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## The Concept of Development in Developmental Psychopathology

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### Abstract

So important is the perspective of development for understanding psychopathology that it spawned a new discipline—“developmental psychopathology”—which has seen remarkable advances since its introduction, but has yet to completely fulfill its promise. To do this requires maintaining a thoroughgoing developmental perspective. When we take development seriously, there are implications for how we understand psychopathology, describe and conceptualize the origins and course of disorder, and interpret research findings. From this perspective, disorders are complex products of development; for example, we can view neurophysiological associates of disorder not as causes but as markers, the development of which we need to understand. Research on developmental psychopathology requires an examination of the history of problem behavior from early in life, and it unites multiple features of adaptation and maladaptation (contextual, experiential, physiological, and genetic).

### Keywords

Development; Psychopathology; Adaptation; Maladaptation

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Developmental psychopathology is a young discipline. When Achenbach published his pioneering text in 1974, he said this field “hardly yet exists.” He was correct, because whereas abnormal psychology and child psychiatry were well established, the new discipline had not yet been distinguished from each of these. Its goal was to integrate these disciplines, as well as developmental psychology, encompassing both normal and atypical development. It was a decade before a special issue of *Child Development*, edited by Dante Cicchetti (1984), fully articulated this perspective, defining the field as “the study of the origins and course of individual patterns of behavioral maladaptation, whatever the age of onset, whatever the causes, ... however complex the course of the developmental pattern may be” (Sroufe & Rutter, 1984, p. 18). The discipline was not restricted to study of child problems, or even problems alone, but dealt with the comparative study of patterns of adaptation over time. As Michael Rutter said at an NIMH conference on depression, “developmental psychopathology is first and foremost about the study of development.”

This article briefly describes some of the extraordinary accomplishments of this young discipline, and then raises some concerns about its current course and future. The progress is beyond what anyone could have imagined 25 years ago, yet this dynamic field has not yet completely fulfilled its twin promises of (1) identifying premorbid patterns of maladaptation that allow targeted early intervention and prevention, and (2) yielding a classification system informed by empirical study of individual development from the ground up, rather than

simply a downward extension of adult categories of disturbance or acceptance of clinic-derived child entities. I argue that the key to moving forward is to maintain a thoroughgoing developmental perspective.

I want to be clear that both empirical and conceptual progress in this field has been enormous. Scholars have published prominent handbooks, and a prestigious journal devoted specifically to this field is firmly established. Thousands of articles, covering every feature of functioning from molecular to molar, have furthered the understanding of the origins and course of disorder. Research has dealt with the correlates of child problems, ranging from neurophysiological markers to laboratory assessments of child behavior to social context. Many of these investigations have been informed by studies of normal development, as this field prescribes. Thus, as we have discovered the centrality of certain capacities in normal development and how they are integrated (e.g., Eisenberg, et al., 2003), these have become central to the study of childhood problems (e.g., Nigg, 2006). Such correlation research is critical for understanding the integrated nature of development, and it is important background for further developmental study. We can now ask, for example, *how* executive function develops, whether it precedes or follows other features of child problems, and how various features of particular problems come to be patterned over time.

Moreover, there are now empirical demonstrations that we can predict both child and adult disorders using a variety of risk factors, and that antecedent-consequent linkages are often complex. Investigators have demonstrated transformations of manifest behavior across developmental periods (for example, the pathway from extreme, age-inappropriate oppositional behavior to conduct problems to antisocial personality disorder; e.g., Loeber et al., 1993). We know much about the interplay of risk and protective factors, and we know that similar processes underlie vulnerability to disorder and resilience (Sroufe, 2007). Finally, there is evidence that trajectories of behavior may be more significant than manifest problems at a given time. For example, adolescent conduct problems without a history of problem behavior may not be as significant as continuous problems beginning in childhood (Moffitt, 1993). Others showed that the factors that initiate disordered trajectories may be different than ones that maintain them, and that problems (such as depression) that appear to be similar in childhood and adulthood might represent different disorders, because they have distinctive antecedents and/or different outcomes (e.g., Duggal, Carlson, Sroufe, & Egeland, 2001; Quevedo, 2008).

