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The promise of developmental psychopathology: Past and present

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Abstract

Progress in the field of developmental psychopathology is appraised in general and with regard to the particular lens of our understanding of the development of disorder. In general, the outpouring of research on various features of disorder and underlying processes could not have even been imagined 25 years ago. The progress is dazzling. At the same time, work on the development of disorders, beginning with antecedent patterns of adaptation, pales in comparison with work on the correlates of disorder. However, progress has been made. It is well established that the brain develops in the context of experience and that organism and environment continually interact over time. Something is now known about pathways leading to certain disorders and what initiates and impels individuals along them. If developmental psychopathology is to completely fulfill its promise of offering new ways of conceptualizing disorder and new guidance for prevention and intervention, much more work on developmental processes and a new way of exploring the development of disorder will be needed. Such a path is suggested.

The major premise of the developmental psychopathology perspective is that psychopathology develops. Moreover, it develops according to the same principles that govern all aspects of human development, whether it is the human embryo, the brain, normal capacities such as the ability to regulate emotions or engage in competent social relations, or the development of the personality. Thus, this perspective has offered enormous promise from the outset. If pathology develops in a lawful manner, it should be possible to identify precursors and pathways leading to disorder, along with factors that propel individuals along such pathways or deflect them back toward more functional adaptation. Prevention and intervention would be informed. At the same time, conjoint study of normal and atypical development would further illuminate developmental processes themselves. For example, what determines continuity or discontinuity in development? How does prior experience exercise its impact in changed environmental circumstances? Following developmental change, does prior adaptation remain latent or is it erased? A myriad of more subtle, nuanced questions also come to the fore.

Three decades ago, with publication of the special issue of *Child Development* (Cicchetti, 1984), and 25 years ago, with the inaugural publication of *Development and Psychopathology*, there was reason for optimism that this field could fulfill its promise. This was because of some of the unique strengths of this discipline. Developmental psychopathology is a particular field of inquiry, although it is not entirely easy to delimit (Sroufe & Rutter, 1984). It is broader than the study of child disorder because the origins and course of adult conditions are equally of interest. It is even broader than the study of disorder itself because of a keen interest in those individuals at risk for disorder who develop well and because disorder itself is defined as deviation from the normal. The starting point for understanding disorder is understanding how most individuals develop so as not to have a particular problem. The goal for this field was to understand, first, how individuals typically develop the array of capacities that allow them to cope effectively with challenges posed by each developmental period and, second, what leads to failures to develop these capacities or these functional patterns of adaptation. The particular nature of the failures would forecast particular kinds of later problems.

This seemed like a plausible goal, and meaningful work had already been done. We knew something about the complexity of development; for example, there was heterotypic as well as homotypic continuity. We had reasonable descriptions of the series of issues children had to negotiate age by age (e.g., Sander, 1975); and we knew a lot about normative cognitive, social, and emotional development. We had risk research strategies and longitudinal strategies that were well suited for exploring variations in individual development. Although there was no overarching theory of developmental psychopathology (and it can be argued that this is not even a realistic goal; Rutter, 2013 [this issue]), there were pertinent theories sufficient to offer guidance. Attachment theory as framed by Bowlby (1973), for example, meets all of the tenets of the developmental psychopathology perspective. It begins with normal development, sees disturbance as lying in compromised developmental processes, focuses on uncovering the roots of disturbance long before disturbance proper exists, and makes specific statements about the power and limits of

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early experience. Other theories (e.g., Werner, 1948) were instructive regarding the nature of development, that is, how development works. Thus, there were concepts and tools available, with many more valuable assessment and analytic tools soon to come.

How have we done? By most standards this discipline has matured quite rapidly. No one could have imagined the outpouring of research and scholarly work in this field or the astounding rise to prominence of this journal. This body of work is so vast that it cannot even be summarized in a single article or even a single volume. The amount of descriptive work on pathology has been stunning. We have a vastly increased understanding of antecedents and correlates of problem behavior. Neurophysiological correlates alone account for a multitude of papers. The outpouring of work on brain development and gene–environment interaction could not have been anticipated and is certain to continue moving the field forward. For certain kinds of problems, we know something about their developmental course and the distinctiveness of predictors when problems emerge in different periods of life. This has been particularly crucial because it confirms the developmental psychopathology precept that understanding developmental course is more important than cataloguing manifest symptoms alone. Individuals may have comparable problems; but if they arrived at these problems following different developmental pathways, there are differing implications for their future functioning (Moffitt, 1993). We have rather fully confirmed the critical importance of early experience (e.g., Stiles, 2008). We even have learned something about *how* early experience has its influence. There is now a more sophisticated understanding of the developmental process in general. Finally, we have begun demystifying both pathology and resilience. I will elaborate on some of these achievements on later pages.

Although we are clearly closer to fulfilling the promise of this field, enthusiasm about our progress is restrained by one certainty: in the course of all of this research, we have still learned too little about how pathology *develops*. We are still at the very early stages of elaborating patterns of adaptation in early life that forecast different kinds of problems that will emerge. The vast majority of work accomplished takes DSM (or sometimes ICD) categories as the starting point. This was understandable given that it was the system in place. The problem is that finding contemporary correlates (even neural correlates) of DSM-based disorders in general tells us nothing about development. One of the most important achievements of DSM-based research was to uncover the massive problems with this system. So-called "comorbidity" is rampant (e.g., Caron & Rutter, 1991). Major child categories overlap substantially with other major child categories; for example, half the children who fit the criteria for attention-deficit/hyperactivity disorder (ADHD) will also fit criteria for conduct disorder or depression or anxiety disorder. The term *comorbidity* reveals a clinging to views of problem behaviors as entities, despite the evidence to the contrary. A developmental approach could do much to clarify this problem.

