with certain adult disorders and with occasional childhood disturbances. More general validity of the model is then simply taken for granted and not examined. Moreover, the classification of childhood problems currently in use has served certain purposes in research. Categories such as ADHD promote communication to a degree; they summarize a set of behaviors in shorthand fashion and provide a starting point for research on etiology and treatment. However, the fact that the DSM system is being used cannot be taken as support for its validity. There are children who are impulsive, aggressive, anxious, and so forth, with frequencies of behavioral manifestation showing notable stability in childhood. But this is not evidence of syndromic integrity and not evidence of endogenous pathogens as primary causes.

More research is needed that examines the integrity of existing diagnostic categories and that seeks to uncover new, coherent groupings of problems. Especially important will be research that begins by defining early patterns of adaptation and then follows individuals

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showing such patterns to observed families of outcomes. This contrasts sharply with the current dominant approach of simply assuming the validity of existing categories and then seeking antecedents. When these two approaches converge, one would, of course, have considerable confidence in the meaningfulness of the taxa in question. At the same time, further efforts to group developmental trajectories meaningfully should be given high priority by both researchers and funding agencies. The distinction between adolescent limited and developmentally persistent conduct problems (Moffitt, 1993) is an excellent example. The problems of these groups of children are more appropriately distinguished, rather than being lumped into the same category. It seems likely that developmental analysis will reveal similar distinctions among those who at some time show depression and other problems as well. In general, what is needed is a fresh examination of the whole issue of classification in child psychopathology, based on developmental research.

A serious consequence of the current dominance of the medical model has been its constraining effect on the conduct of research and interpretation of findings. This is highlighted when it is contrasted to a developmental model. Under the aegis of the medical model, environment is defined narrowly (as toxins), precursors are seen as pathogens or simply early forms of the disorder, and course is viewed as linear. Too often problems are considered conditions that children have. Thus, the role of experience is relatively neglected in research on childhood problems and there is a preoccupation with finding the "gene that causes" disorder or the locus of neuropathology. Little research is conducted on experiential risk factors, early adaptation, or processes of change. "Treatment" is viewed narrowly as symptom management and few guides for early intervention or primary prevention are uncovered.

More extensive and encompassing research is needed on risk factors for disorder. As Rutter (1996) has recently stated "the understanding of environmental risk factors in both depressive and (even more so) anxiety disorders in children and adolescents is decidedly limited" (p. 224). This statement can be extended to most childhood disorders if one broadens environment to include experiential factors, rather than simply demographic variables and aspects of the physical environment. Especially important is longitudinal research beginning prior to the onset of disorder. Such research not only is necessary for untangling causal mechanisms and processes, it is the key to resolving the classification problems discussed above.

Currently, for example, much discussion centers around ADHD with and without conduct disorders (e.g., Rutter, 1996). Outcomes for children so diagnosed are very different, but it is not clear what the implications are for classification. Such discussion would be enlightened enormously by antecedent data. Are there distinctive origins of the comorbid pattern, or is it simply a combination of the precursors of ADHD and CD? Are the antecedents of CD alone (or ADHD alone) distinctive from antecedents of those showing the combined pattern (see Loeber, Brinthaupt, & Green, 1990)? Is the comorbid pattern itself in fact heterogeneous? Such questions must be approached developmentally. It is not enough to examine selective correlates of already manifest disorder. In addition to having a broad net of theoretically derived, potentially differentiating variables, it is necessary to examine the relationships between predictors and problem behaviors over time and across ages. Some variables may have stronger differential links with onset of problems, while others may be more tied to persistence or desistance (August et al., 1996). Only longitudinal research can resolve these issues.

Lastly, much more research is needed on processes of continuity and change, again with renewed emphasis on experiential factors (e.g., changing support and guidance of the child). With regard to desistance, both early experiential antecedents (which may provide a foundation for resiliency) and contemporary supports command study. In general, there has been far too little investigation of the interaction between prior adaptation and current risks or changing support.

In conclusion, within a developmental approach problems are viewed as adaptations.

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They may be compromising of development to be sure, but as adaptations they are subject to change as well as forces for continuity. This is especially true as challenges to adaptation are changed. Understanding pathways of adaptation has promise for both effective prevention and broadened approaches to later intervention. As with resilience, coping capacity, and personality in general, maladaptation and, ultimately, disorder may also be presumed to develop. The same laws that govern normal development govern the pathological as well (Cicchetti, 1984; Cicchetti & Sroufe, 1976; Loevinger, 1976).

References

Ainsworth, M., Blehar, M., Waters, E., & Wall, S. (1978). *Patterns of attachment.* Hillsdale, NJ: Erlbaum.