There have been notable conceptual advances as well. Scholars have widely embraced the probabilistic, systemic, context-based, multilevel nature of psychopathology (e.g., Cicchetti, 2007b; Masten, 2007). New developmental concepts are now central, such as developmental pathways, multi- and equifinality, heterotypic continuity, and lawful discontinuity. That the same risk can lead to multiple problems, and that there are multiple pathways to the same problem, points us to developmental process studies. At the same time, the discipline has become truly multidisciplinary. Those concerned primarily with environmental features of development have become interested in gene-environment interaction (e.g., Belsky, Bakersman-Kranenburg, & van IJzendoorn, 2007; Bakersman-Kranenburg & van IJzendoorn, 2007), whereas those interested in biological bases of behavior or temperament have become concerned with the modifying role of environmental features (e.g., Rothbart & Bates, 2006). Recently, an entire issue of *Development and Psychopathology* was devoted to gene-environment interaction (Cicchetti, 2007a). Our expanding knowledge of brain functioning and the clear demonstrations of experience-dependent brain development support such integrative efforts.

It would be easy to extend this list of accomplishments, as both the effort and the output in this field have been extraordinary. Despite this progress, however, a counter-trend has

occasionally arisen that at times erodes a developmental perspective: the tendency to treat biological correlates of functioning, and even characteristic child behavior, as somehow lying outside of the domain of development—of simply being endogenous givens. Reflecting this, the vast majority of current studies in the psychiatric literature simply compare those who do and do not qualify for some disorder on some biological variable (see, for example, the review of fMRI studies of ADHD by Dickstein, Bannon, Castellanos, & Milham, 2006). Given the integrated nature of development, it is impossible to avoid discovering large numbers of such differences. Seriously disturbed functioning will doubtless manifest in brain/body just as in interpersonal relationships and daily functioning.

However, studies that reveal some difference in brain physiology between two groups, even those that use children, cannot answer the basic developmental questions of origins and course, but can only stimulate developmental inquiry. We can spend \$21 million on the neurophysiological correlates of anorexia, as Chavez and Insel (2007) describe, and learn almost nothing about how individuals develop this problem. This is not because genes and neurophysiology are unimportant, but because they derive their importance within complex developmental analysis. Only developmental studies can reveal whether obtained associates are true antecedents, markers, or consequences of a disordered process. Experience influences physiology just as physiology influences experience (Grossman et al., 2003). Only developmental studies can reveal how genetic or physiological features interplay with the other features of a developing problem.

If there is an overall message from our 30-year study of individual adaptation, it is that persons develop (Sroufe, Egeland, Carlson, & Collins, 2005). We are not simply born to be who we become. Our patterns of adaptation and maladaptation, our particular liabilities and strengths, whether and how we are vulnerable or resilient—all are complex products of a lengthy developmental process. Likewise, the forms of psychopathology that any of us show are developmental outcomes (Sroufe, 1997). Maintaining a developmental perspective is crucial to continued progress in the field; it guides us toward more penetrating questions and a deeper understanding of disturbance. To substantiate this claim, I need to first say a few words about the nature of development.

## THE NATURE OF DEVELOPMENT

Whether considering growth of the embryo, formation of the brain, or the emergence of the personality, development always is governed by certain principles. Notably, development is “hierarchically integrated” (Werner & Kaplan, 1963) or “cumulative” (Sroufe & Cooper, 1988). Later forms build on earlier foundations that are themselves transformed in the process. This cumulative feature of development makes clear that there are *three* determinants of behavior and development: genes, environment, and past development.