We need studies that unpack the heterogeneity in current categories by examining differential antecedents and pathways. There still are too few efforts to provide comprehensive developmental accounts of any group of problems. As just one of many examples, thus far no one has traced the course of anorexia from early developmental antecedents (such as rigid parental control in the preschool years) to early patterns of behavior, through changing manifestations, and ultimately to disorder itself; rather, papers focus on contemporary correlates and retrospective accounts of early feeding issues, reflecting an inappropriately simple developmental perspective. At the same time, a start on a truly developmental account could be made with longitudinal data that exist. For some problems there *are* a scattering of developmental accounts, and some of these will be touched upon later.

To be fair, in the 1980s it was perhaps too soon to address the vexing problem of understanding in a comprehensive way the development of disorder. Establishing correlates of disorders as currently defined was an understandable place to begin. One way to appraise the current state of the field is to say the following: much necessary background research and conceptualizing has been accomplished, and we are now in an excellent position to take on the challenging tasks that still lie before us. There has been some advance in our understanding of the development of psychopathology. We know organism and environment interact in an ongoing way; we know early experience has a special place in development, and we know something about why, and we know much more than ever that a developmental perspective provides a powerful way of looking at disordered behavior. Everything develops, and that includes disorder.

The Development of Psychopathology

Other papers in this Special Issue will describe features associated with a full array of disorders. Here, I will cite no studies that simply compare those who fit some DSM criteria with some other group or a control group on some contemporaneous variables. Such differences are legion and, although they can generate useful hypotheses, they intrinsically reveal little about how the problems came to be. For example, if it is shown that reduced hippocampal volume is associated with a certain disorder, one cannot conclude that a small hippocampus was the root of the problem. There is now even human data suggesting that harsh experience may lead to hippocampal reduction as well as to psychopathology (Luby et al., 2012). My focus will be on what we have learned about developmental processes and psychopathology in the last three decades, emphasizing findings from the Minnesota Longitudinal Study of Risk and Adaptation (Sroufe, Egeland, Carlson, & Collins, 2005).

Two propositions about development will be the focus of the discussion. First, development is "cumulative" or, as Stiles (2008) puts it when talking about brain development, it always "builds upon itself." Each phase provides a foundation for what is to come. Past development shapes subsequent

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development (Masten & Cicchetti, 2010). This is a major explanation for why adaptation at each age is related to adaptation at the next (Sroufe et al., 2005). To be sure, discontinuities and transformations may be profound at later phases or in response to changing contexts, but nothing is lost. Butterflies, however different in form from caterpillars, nonetheless lay their eggs on the type of foliage that nurtured them as larva (Sackett, Sameroff, Cairns, & Suomi, 1981). To provide an example more directly pertinent to psychopathology, monkeys who have been extremely deprived as infants can be rehabilitated to normal functioning within a well-functioning social group and will be behaviorally indistinguishable from the others. Nevertheless, when experiencing the stress of being caged for testing, they show their uniquely characteristic stereotypies from the deprived infant period (Novak, O'Neill, Beckley, & Suomi, 1992).

Second, the other proposition is that development is best characterized by probabilistic pathways rather than by linear causality. Early adversity or early maladaptation does not ineluctably lead to pathology; rather, it initiates a process that may be more likely lead to pathology if that pathway continues to be supported. Bowlby (1973) used the analogy of branching train tracks in a railway yard, and five specific corollaries follow from his model: (a) any starting path may have numerous possible outcomes (multifinality), (b) two different initial paths may lead to the same outcome (equifinality), (c) change is possible all along the way, (d) change is constrained by how long the pathway has been followed, and (e) pathology is defined by sustained deviation from functional pathways.

Evidence has accumulated in the last 25 years in support of all of these propositions. Examples can be taken from either positive adaptation or the development of disturbance. I will begin with the example of resilience, which clearly reveals the cumulative nature of development (Supkoff, Puig, & Sroufe, 2012). Resilience is an acquired capacity. For example, stress predicts behavior problems but not for all children. Some are "resilient" by this simple, circular definition. However, developmental study shows that such stress resistance is related to a history of supportive care and secure attachment in infancy (Pianta, Egeland, & Sroufe, 1990). Another example comes from continuity and change in behavior problems. If children show consistent problems during the preschool period (ages 3–5), they are likely to show problems in third grade, but not all children do. Some recover normal functioning and may be called resilient because of this. However, such recovery is predictable based on a history of early positive care and adaptation in the first 2 years (Sroufe, Egeland, & Kreutzer, 1990). This history is not erased by the period of difficulty but remains a latent force to be tapped in later circumstances. Of course, cumulative development also means that it is not just early experience that accounts for resilience. Increased social support and reduced stress also account for a shift away from problem behavior to adequate functioning (Sroufe, 1999). From preschool to middle childhood and from middle childhood to adolescence, taking into account both positive early history and improved circumstances leaves little unexplained in these positive changes.

As a final example of positive change being rooted in both history and current opportunities, consider some preliminary findings from our project with regard to depression. Depression shows notable stability from adolescence to adulthood. However, there is discontinuity as well. There has been great interest recently in "turning points," which are opportunities for growth, such as finding a partner (e.g., Elder, 1986; Rutter, 1996). We also find that forming a stable partnership accounts for some decline in depression. However, this change is stronger if the individuals also had secure attachments in infancy. In other words, some individuals are better able to take advantage of opportunities, based on their histories. Specifying which subpopulations respond to developmental opportunities is the key issue in turning points research (Rutter, 1996).

Note that all of this works the same way when talking about disturbance. Children may do well following a period of maladaptation; nonetheless, they retain a vulnerability based on the earlier period and are more vulnerable to difficulties as support declines or stress increases (Sroufe et al, 2005). In addition, just as some individuals are better able to take advantage of opportunities, others are more vulnerable to adversity. Most writers on the topic of "differential susceptibility" are focused on genetic explanations (Belsky & Pluess, 2009), but past experience is also involved (Rutter, 1996). For example, trauma, such as physical abuse in the preschool years, is associated with a tendency to dissociate in later life. However, this is not true for some. That is, some children are more susceptible to this adversity. Research shows that those with a disorganized attachment as infants (now rather clearly linked to frightening or unfathomable parental behavior; e.g., Jacobvitz, Hazen, & Riggs, 1997) have significantly higher dissociation scores at age 19 years than do those with secure attachment histories who experienced the same degree of early childhood adversity (Ogawa, Sroufe, Weinfield, Carlson, & Egeland, 1997). Health and pathology, resilience and vulnerability, are subject to the same principles of development. Development is cumulative.