American Psychiatric Association (1987). *Diagnostic and statistical manual of mental disorders.* (3rd ed. rev.). Washington, DC: Author.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders.* (4th ed.). Washington, DC: Author.

Anderson, J., Williams, S., McGee, R., & Silva, P. (1987). DSM III disorders in preadolescent children: Prevalence in a large sample from the general population. *Archives of General Psychiatry, 44,* 69–76.

August, G., MacDonald, A., Realmuto, G., & Skare, S. (1996). Hyperactive and aggressive pathways: Effects of demographic, family, and child characteristics on children's adaptive functioning. *Journal of Clinical Child Psychology, 25,* 341–351.

Blatt, S. (1995). Representational structures in psychopathology. In D. Cicchetti & S. Toth (Eds.), *Rochester symposium on developmental psychopathology: Vol. 6. Emotion, cognition, and representation* (pp. 1–34). Rochester, NY: University of Rochester Press.

Bowlby, J. (1973). *Separation.* New York: Basic Books.

Campbell, S. (1990). The socialization and social development of hyperactive children. In M. Lewis & S. Miller (Eds.), *Handbook of developmental psychopathology* (pp. 77–92). New York: Plenum.

Carlson, E. *A longitudinal study of attachment disorganization*. Manuscript submitted for publication.

Carlson, E., Jacobvitz, & Sroufe, L. A. (1995). A developmental study of inattentiveness and hyperactivity. *Child Development, 66,* 37–54.

Caron, C., & Rutter, M., (1991). Comorbidity in child psychopathology: Concepts, issues, and research strategies. *Journal of Child Psychology and Psychiatry, 32,* 1063–1079.

Cicchetti, D. (1984). The emergence of developmental psychopathology. *Child Development, 55,* 1–7.

Cicchetti, D., & Rogosch, F. (Eds.) (1996). Developmental pathways: Diversity in process and outcome [Special Issue]. *Development and Psychopathology, 8*, 597–600.

Cicchetti, D., & Sroufe, L. A. (1976). The relationship between affective and cognitive development in Down's syndrome infants. *Child Development, 47,* 920–929.

Cicchetti, D., & Tucker, D. (1994). Development and self-regulatory structures of the mind. *Development and Psychopathology, 6,* 533–549.

Cohen, J., & Stewart, I. (1994). *The collapse of chaos.* New York: Penguin Books.

Crick, N., & Dodge, K. (1994). A review and reformulation of social information-processing mechanisms in children's social adjustment. *Psychological Bulletin, 115,* 74–101.

Egeland, B., Carlson, E., & Sroufe, L. A. (1993). Resilience as process. *Development and Psychopathology, 5,* 517–528.

Eisenberg, L. (1977). Development as a unifying concept in psychiatry. *British Journal of Psychiatry, 131,* 225–237.

Elicker, J., Englund, M., & Sroufe, L. A. (1992). Predicting peer competence and peer relationships in childhood from early parent–child relationships. In R. Parke & G. Ladd (Eds.), *Family–peer relationships: Modes of linkage* (pp. 77–106). Hillsdale, NJ: Erlbaum.

Fox, N., Kimmerly, N., & Schafer, W. (1991). Attachment to mother/attachment to father: A meta-analysis. *Child Development, 62,* 210–225.

Frick, P., & Lahey, B. (1991). The nature and characteristics of attention-deficit hyperactivity disorder. *School Psychology Review, 20,* 163–173.

Goodman, R., & Stevenson, J. (1989). A twin study of hyperactivity. *Journal of Child Psychology and Psychiatry, 30,* 691–709.

Gottesman, N. (1995). *Environmental and familial antecedents of sixth grade aggression.* Unpublished doctoral dissertation. University of Minnesota.

Gottlieb, G. (1991). Experiential canalization of behavioral development: Theory. *Developmental Psychology, 27,* 4–13.

Greenough, W., Black, J., & Wallace, C. (1987). Experience and brain development. *Child Development, 58,* 539–559.

Harrington, R. (1994). Affective disorders. In M. Rutter, E. Taylor, & L. Hersov (Eds.), *Child and adolescent psychiatry* (pp. 330–350). London: Blackwell.

Hinshaw, S., & McHale, J. (1991). Stimulant medications and the social interactions of hyperactive children. In D. Gilbert & J. Connolly (Eds.), *Personality, social skills and psychopathology* (pp. 229–253). New York: Plenum.