This neglected third feature is critical. Consider development of a chick embryo. If, at an early stage when leg and wing buds are just emerging, one removes a bit of tissue from the base of the leg bud and places it at the tip of the wing bud, there can be an interesting result. This tissue, which if left alone would have differentiated to become part of a thigh, now becomes a normal-looking part of the wing tip. (This is an environmental effect, because the surrounding cells “induce” it to become part of the wing by turning certain genes on or off; Arms & Camp, 1987; Rutter, 2007). If the transfer occurs a bit later, it does not take; that is, anomalous flesh grows at the tip of the wing. This is because the tissue was already “committed” to becoming leg tissue at this phase. Most interesting, if the transfer occurs at a precise point—not too early or late—one gets an amazing result. The tissue becomes neither normal wing nor anomalous leg tissue, but a claw! This is because the incipient thigh tissue is already committed to becoming leg tissue, but it is not fully committed to becoming thigh.

The surrounding cells can still induce it to become a tip, not the tip of a wing but rather the tip of a leg, a claw. This illustration supports each feature of development. Genes are involved; a fin does not result in any case. Context too is important, in this case the surrounding cells. But so is past development. The intervening event has notably different impact depending on when it happens; that is, depending on the prior development of the organism. History conditions the impact of later developmental inputs. It is never just genes and environment but always genes, environment, and history that determine growth. Once time enters the picture, there is organism, and it is the organism that then interacts with environment. Recent discoveries concerning methylation make clear that even genetic effects are influenced by past experience and the environmental changes it creates, underscoring further the role of history (e.g., Kaffman & Meany, 2007).

To fully understand the origins and course of psychopathology, we need to understand the developmental history of the person. This is not always obvious, because developmental linkages are often complex. Not only is there heterotypic continuity, in which the same characteristic or tendency may be manifest in different ways over time, there is also developmental coherence of patterns of adaptation. Thus, for example, conduct problems in childhood may predict adult depression better than does childhood depression (Robins & Price, 1991). Such complexities are what made the discipline of developmental psychopathology necessary.

## IMPLICATIONS OF A DEVELOPMENTAL PERSPECTIVE

A developmental perspective alters description of concepts, thinking about causation, and interpretation of research findings. It fundamentally changes the research agenda. We view etiological factors within a complex causal network. Interactive systems concepts replace linear causal thinking. Scholars widely embrace these ideas; nonetheless, we often slip into single pathogen, linear causal language. We grant putative physiological features privileged causal status. (He is impulsive because he has an executive function defect. He is shy because of inhibited temperament.)

### Concepts and Interpretations

Outside of a developmental perspective, researchers often describe genetic features in causal terms. In behavior genetic studies, this can mean equating “heritable” with “genetic” and then claiming explanation. Numerous papers report, for example, that Attention Deficit/Hyperactivity Disorder shows a heritability of 0.70, and then conclude that it is “largely genetic.” Such interpretations, in addition to being fallacious, discourage developmental study in general and the search for environmental features in particular. First, any gene-environment interaction that is present is included in the genetic component in such analyses. Second, heritability estimates inversely vary with environmental diversity. (With one twin reared in a Romanian orphanage and the other in a supportive home environment, H squared would approach 0 for most psychological traits. All we can really conclude is that H squared is not 0; Turkheimer, 1998.) Finally, the 0.70 figure is based on parent reports, wherein dizygotic twins are generally rated as more dissimilar than they should be, thus distorting the ratio. Using teacher reports, the estimate is 0.50, and using observations it is merely 0.20 (see Sroufe, 2007 for more detail). Our own longitudinal data show a clear role for environmental features, including intrusive and overstimulating early parenting, in the development of ADHD (Carlson, Jacobvitz, & Sroufe, 1995). Environment is important, as are genes. But one is not more important than the other.