There has also been forthcoming evidence supporting the five propositions derived from a developmental pathways model. Multifinality, for example, is well established. Studies showed decades ago that what would now be called conduct disorders in childhood were the forerunners not only of adult alcoholism and antisocial behavior but also of internalizing disorders, such as depression and even schizophrenia (Robins, 1966). There has now been an outpouring of evidence that child maltreatment predicts a panoply of later problems, with the multifinality including both internalizing and externalizing disorders and a range of specific disorders from anxiety and depression to posttraumatic stress disorder, borderline personality disorder, and schizophrenia (Cicchetti & Valentino, 2006; Read, Perry, Moskowitz, & Connolly, 2001). The abuse literature is also pertinent to the question

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of equifinality. Child sexual abuse, for example, is a strong predictor of adolescent depression; yet most girls suffering from depression in adolescence were not sexually abused (Duggal, Carlson, Sroufe, & Egeland, 2001).

My research, which has direct assessments of adaptation and contextual factors at every developmental period beginning at birth (and even before birth with regard to risk factors), clearly illustrates the third and fourth propositions above. Change *is* possible at every point in development, but the longer a pathway is followed the more difficult change becomes. For example, anxious attachment in infancy and maladaptation and/or adverse circumstances at age 2 or 3 or 4 or 5 years all are modest predictors of later problems. However, maladaptation (or positive functioning) *across* all of these periods much more powerfully predicts outcomes (Sroufe et al., 2005). Stability coefficients for externalizing problems (e.g., ADHD and conduct disorder) become increasingly strong across the childhood years, and it becomes more and more difficult to isolate factors that predict change (Egeland, Kalkoske, Gottesman, & Erickson, 1990). However, change does remain possible. Even those described by Moffitt (1993) and others as having persistent conduct disorder can show remission between adolescence and adulthood, and these changes are predictable from changes in life circumstances (Roisman, Aguilar, & Egeland, 2004).

Finally, it is clear that maladaptation per se can be distinguished from psychopathology. Avoidant and resistant attachment in infancy clearly should not be considered pathology, as the majority of such cases will not later qualify for psychiatric diagnoses. However, they may be thought of as initiating pathways that, if pursued, will in time lead to disturbance. Avoidant attachment, for example, can lead to negative affectivity, noncompliance and continued avoidance at age 3. Such growing alienation at this age promotes later oppositional behavior and, ultimately, serious conduct problems (Egeland, Yates, Appleyard, & van Dulmen, 2002).

Tracing courses of development from risk and maladaptation, in advance of frank pathology, will ultimately allow us to fulfill the promise of this field. In the following section, I discuss developmental pathways with regard to three specific disorders: ADHD, conduct disorders, and depression. Each discussion will again illustrate all of the developmental propositions outlined above.

Development of ADHD

The problems subsumed under the ADHD label provide an excellent example of a developmental account because we know so much about how the capacities for sustained attention, emotion regulation, and flexible self-control develop and about the role of the caregiving environment in these achievements. Although beyond the scope of this paper, a detailed series of steps can be described, based on observational research, wherein what begins as a process of caregivers' helping to stabilize early infant states, progresses to caregiver modulation of infant arousal and expression, to a more active role for the infant, to more fully dyadic regulation, and ultimately, to self-regulation (Sroufe, 1989, 1997).

Understanding how each step is normatively accomplished, and the caregiving assistance needed, it is then possible to generate hypotheses about how the process can go awry at each point and what would continue to propel the child along the pathway toward ADHD problems or deflect him or her off of it. This analysis is what led us emphasize "intrusive" care at age 6 months. At this age infants have very modest capacities to regulate arousal. However, they do have a primitive "signal" system, for example, turning away during face-to-face interaction when arousal becomes too high. Caregivers properly reading such behaviors slow the pace of stimulation, building it up again as the infant is ready, thereby extending the capacity for coping with higher levels of arousal (Brazelton, Kowslowski, & Main, 1974). In contrast, caregivers who intensely stimulate an unprepared infant, whose behavior is at cross-purposes with the infant's readiness, repeatedly overwhelm and compromise the infant's capacities.

Likewise, our analysis led us to a particular measure at age 42 months, which took into account the child's fledgling capabilities for self-regulation. At this age, the parents' task is to let children stretch coping capacities, regulating themselves when they can. Only when the child's capacities are about to be exceeded does the sensitive parent step in with regulatory assistance. However, some parents precisely *add* load to the child at these moments, teasing, ridiculing, pushing, distracting with flirting or giggling, and other behaviors that we subsume (for other theoretical reasons) under the label of "parent–child boundary violations" (Sroufe, Jacobvitz, Mangelsdorf, deAngelo, & Ward, 1985). Such ill-timed and poor-quality stimulation would critically disrupt growing capacities for regulation.

Our outcome studies confirmed this analysis and these two variables in particular. Both were related to ADHD symptoms across childhood (Carlson, Jacobvitz, & Sroufe, 1995; Sroufe et al., 2005). It is noteworthy that measures of newborn neurological anomalies and measures of early infant temperament (most notably activity level) fared poorly in predicting ADHD behaviors. Only by age 42 months did a child measure ("distractibility" in a problem-solving situation) show a meaningful relation to later ADHD. Such distractibility itself was predicted by our earlier parent intrusiveness measure but not by any endogenous measure. ADHD symptoms in early elementary school were even more powerful than was distractibility in predicting problems at age 11 years. Still, even though the pattern became more stable across these years, decreases in family life stress and increases in family social support accounted for a significant lessening of ADHD symptoms. This and other findings on the importance of contextual variables should dissuade readers from seeing these data as blaming parents.