Hobson, R. P., & Patrick, M. (1995). Emotion, cognition, and representation: The interpersonal domain. In D. Cicchetti & S. Toth (Eds.), *Rochester symposium on developmental psychopathology: Vol. 6. Emotion, cognition, and representation* (pp. 167–190). Rochester, NY: University of Rochester Press.

Jacobvitz, D., Sroufe, L. A., Stewart, M., & Leffert, N. (1990). Treatment of attentional and hyperactivity problems in children with sympathomimetic drugs: A comprehensive review. *Journal of the American Academy of Child Psychiatry, 29,* 677–688.

Jacobvitz, D., & Sroufe, L. A. (1987). The early caregiver–child relationship and attention deficit disorder with hyperactivity in kindergarten. *Child Development, 58,* 1488–1495.

Kraemer, G. (1992). A psychobiological theory of attachment. *Behavioral and Brain Sciences, 15,* 493–511.

https://doi.org/10.1017/S0954579497002046 Published online by Cambridge University Press

Lazare, A. (1973). Hidden conceptual models in clinical psychiatry. *New England Journal of Medicine, 288,* 345–350.

Loeber, R. (1991). Questions and advances in the study of developmental pathways. In D. Cicchetti & S. Toth (Eds.), *Rochester symposium on developmental psychopathology* (Vol. 3, pp. 97–115). Rochester, NY: Rochester University Press.

Loeber, R., Brinthaupt, V., & Green, S. (1990). Attention deficits, impulsivity, and hyperactivity with or without conduct problems: Relationships to delinquency and unique contextual factors. In R. McMahon & R. Peters (Eds.), *Behavior disorders of adolescence: Research, intervention, and policy in clinical and school settings* (pp. 39–61). New York: Plenum.

Loeber, R., Wang, P., Keenan, K., Giroux, B., Stouthamer–Loeber, M., VanKammen, W., & Maughan, B. (1993). Developmental pathways in disruptive child behaviors. *Development and Psychopathology, 5,* 103–134.

Loevinger, J. (1976). *Ego development.* San Francisco: Jossey–Bass.

Main, M., & Hesse, E. (1990). Parents' unresolved traumatic experiences are related to infant disorganized attachment status. In M. Greenberg, D. Cicchetti, & E. M. Cummings (Eds.), *Attachment in the preschool years* (pp. 161–182). Chicago: University of Chicago Press.

Masten, A., & Coatsworth, J. D. (1995). Competence, resilience, and psychopathology. In D. Cicchetti & D. Cohen (Eds.), *Developmental Psychopathology* (Vol. 2, pp. 715–752). New York: Wiley.

Moffitt, T. (1993). Adolescence-limited and life-course persistent antisocial behavior: A developmental taxonomy. *Psychological Review, 100,* 674–701.

Patterson, G., DeBaryshe, B., & Ramsey, E. (1989). A developmental perspective on antisocial behavior. *American Psychologist, 44,* 329–335.

Renken, B., Egeland, B., Marvinney, D., Sroufe, L. A., & Mangelsdorf, S. (1989). Early childhood antecedents of aggression and passive withdrawal in early elementary school. *Journal of Personality, 57,* 257–281.

Richters, J., & Cicchetti, D. (1993). Mark Twain meets DSM-III–R: Conduct disorder development, and the concept of harmful dysfunction. *Development and Psychopathology, 5,* 5–29.

Rieder, C., & Cicchetti, D. (1989). Organizational perspective on cognitive control functioning and cognitive-affective balance in maltreated children. *Developmental Psychology, 25,* 382–393.

Robins, L., & Price, R. (1991). Adult disorders predicted by childhood conduct problems. *Psychiatry, 54,* 116–132.

Rogosch, F., Cicchetti, D., & Aber, L. (1995). The role of child maltreatment in early deviations in cognitive and affective processing abilities and later peer relationships. *Development and Psychopathology, 7,* 591–610.

Rutter, M. (1980). Introduction. In M. Rutter (Ed.), *Scientific foundations of developmental psychiatry.* London: Heineman.

Rutter, M. (1996). Developmental psychopathology: Concepts and prospects. In M. Lenzenweger & J. Havgaard (Eds.), *Frontiers of developmental psychopathology* (pp. 209–237). New York: Oxford University Press.

Rutter, M., Taylor, E., & Hersov, L. (1994). *Child and adolescent psychiatry.* London: Blackwell.

Sameroff, A., & Fiese, B. (1989). Transactional regulation and early intervention. In S. J. Meisels & J. Shonkoff (Eds.), *Early intervention: A handbook of theory, practice, and analysis.* New York: Cambridge University Press.

Santostefano, S. (1978). *A biodevelopmental approach to clinical child psychology*. New York: Wiley.