Even recent molecular genetic studies, which importantly illustrate gene-environment interaction, often grant a privileged status to genes. For example, researchers have interpreted the report of Caspi and colleagues (2002)—on the interaction between

polymorphisms of MAO-A and maltreatment with regard to conduct problems—to mean (and only mean) that maltreatment *only* has an effect for genetically vulnerable children; that is, that this genetic variation enables (“moderates”) the effects of maltreatment. Whereas this was one valid interpretation of this particular data set, it is misleading for several reasons. The graphed data show a crossover interaction (Sroufe, 2007). It would be just as valid to say that MAO-A gene variation is associated with misconduct only for maltreated children; it is also possible that this same variation conveys an advantage for well-nurtured children. Thus, it is not a genetic “defect” at all, simply genetic variation. Those arguing that a major impact of some genetic variation is to make individuals more susceptible to *environmental* influence, “for better or worse,” have made this point strongly (e.g., Belsky et al., 2007). This work gives an important direction for developmental study. (See Suomi, 2002, for compelling data of this type with Rhesus monkeys.) Finally, numerous replications of the Caspi study find a main effect for maltreatment (see Taylor & Kim-Cohen, 2007, for a review), and, further, maltreatment has a range of associated outcomes other than conduct problems, so it is doubly shortsighted to imply that maltreatment has an effect only for genetically vulnerable children. Maltreatment will always have consequences. As with any feature of development, what these consequences are, and how profound they will be, depends on a host of other factors in play during development.

Discussion of temperament, or individual variation in general, also sometimes shows an erosion of developmental thinking. Concepts such as reactivity thresholds, and more current constructs such as executive function, effortful control, emotionality, and self-regulation, are of vital interest to developmental psychopathologists. Used descriptively, simply as patterns of variation, they can fit within a developmental framework. Researchers have shown, for example, that externalizing children have less self-control, that internalizing children tend to be shy, that “difficult temperament” is associated with a range of psychiatric problems, that teens high on self-regulation are less influenced by deviant peers, and, in general, that children show varied reactions to what are apparently the same stressors, among many other findings (see Nigg, 2006; Sroufe, 2007 for reviews). From a developmental perspective, such replicable findings lead not to conclusions but to a series of questions. What are the origins of these characteristics? How do they evolve over time? Are they detectable prior to and independent from the measures of disturbance? What is their place in more integrative patterns of adaptation that forecast disorder? There are many examples of researchers approaching such questions thoughtfully (see, e.g., Rothbart & Bates, 2006, for a review).

But the majority of reports on temperament and psychopathology use these concepts causally. They conclude, for example, that children have conduct problems *because* they lack effortful control, even though both are measured at the same age and may simply result from a common developmental process. Likewise, researchers interpret variations in reaction to stress as attributable to endogenous sensitivities, which may discourage further study. They label observed impulsiveness as “temperamental impulsiveness,” as though the behavior has now been explained. “Child effects,” which simply means that some measure of child variation correlates with some measure of environmental variation, serve as putative evidence of inherent child differences that influence development. Individual variation of almost any kind, even if observed in adolescence and labeled as temperament, is assumed to be largely endogenous (though “modifiable”) and then is used to explain the outcome. Physiological correlates of temperament are used to support such arguments, but are actually evidence for the integrated nature of development, not that temperamental variation is due to endogenous differences. This causal thinking cuts off developmental study. How children come to have differential sensitivities in fact needs continued investigation.

Our own study shows that self-regulation, behavioral control, expressed emotion, and executive function capacities are all developmental achievements, based on a substantial



history and a host of factors (Sroufe et al., 2005). Even “child effects” in general develop; that is, the power of child variation to predict later behavior increases with age. Direct measures of child functioning obtained after age 3 predict later disturbance better than do measures obtained during the earliest years, when measures of parenting and parent-child relationships are stronger.

Let me be clear that these concerns about language usage also apply to those studying environmental features of development. For example, when attachment researchers like myself slip and refer to a child as “secure,” when all we have is an assessment of a specific attachment *relationship* (usually at some earlier time), we too are implying more than we know.

### The Research Agenda

Maintaining a developmental perspective alters the research agenda. Psychopathology is not a condition that some individuals simply have or are born to have; rather, it is the outcome of a developmental process. It derives from the successive adaptations of individuals in their environment across time, each adaptation providing a foundation for the next. Thus, of interest are converging processes that initiate a maladaptive pathway and processes that keep individuals on the pathway to disorder or deflect them back toward normal functioning.