There is one final point: only one of dozens of endogenous variables examined in infancy was related to ADHD symptoms. This was the "motor maturity" scale on the Brazelton

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Neonatal Behavioral Assessment Scale (not the most compelling scale variable one might suggest). It was of interest to us that clinical cases accounted for by this variable showed virtually no overlap with cases accounted for by intrusive care. Conclusions here must be tentative because motor maturity was one of many variables and was not predicted a priori to relate to ADHD. However, this is an example of possibly different routes to the same disorder. We need much more research like this.

Developmental approaches to conduct problems

Drawing on the Dunedin longitudinal data, Moffitt (1993) propelled the field toward a developmental perspective on conduct problems. Moffitt's insight was that adolescents exhibiting conduct problems might represent two distinct groups, and developmental course might be the crucial distinction. Some teens had a rather continuous history of problems beginning in early childhood, which are called life-course-persistent (LCP) cases. For others, the problems emerge in the teenage years following a childhood without notable problems (adolescent limited). Moffitt reasoned that the latter pattern was more *normative*, perhaps representing a striving for autonomy or identification with deviant youths. Because of the different histories, and in keeping with pathways propositions outlined above, she reasoned that remission would be more likely in the adolescent-onset group and that their early childhoods would be unremarkable. Based on low verbal IQ performance in middle childhood and persistence itself, Moffitt considered the LCP group to represent a "neuropsychological disorder," more in keeping with the disease model underlying the DSM.

We found the first part of Moffitt's hypothesis compelling and even inspiring. The idea that individuals with manifestly similar problems could represent two distinctive groups based on history was a critical affirmation of the developmental pathways model. Moreover, the use of developmental data, both before and after symptom manifestation, was an important guide for future research. We were more skeptical of some of her specific assertions about the LCP group, especially given certain limits of the Dunedin data set in the early years.

With our Minnesota longitudinal data set we, too, were able to distinguish a group of individuals whose problems began early and persisted and another group whose problems emerged first in adolescence (Aguilar, Sroufe, Egeland, & Carlson, 2000). We also constituted a third group of those never manifesting conduct problems. Like Moffitt, we found that those in the adolescent-onset group had early histories as benign as the never-antisocial group. The LCP group had a distinctive early history as well. Consistent with voluminous research (e.g., Burnette & Cicchetti, 2012; Eron & Huesman 1990; Farrington, 1995; Patterson & Dishion, 1988), it was characterized by a history of psychosocial adversity. Even individual psychosocial variables distinguished the LCPs, including avoidant and disorganized attachment, parental emotional unavailability, physical abuse, mother being single at birth, and high family life stress, all assessed in the earliest years of life. However, it was not predicted by any of a large number of endogenous variables (perinatal difficulties, newborn neurological status, physical anomalies, infant temperament, or preschool intelligence test performance), either singly or collectively. Verbal problems were a feature of this group *but only beginning in middle childhood*, not the preschool years. Thus, it is better viewed as an outcome of being on this pathway rather than as a root cause. We certainly agree with Moffitt and a host of others (e.g., Gilliom & Shaw, 2004) that early onset problems are pernicious and persistent. However, rather than concluding that this is evidence of endogenous causation, we would make the developmental point that, for some patterns of maladaptation, persistence is generated by the pattern itself. As we concluded based on ample evidence: "Because harsh, chaotic treatment leads to interpersonal alienation and anger, a lack of internalized empathy, and impulse control problems due to early dysregulation, these children engage in disruptive, oppositional, and aggressive behaviors. Such behavior prompts further anger and harsh treatment from parents, and alienates teachers and peers, leading to further rejection all around . . ." (Sroufe et al., 2005, pp. 256–257). One need not assume any endogenous precondition to account for persistence.

In the follow-up studies of our three groups, we found that the LCP group showed the most problem behaviors in adulthood. Even so, some remitted, and this was predicted by both work and relationship opportunities (Roisman et al., 2004). It is quite interesting that the adolescent-onset group, although showing significantly fewer adult problems than did the LCP group, nonetheless showed significantly more problems than the never-antisocial group. Thus, *adolescent onset* is a more appropriate term than *adolescent limited*. This is actually consistent with some of Moffitt's ideas. She argued that even though the adolescent-onset cases are not pathological, still there are hazards of this pattern, such as the potential for drug dependency that could entrap them on this pathway. There is rich material here for future research.

A developmental perspective on depression

Depression offers a final example of the utility and power of a developmental approach. For some time it has been thought that childhood depression and adolescent depression might represent distinctive pathways and that childhood and adult depression might be distinctive conditions (e.g., Harrington, Rutter, & Fombonne, 1996). For instance, childhood depression is more rare than adult depression. In addition, in childhood the gender ratio is close to equal with a slight predominance of boys. Then, in adolescence and adulthood, there are notably more females than males. Harrington and colleagues argued that adolescent depression might be better thought of as early onset adult depression rather than an extension of childhood depression, likely having a more notable genetic component than childhood depression. There is little stability between childhood and adult depression. It has even been

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reported that conduct problems in childhood are a better predictor of adult depression than is childhood depression (Robins & Price, 1991). In our study we found a correlation of only .22 between childhood and adolescent depression, which is dramatically smaller than the stability coefficient for conduct problems (Duggal et al., 2001). All of this suggests that these may be different conditions.

Parallel to the case of conduct problems, an examination of developmental antecedents was the key. This analysis revealed notable differences in these pathways. As studies of maltreatment have found (e.g., Cicchetti &Toth, 2005), we too found that measures of early adversity strongly predicted childhood depression. The single strongest predictor was abuse (.30), but a poor early parental care composite based on observation, early life stress, and low parental social support predicted equally. Mother's depression was also related to childhood depression, and some of what is captured by this measure may be genetic variance. The four psychosocial variables still explained 13% of the variance, even after taking maternal depression into account.