Schachar, R., & Wachsmuth, R. (1990). Hyperactivity and parental psychopathology. *Journal of Child Psychology and Psychiatry, 31,* 381–392.

Schore, A. (1994). *Affect regulation and the origin of the self.* Hillsdale, NJ: Erlbaum.

Sroufe, L. A. (1988). The role of infant–caregiver attachment in development. In J. Belsky & T. Nezworski (Eds.), *Clinical implications of attachment* (pp. 18–38). Hillsdale, NJ: Erlbaum.

Sroufe, L. A. (1989). Pathways to adaptation and maladaptation: Psychopathology as developmental deviation. In D. Cicchetti (Ed.), *Rochester symposium on development psychopathology* (Vol. 1, pp. 13–40). Hillsdale, NJ: Erlbaum.

Sroufe, L. A. (1990). An organizational perspective on the self. In D. Cicchetti & M. Beeghly (Eds.), *Transitions from infancy to childhood: The self* (pp. 281–307). Chicago: The University of Chicago Press.

Sroufe, L. A., Carlson, E., & Shulman, S. (1993). Individuals in relationships: Development from infancy through adolescence. In D. Funder, R. Parke, C. Tomlinson–Keasey, & K. Widman (Eds.), *Studying lives through time: Personality and development* (pp. 315–342). Washington, DC: American Psychological Association.

Sroufe, L. A., Egeland, B., & Carlson, E. (in press). One social world. In W. A. Collins & B. Laursen (Eds.), *Relationships as developmental contexts: The Minnesota symposia on child psychology* (Vol. 29). Hillsdale, NJ: Erlbaum.

Sroufe, L. A., & Egeland, B. (1991). Illustrations of person and environment interaction from a longitudinal study. In T. Wachs & R. Plomin (Eds.), *Conceptualization and measurement of organism–environment interaction* (pp. 68–84). Washington, DC: American Psychological Association.

Sroufe, L. A., Egeland, B., & Kreutzer, T. (1990). The fate of early experience following developmental change: Longitudinal approaches to individual adaptation in childhood. *Child Development, 61,* 1363–1373.

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

Suess, G. J., Grossmann, K. E., & Sroufe, L. A. (1992). Effects of infant attachment to mother and father on quality of adaptation in preschool: From dyadic to individual organization of self. *International Journal of Behavioral Development, 15,* 43–66.

Taylor, E. (1994). Syndromes of attention deficit and overactivity. In M. Rutter, E. Taylor, & L. Hersov (Eds.), *Child and adolescent psychiatry* (pp. 285–307). London: Blackwell.

Thelen, E. (1990). Dynamical systems and the generation of individual differences. In J. Colombo & J. Fagan (Eds.), *Individual differences in infancy* (pp. 19–43). Hillsdale, NJ: Erlbaum.

Troy, M., & Sroufe, L. A. (1987). Victimization among preschoolers: The role of attachment relationship history. *Journal of the American Academy of Child and Adolescent Psychiatry, 26,* 166–172.

https://doi.org/10.1017/S0954579497002046 Published online by Cambridge University Press

Vaughn, B., Egeland, B., Waters, E., & Sroufe, L. A. (1979). Individual differences in infant–mother attachment at 12 and 18 months: Stability and change in families under stress. *Child Development, 50,* 971–975.

Waddington, C. (1957). *The strategy of genes.* London: Allen & Unwin.

Wakefield, J. C. (1992a). The concept of mental disorder: On the boundary between biological facts and social values. *American Psychologist, 47,* 373–388.

Wakefield, J. C. (1992b). Disorder as harmful dysfunction: A conceptual critique of DSM III–R's definition of mental disorder. *Psychological Review, 99,* 232–247.

Warren, S., Egeland, B., Huston, L., & Sroufe, L. A. (in press). Child and adolescent anxiety disorders and early attachment. *Journal of the American Academy of Child and Adolescent Psychiatry*.

Werner, E., & Smith, R. (1992). *Overcoming the odds.* Ithaca, NY: Cornell University Press.

Zametkin, A., Nordahl, T., Gross, M., King, A., Semple, W., Ramsey, J., Hamburger, S., & Cohen, R. (1990). Cerebral glucose metabolism in adults with hyperactivity of childhood onset. *New England Journal of Medicine, 323,* 1361–1366.

Zeenah, C., Mammen, O., & Lieberman, A. (1994). Disorders of attachment. In *Handbook of infant mental health* (pp. 332–349). New York: Guilford Press.

https://doi.org/10.1017/S0954579497002046 Published online by Cambridge University Press