There are numerous implications of this pathways model. One priority is to identify early patterns of maladaptation that, although not properly viewed as disorder, nonetheless are probabilistically related to later disorder. Such identification will be crucial for prevention efforts. Second, investigators must identify the convergence of factors that promote development of this early pattern. Timing is important in such an analysis. Our research shows that adversities such as maltreatment or witnessing parental violence have a more powerful effect for later pathology when they occur in the preschool years than when they occur in middle childhood (e.g., Appleyard, Egeland, van Dulmen, & Sroufe, 2005; Yates, Dodds, Sroufe, & Egeland, 2003). Other work suggests that, in general, trauma has more devastating consequences for young children than for older children or adults (Perry & Szalavitz, 2006), and that cumulative adversity has more negative consequences than early adversity alone (e.g., Ogawa, Sroufe, Weinfield, Carlson, & Egeland, 1997). Early experience and early adaptation come to the fore within a developmental perspective because of the role of initiating conditions in transactional, systems models. Subsequent influences are, in part, conditioned by the adaptation already forged.

Further questions concern how patterns tend to be perpetuated, and how intervening factors can disrupt such a course? How do factors promoting continuity or deflection back toward normality change over time, and what promotes desistance following onset of full-fledged disorder? In particular, do different factors promote desistance for individuals arriving at a common disorder via distinctive developmental pathways? A developmental pathways model leads to a broader view of intervention and treatment than does a pathogen model. Helping a child return to a more positive pathway is a different therapeutic process than “treating” the symptoms of a disorder.

Much current research is simply two-group research that begins with a disorder and proceeds backward to find its cause. Thus, for example, investigators compare children who meet the criteria for ADHD with children who do not on some variable, usually a physiological measure or a behavioral measure presumed to reflect brain dysfunction (Dickstein et al., 2006). Such studies are mute regarding development. A developmental perspective, on the other hand, suggests forward-going research beginning with early adaptation. Researchers study how attention and self-regulation capacities normally develop, beginning in infancy, and investigate various factors that may disrupt these normal

pathways, including parenting, life stress, and other environmental features. Such study has shown that the development of ADHD is predictable long before the child meets the criteria for diagnosis, and that environmental change can account for changing manifestation of ADHD problems (e.g., Carlson et al., 1995). Physiological correlates of disorder, of course, remain important in a developmental approach. They may help confirm the disordered pathway, may indicate that such a pattern is emerging, and may converge with other factors to initiate or maintain a pathway. One shortcoming of our longitudinal study is that we did not have early physiological variables, and were we to start such a study now we would certainly include them. We did find that infant temperament variables, although not often predictive of disturbance by themselves, did have important interactive effects with experiential variables (Sroufe et al., 2005).

Like variations in parenting, physiological correlates also suggest hypotheses for developmental study, but only prospective, longitudinal research, beginning in infancy, can reveal the processes leading to disorder. Currently, neither scholars nor government agencies are prioritizing research that proceeds from early patterns of adaptation to later problems. This would provide a new approach to a more serviceable system for classification.

## CONCLUSION

All phenomena of interest to developmental psychopathologists result from complex processes, involving multiple features operating over time. Everything develops. This includes executive competence, effortful control, behavior inhibition, EEG asymmetry, and biochemical imbalances in the brain. When we uncover an association between one of these variables and psychopathology, this should launch a developmental inquiry, not lead to causal conclusions. At the least, it is important to know if the physiological or behavioral indicator was in fact antecedent to emergent pathology, how this marker itself developed, and what developmental process links it to psychopathology. In our longitudinal work, we found that attachment history predicted later functioning far better when we combined it with other measures of parenting and a host of contextual factors. We argued that this did not trivialize the importance of attachment. Likewise, a developmental perspective does not trivialize the role of genes, neurophysiology, or temperament in the study of psychopathology. Rather, they too will receive enhanced importance when placed within a developmental perspective.

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