For adolescent depression, maternal depression rivaled psychosocial variables in predictive power. Here there was a strong interaction with gender, revealing that maternal depression had a much greater impact for depression in girls. This was even clearer when we looked at clinical groups (those with scale scores high enough to receive diagnoses). Both abuse and early family stress were significantly higher for childhood depression than for adolescent depression. Only maternal depression distinguished the adolescent clinical cases from controls. This is in keeping with the Harrington hypothesis of a higher genetic loading for adolescent depression. We further looked at those cases where depression began in childhood and continued into adolescence. We did not find maternal depression to be more prominent for this group of children, as might have been the case if they represented a subgroup of very early onset adult depression. They were better characterized by early adversity. We have now gathered DNA samples and are following our participants well into adulthood to further clarify these issues.

A Special Role for Early Experience

In the mid-1980s it was still a matter of contention whether early experience played any special role in development (e.g., Kagan, 1984). However, if development is "cumulative," if it "builds upon itself," then clearly early experience must have a special role, precisely because it comes at the beginning. Perhaps the most compelling data on this issue come from the animal studies of brain development, in which it has been shown quite convincingly that variations in very early experience exert a powerful impact on the developing brain and on the expression of genes (e.g., Kaffman & Meaney, 2007; Stiles, 2008; Weaver, Meaney, & Szyf, 2005). Human work is just beginning, but the neurophysiological effects of early institutional rearing appear to be quite clear (Marshall, Reeb, Fox, Nelson, & Zeanah, 2008).

Children experiencing early adversity, such as maltreatment, are much more likely to demonstrate various kinds of behavior problems later (Cicchetti & Valentino, 2006). In our own study we were able to examine adversities, such as witnessing violence, being physically abused, and experiencing high life stress, in both early childhood and middle childhood. It is true that most often adversity in early childhood is more potent than comparable adversity in middle childhood, even though that period was closer to assessed outcomes (Appleyard, Egeland, van Dulmen, & Sroufe, 2005). There are exceptions, of course; for example, a stable male presence in the home in middle childhood is a more powerful promotive factor with regard to boys' adolescent behavior problems than is a stable male presence in early childhood (Sroufe & Pierce, 1999). Timing is the more crucial variable, but many times early experience is critical.

Predictions from early in life are sometimes quite powerful. Disorganized attachment in infancy correlates .40 with psychiatric symptoms at age 17 years, whereas measures of temperament in infancy do not (Carlson, 1998). Dropping out of school can be predicted with 77% accuracy by age 3 from measures of care and support, and this is true with IQ controlled (Jimerson, Egeland, Sroufe, & Carlson, 2000). Although it is true that early experience is correlated with later experience, early experience often retains its effect with later experience controlled (Sroufe et al., 2005).

Moreover, later experience is partially an outgrowth of early experience. We now know something about this process, that is, how early experience exerts its effect or how it is carried forward. Of course, if experience alters brain structure and function and genetic expression, it is already internalized in that sense. At other levels, early experience impacts the child's pattern of adaptation and representations of self, other and relationships (Carlson, Sroufe, & Egeland, 2004; Fury, Carlson, & Sroufe, 1997; Toth, Maughan, Manly, Spagnola, & Cicchetti, 2002). For example, children whose desires for emotional closeness are chronically rejected not only form avoidant patterns of attachment but also take their experience-based expectations and typical ways of coping with them into other settings. These affect how they react to situations, how they interpret the behavior of others, and the environments they select and create. In preschool, it is predictable that they tend to be aggressive or mean and not empathic with others (Kestenbaum, Farber, & Sroufe, 1983). They tend to isolate themselves and, although they spend a lot of time in the vicinity of teachers, they explicitly do *not* go to them when threatened or distressed (Sroufe, 1983). It is not surprising that they are sociometrically rejected by peers and treated in a controlling, disciplinary, and at times even angry way by teachers (Sroufe, 1983; Sroufe & Fleeson, 1988). In a German study, using cartoon frames, it was found that children with this history are more likely interpret ambiguous situations as implying threat or hostility, as in the following example (Suess, Grossmann, & Sroufe, 1992): a child is building a block tower. Another child walks by. In the final frame the tower has crumbled. Children with secure

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histories think it must have been an accident; those with avoidant histories think it was purposeful. These patterns of behaving (and what they elicit), these reactions to events, and these ways of construing the world all serve to perpetuate the previously established pattern. Children with different histories, interpret, react to, and create different experiences (e.g., Dodge, Pettit, Bates, & Valente, 1995).

Psychopathology From the Ground Up

Work on patterns of attachment in infancy provides a glimpse of a new developmental approach to disorder, an approach that begins with early patterns of adaptation and goes forward, rather than the other way around (Sroufe et al., 2005). As they develop, those with histories of avoidant and resistant attachment show many similar problems, broadly speaking. They are ineffective with peers, dependent on teachers, inflexible, and generally not well regulated. Both groups are even aggressive. However, the quality and patterning of their behavior is quite different. Those with avoidant histories are self-isolated and are rejected by peers because of their aloofness or "mean" behavior; whereas those with resistant histories want to be with others, hover around the group, and simply fail to be well accepted because of their ineptness and social immaturity (Sroufe, 1983). For those with resistant histories, dependency on teachers is straightforward, as they continually seek them out. Conversely, those with avoidant histories are very indirect in seeking contact and specifically do not go to teachers when hurt or upset. However, children in both groups wind up sitting next to teachers more often in circle time than children with secure histories. The aggression of children in the two groups likewise is of vastly different quality. Those with resistant histories tend to flail out when frustrated or upset, which happens to them easily. Only those with avoidant histories are likely to engage in deliberate, calculated aggression, for example, poking a child in the stomach after she says she has a stomachache (Kestenbaum, Farber, & Sroufe, 1989).

These patterns are interesting both because of their origins and because of the outcomes with which they are associated. Those with resistant histories often have experienced inconsistent or chaotic parenting. They cannot fully depend on reliable parent responses when they are threatened or needy. They have adapted to this circumstance by setting the threshold for threat lower, by being chronically wary and vigilant, by hovering near the parent, by going to them at the slightest provocation, and in general, by expressing attachment needs with great intensity (Main, 1990). This helps to fill the gap left by the parent and is adaptive in that sense, but it is at the expense of exploration and the emergence of autonomy. Without adequate parental assistance in developing emotion regulation and without a history of effective play and discovery, they make poor play partners in preschool.

In contrast, those with avoidant histories evolve a strategy of "minimizing" attachment (Main, 1990). In the face of chronic rebuff and rejection, especially in circumstances where the infant brings a tender need to the caregiver, these children learn to cut off feelings and inhibit direct expressions of attachment. By not going to the caregiver when threatened, they forestall further rejection and can stay in the vicinity of the caregiver in the event of a more dire circumstance. However, they are left without a sense of deep connection with others, with low feelings of self-worth, and a self-fulfilling pattern of alienation and isolation. If they do not go to a teacher when disappointed or upset, they cannot discover that teachers will respond. Alienating other children and angering teachers with bullying behavior leaves them feeling more cut off from others as they go forward.

Let us now consider outcomes. The pattern of resistant attachment in infancy is uniquely related to anxiety disorders in adulthood whereas avoidance is not (Warren, Huston, Egeland, & Sroufe, 1997). The connection to anxiety is predictable given the early history. Those with resistant histories were obligated to retain some sense of safety by maintaining vigilance regarding threat and by reacting strongly when the slightest threat occurred. In marked contrast, avoidant histories predict later conduct problems, and there are some data showing that this is mediated by alienation (Egeland et al., 2002). It is interesting to note that there is no distinction with regard to depression; it is linked to both avoidance and resistance. Although we do not have adequate data here, I suggest that these findings reflect two distinctive pathways: through helplessness and anxiety in the case of resistance and through alienation, anger, and hopelessness in the case of avoidance. This hypothesis should be pursued.

Conclusion: A Path Forward

The developmental work on attachment suggests a different path forward from the continued search for correlates of DSM-based categories. The path would be one in which we begin with early patterns of adaptation, trace their courses and manifestations in different phases of development, and document when and how they may be manifest in disorder.

Although this task will be challenging, we are further along than first meets the eye. First, the approach using patterns of attachment can be readily expanded. It is not difficult to array an elaborated series of central developmental issues that all children face (see Table 1). There are some data indicating that individual variations with regard to these issues are linked age by age (Sroufe et al., 2005). Moreover, when teachers who were blind to early history grouped children into a set of profiles based on the preschool-age issues, it was shown that these were forecast by patterns of infant attachment that preceded them (Sroufe, 1983). Elaborating and describing variations in adaptation with regard to the complete set of issues would move us well beyond what has already been accomplished within an attachment framework alone.

Second, there is already wide agreement and a proliferation of research on the capacities at the center of all of these issues. These capacities lie at the core of healthy or unhealthy

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Table 1. *Salient issues of development*

Infancy
Major issue: formation of an effective attachment
Subsidiary issues
Basic state and arousal regulation
Development of reciprocity
Dyadic regulation of emotion
Toddler Period
Major issue: guided self-regulation
Subsidiary issues
Increased autonomy
Increased awareness of self and others
Awareness of standards for behavior
Self-conscious emotions
Preschool Period
Major issue: self-regulation
Subsidiary issues
Self-reliance with support (agency)
Self-management
Expanding social world
Internalization of rules and values
School Years
Major issue: competence
Subsidiary issues
Personal effectance
Self-integration
Competence with peers
Place in group
Functioning in group
Loyal friendships
Competence in school
Adolescence
Major issue: individuation
Subsidiary issues
Autonomy with connectedness
Identity
Peer network competence
Place in network
Functioning in network
Intimate relationships
Coordinating school, work, and social life
Transition to Adulthood
Major issue: emancipation
Subsidiary issues
Launching a life course
Financial responsibility
Adult social competence
Coordinating partnerships and friendships
Coordinating colleagues, partners, and friends
Stable partnerships
Coordinating work, training, career, and life

psychological development. They include the capacity for stable emotional connection with others, internalization of standards for conduct, and achieving flexible self-regulation. The latter is constituted by current hot topics in the field, such as emotion regulation, executive function, and effortful control. The centrality of these aspects of functioning is obvious, even using DSM categories as the criterion: Relationship and emotion regulation problems lie at the heart of all major DSM categories (Cole, Michel, & O'Connell-Teti, 1994; Sroufe, Duggal, Weinfield, & Carlson, 2000). Moreover, not only are these capacities well described, there is evidence that they are outgrowths of quality of care and patterns of adaptation in infancy (e.g., Bernier, Carlson, & Whipple, 2010; Kochanska, 2002; Sroufe et al., 2005; Sroufe, Schork, Motti, Lawroski, & LaFreniere, 1984).

What lie before us now are two tasks. The first is to fully understand the development of these core capacities and how their mastery may be supported or allowed to go off track. The second, and more challenging, is to evolve a lexicon of variation in patterns of adaptation with regard to their manifestation at each age. A primitive beginning was made by a working group at the Center for Advanced Study in the Behavioral Sciences three decades ago (Sameroff & Emde, 1989). In their system, what begin as patterns of relationship disturbance involving overregulation, underregulation, and various forms of dysregulation evolve to individual patterns of disturbance in childhood. Much more elaboration is clearly needed. It will require detailed longitudinal studies to define patterns of adaptation and trace them over time. However, such a "human adaptation project" will not require anything like the effort and cost of the human genome project and likely will bring us to a much greater understanding of human psychological health and disturbance.

It is an exciting time in the field of developmental psychopathology. We have learned much about brain development, gene expression and its relation to experience, and the role of early experience. We now have a more complete understanding that when there is disorder it is manifest in brain functioning, in expectations and appraisals, in emotional life, and in social relationships. We have made a start at explicating developmental processes and pathways to disorder. It is in carrying forward such work that developmental psychopathology will fulfill its promise of delivering a new way to conceptualize disorder, descriptions in terms of development and not simply manifest behaviors, and guidance for prevention and early intervention.

References

Aguilar, B., Sroufe, L. A., Egeland, B., & Carlson, E. (2000). Distinguishing the early-onset/persistent and adolescent-onset antisocial behavior types: From birth to 16 years. *Development and Psychopathology*, *12*, 109–132.

Appleyard, K., Egeland, B., van Dulmen, M., & Sroufe, L. A. (2005). When more is not better: The role of cumulative risk in child behavior outcomes. *Journal of Child Psychology and Psychiatry 46*, 235–245.

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin*, *135*, 885–908.

https://doi.org/10.1017/S0954579413000576 Published online by Cambridge University Press

Bernier, A., Carlson, S., & Whipple, N. (2010). From external regulation to self-regulation: Early parenting precursors of young children's executive functioning. *Child Development*, *81*, 326–339.

Bowlby, J. (1973). *Attachment and loss: Vol. 2. Separation*. New York: Basic Books.

Brazelton, T. B., Kowslowski, B., & Main, M. (1974). The origins of reciprocity: The early mother-input interaction. In M. Lewis & L. Rosenblum (Eds.), *The effect of the infant on its caregiver* (pp. 49–76). New York: Wiley.

Burnette, M. L., & Cicchetti, D. (Eds.). (2012). Multilevel approaches toward understanding the development of antisociality: Current research and future directions [Special issue]. *Development and Psychopathology*, *24*, 703–1155.

Carlson, E. A. (1998). A prospective longitudinal study of attachment disorganization/disorientation. *Child Development*, *69*, 1107–1128.

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

Carlson, E. A., Sroufe, L. A., & Egeland, B. (2004). The construction of experience: A longitudinal study of representation and behavior. *Child Development*, *75*, 66–83.

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., & Toth, S. L. (2005). Child maltreatment. *Annual Review of Clinical Psychology*, *1*, 409–438.

Cicchetti, D., & Valentino, K. (2006). An ecological–transactional perspective on child maltreatment: Failure of the average expectable environment and its influence on child development. In D. Cicchetti & D. Cohen (Eds.), *Development and psychopathology: Vol. 3. Risk, disorder, and adaptation* (2nd ed., pp. 129–201). New York: Wiley.

Cole, P. M., Michel, M. K., & O'Connell-Teti, L. (1994). The development of emotion regulation and dysregulation: A clinical perspective. *Monographs of the Society for Research in Child Development*, *59*(2–3, Serial No. 240).

Dodge, K., Pettit, G., Bates, J., & Valente, E. (1995). Social information-processing patterns partially mediate the effect of early physical abuse on later conduct problems. *Journal of Abnormal Psychology*, *104*, 632–643.

Duggal, S., Carlson, E. A., Sroufe, L. A., & Egeland, B. (2001). Depressive symptomatology in childhood and adolescence. *Development and Psychopathology*, *13*, 143–164.

Egeland, B., Kalkoske, M., Gottesman, N., & Erickson, M. (1990). Preschool behavior problems: Stability and factors accounting for change. *Journal of Child Psychology and Psychiatry*, *31*, 891–909.

Egeland, B., Yates, T., Appleyard, K., & van Dulmen, M. (2002). The long-term consequences of maltreatment in the early years: A developmental pathway model to antisocial behavior. *Children's Services: Social Policy, Research, and Practice*, *5*, 249–260.

Elder, G. Jr. (1986). Military times and turning points in men's lives. *Developmental Psychology*, *22*, 233–245.

Eron, L., & Huesman, L. (1990). The stability of aggressive behavior—Even into the third generation. In M. Lewis & S. Miller (Eds.), *Handbook of developmental psychopathology* (pp. 147–156). New York: Plenum Press.

Farrington, D. (1995). The Twelfth Jack Tizard Memorial Lecture: The development of offending and antisocial behavior in childhood: Key findings from the Cambridge Study of Delinquent Development. *Journal of Child Psychology and Psychiatry*, *36*, 929–964.

Fury, G., Carlson, E. A., & Sroufe, L. A. (1997). Children's representations of attachment relationships in family drawings. *Child Development*, *68*, 1154–1164.

Gilliom, M., & Shaw, D. (2004). Codevelopment of externalizing and internalizing problems in early childhood. *Development and Psychopathology*, *16*, 313–334.

Harrington, R., Rutter, M., & Fombonne, E. (1996). Developmental pathways in depression: Multiple meanings, antecedents, and endpoints. *Development and Psychopathology*, *8*, 601–618.

Jacobvitz, D., Hazen, N., & Riggs, S. (1997, April). *Disorganized mental processes in mothers, frightened/frightening caregiving, and disorganized/disoriented behavior in infancy.* Paper presented at the meeting of the Society for Research in Child Development, Washington, DC.

Jimerson, S., Egeland, B., Sroufe, L. A., & Carlson, E. A. (2000). A prospective, longitudinal study of high-school dropouts: Examining multiple predictors across development. *Journal of School Psychology*, *38*, 525–549.

Kaffman, A., & Meaney, M. (2007). Neurodevelopmental sequelae of postnatal maternal care in rodents: Clinical and research implications of molecular insights. *Journal of Child Psychology and Psychiatry*, *48*, 224–244.

Kagan, J. (1984). *The nature of the child*. New York: Basic Books.

Kestenbaum, R., Farber, E., & Sroufe, L. A. (1989). Individual differences in empathy among preschoolers: Concurrent and predictive validity. In N. Eisenberg (Ed.), *Empathy and related emotional responses* (pp. 51–56). San Francisco, CA: Jossey–Bass.

Kochanska, G. (2002). Mutually responsive orientation between mothers and their young children: A context for the early development of conscience. *Current Directions in Psychological Science*, *11*, 191–195.

Luby, J., Barch, D., Belden, A., Gaffrey, M., Tillman, R., Babb, C., et al. (2012). Maternal support in early childhood predicts larger hippocampal volumes at school age. *Proceedings of the National Academy of Sciences*, *109*, 2854–2859.

Main, M. (1990). Cross-cultural studies of attachment organization: Recent studies, changing methodologies, and the concept of conditional strategies. *Human Development*, *33*, 48–61.

Marshall, P., Reeb, B, Fox, N., Nelson, C., & Zeanah, C. (2008). Effects of early intervention on EEG power and coherence in previously institutionalized children in Romania. *Development and Psychopathology*, *20*, 861–880.

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

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

Novak, M., O'Neill, P., Beckley, S., & Suomi, S. (1992). Naturalistic environments for captive primates. In E. Gibbons, E. Wyers, & E. Waters (Eds.), *Naturalistic habitats in captivity* (pp. 236–258). New York: Academic Press.

Ogawa, J. R., Sroufe, L. A., Weinfield, N. S., Carlson, E. A., & Egeland, B. (1997). Development and the fragmented self: Longitudinal study of dissociative symptomatology in a nonclinical sample. *Development and Psychopathology*, *9*, 855–879.

Patterson, G., & Dishion, T. (1988). A mechanism for transmitting the antisocial trait across generations. In R. Hinde & J. Stevenson-Hinde (Eds.), *Relations between relationships within families* (pp. 283–310). Oxford: Oxford University Press.

Pianta, R. C., Egeland, B., & Sroufe, L. A. (1990). Maternal stress and children's development: Prediction of school outcomes and identification of protective factors. In J. Rolf, A. S. Masten, D. Cicchetti, K. H. Neuchterlein, & S. Weintraub (Eds.), *Risk and protective factors in the development of psychopathology* (pp. 215–235). New York: Cambridge University Press.

Read, J., Perry, D. Moskowitz, A., & Connolly, J. (2001). The contribution of early traumatic events to schizophrenia in some patients: A traumagenic neurodevelopmental model. *Psychiatry: Interpersonal and Biological Processes*, *64*, 319–345.

Robins, L. (1966). *Deviant children grown up*. Baltimore, MD: Williams & Wilkins.

Robins, L., & Price, R. (1991). Adult disorders predicted by childhood conduct problems: Results from the NIMH Epidemiological Catchment Area project. *Psychiatry*, *54*, 116–132.

Roisman, G., Aguilar, B., & Egeland, B. (2004). Antisocial behavior in the transition to adulthood: The independent and interactive roles of developmental history and emerging developmental tasks. *Development and Psychopathology*, *16*, 857–871.

Rutter, M. (1996). Transitions and turning points in developmental psychopathology: As applied to the age span between childhood and mid-adulthood. *International Journal of Behavioral Development*, *19*, 603–626.

Rutter, M. (2013). Developmental psychopathology: A paradigm shift or just a relabeling? *Development and Psychopathology*, *25*(4, pt. 2), 1201–1213.

Sackett, J., Sameroff, A., Cairns, R., & Suomi, S. (1981). Continuity in behavioral development. In K. Immelman, G. Barlow, I. Petrinovitch & M. Main (Eds.), *Behavioral development* (pp. 23–57). Cambridge: Cambridge University Press.

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

Sander, L. (1975). Infant and caretaking environment. In E. J. Anthony (Ed.), *Explorations in child psychiatry* (pp. 129–165). New York: Plenum Press.

Sroufe, L. A. (1983). Infant–caregiver attachment and patterns of adaptation in preschool: The roots of maladaptation and competence. In M. Perlmutter (Ed.), *Minnesota symposia on child psychology* (Vol. 16, pp. 129–135). Hillsdale, NJ: Erlbaum.

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

Sroufe, L. A. (1997). Psychopathology as an outcome of development. *Development and Psychopathology*, *9*, 251–268.

Sroufe, L. A. (1999). *Changing the odds: The development of resilience.* Paper presented at the meeting of the American Association for the Advancement of Science, Anaheim, CA.

Sroufe, L. A., Duggal, S., Weinfield, N. S., & Carlson, E. (2000). Relationships, development, and psychopathology. In M. L. A. Sameroff (Ed.), *Handbook of developmental psychopathology* (2nd ed., pp. 75–92). New York: Kluwer Academic/Plenum Press.

Sroufe, L. A., Egeland, B., Carlson, E., & Collins, W. A. (2005). *The development of the person*. New York: Guilford Press.

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., & Fleeson, J. (1988). The coherence of family relationships. In R. A. Hinde & J. Stevenson-Hinde (Eds.), *Relationships within families: Mutual influences* (pp. 27–47). Oxford: Oxford University Press.

Sroufe, L. A., Jacobvitz, J., Mangelsdorf, S., deAngelo, E., & Ward, M. J. (1985). Generational boundary dissolution between mothers and their preschool children: A relationships systems approach. *Child Development*, *56*, 317–325.

Sroufe, L. A., & Pierce, S. (1999). Men in the family: Associations with juvenile conduct. In G. Cunningham (Ed.), *Just in time research: Children, youth and families* (pp. 19–26). Minneapolis, MN: University of Minnesota Extension Service.

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

Sroufe, L. A., Schork, E., Motti, F., Lawroski, N., & LaFreniere, P. (1984). The role of affect in social competence. In C. E. Izard, J. Kagan, & R. Zajonc (Eds.), *Emotions, cognition, and behavior* (pp. 289–319). New York: Plenum Press.

Stiles, J. (2008). *Fundamentals of brain development*. Cambridge, MA: Harvard University Press.

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 Behavioural Development*, *15*, 43–65.

Supkoff, L., Puig, J., & Sroufe, L. A. (2012). Situating resilience in developmental context. In M. Ungar (Ed.), *The social ecology of resilience* (pp. 127–142). New York: Springer.

Toth, S., Maughan, A., Manly, J., Spagnola, M., & Cicchetti, D. (2002). The relative efficacy of two interventions in altering maltreated children's representational models: Implications for attachment theory. *Development and Psychopathology*, *14*, 877–908.

Warren, S., Huston, L., Egeland, B., & Sroufe, L. A. (1997). Child and adolescent anxiety disorders and early attachment. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*, 637–644.

Weaver, C., Meaney, M., & Szyf, M. (2005). Maternal care effects on the hippocampal transcriptome and anxiety-mediated behaviors in the offspring that are reversible in adulthood. *Proceedings of the National Academy of Sciences*, *103*, 3480–3485.

Werner, H. (1948). *The comparative psychology of mental development*. New York: International Universities Press.