*Chapter* 9

# The Place of Development in Developmental Psychopathology

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When Michael Rutter was asked at a meeting 25 years ago for his definition of "developmental psychopathology," he said that it is, as the name says, first and foremost about development. And so it remains today. After all, "the process of development constitutes the crucial link between genetic . . . and environmental variables, between sociology and individual psychology, and between physiogenic and psychogenic causes" (Rutter, 1980, p. 1).

The chapters in this book are remarkable, each beautifully written and informative. Moreover, they are in accord in making two points of profound importance for developmental psychopathology. First, repeatedly and convincingly, they make the point that developmental psychopathology is necessarily a multidisciplinary field. The argument was for *multiple* levels of analysis, not simply different levels. No one argued that the level of focus in his or her chapter was more important than some other level, but rather that it gained in importance when considered in concert with another level of analysis. Thus, for example, the role of environment is not diminished when considered in concert with genes, but amplified. Determining the true nature of development is not a zero-sum game. The second point is closely related. Behavior, disorder, and development are always the result of interdependence, codetermination, or co-actions among multiple levels of influence (see also Gottlieb & Halpern, 2002). Although the terms may have been somewhat different, all authors in this book described an adaptational process wherein child influences environment and environment influences child in an ongoing way. All recognized the inappropriateness of

linear causal models in which some single pathogen is linked ineluctably to a singular outcome or in which there is an artificial separation of mind and body, genes and experience, or child and surrounding context. As Rutter (chapter 1) put it, for example: ". . . the traditional neat and tidy subdivision into genetic effects and environmental effects has broken down" (p. 20). X-REF

In addition, each of the chapters provided important examples of the place of development in a multilevel approach to psychopathology. Rutter (chapter 1) provides a complex model of development in medicine, wherein, for example, changes that in themselves are benign initiate a course of development that leads to disease (as in malignant tumors), or where there is heterotypic continuity in development, in which distinctly different risk factors are potent at different points in the developmental process (as in heart disease). He also gives numerous examples of co-action, including the classic studies of Cadoret and Tienari, which show that the conjoint effects of genetic and environmental liability are far beyond those of either alone. Finally, he provides examples from his work that even disorders widely believed to have genetic involvement, such as schizophrenia, are subject to notable environmental influence.

In chapter 2, Hanson and Gottesman enliven the once more static concepts of "reaction range" and "diathesis-stress" through use of the developmental concept of adaptation. Neither diathesis nor potentials are set at birth, and the reaction range becomes a "reaction surface" in this evolved view. "Human development is more than an interaction term in an analysis of genetic and environmental variances" (p. 32). The dynamic concept of X-REF epigenesis continues to evolve and now includes environmental effects at every level. These authors, as well as Rutter (chapter 1) and Boyce (chapter 3), discuss the exciting new research on methylation that shows that early experience can have lasting effects, not only on behavior but also on the physiological stress reactivity system and on the turning on and off of the glucocorticoid receptor genes themselves. "The previously spurned concept of the inheritance of acquired characteristics is resurfacing at the molecular level . . . but now based on credible data" (p. 32). X-REF

Boyce (chapter 3) presents a general model in which reactive children in high stress environments are placed on long-term trajectories toward physical and mental health problems. Thus, genetic vulnerability would increase the salience of environmental adversity, not reduce it. Moreover, the model is even more complex than first implied because "biological sensitivity to context may not only moderate associations between social context and health, but may be itself a product of early social contextual influences" (p. 63). Biological sensitivity to context is not simply a cause but is an out- X-REF come as well.

Such thinking is consistent with the nonlinear, systemic thinking that runs throughout this book.

Egeland (chapter 4) uses longitudinal data from the Minnesota Parent–Child Project to illustrate the "organized" nature of development. Pathology and resilience are viewed as dynamic constructs, being based not just on current individual characteristics or current circumstances, but on foundations established through prior experience and early adaptation. In addition, change in problem behavior is linked to change in parental stress or parental depression. Such change data make clear that links between parent problems and child behavior are not simply due to genetics. As do other chapters in the volume, the Egeland chapter also illustrates pathways concepts; here, for example, a pathway from maltreatment to alienation to conduct problems. Finally, Egeland provides critical data on the importance of timing. He shows, for example, that early trauma may predict adult problems, such as dissociation, even after accounting for middle childhood problems, and that early trauma is a more powerful predictor of adolescent and adult dissociation than is later trauma, likely because of the young child's lesser capacity to integrate disparate and challenging aspects of experience.

In chapter 5, Fiese and Spagnola adopt many of the same perspectives used by Egeland to look at the level of the total family. Thus, it is family features that are risk or promotive factors, and it is family practices and rituals that are interiorized by the child, in addition to dyadic, parent–child factors. Reciprocal influences between child and family are described. Serious illness of the child (asthma is their example) clearly represents a challenge to the family, but different families are organized differently to respond to this challenge. When routine and affectively positive family practices can be continued and the positive family representation can accommodate an ill child, necessary medical regimens are followed and fewer internalizing disorders result. In contrast, "in cases where the family perceives daily care as a drain on personal resources, children worry more about their asthma symptoms (and) report . . . that their daily activities are often disrupted by health symptoms" (pp. 134–135).

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Dishion and Piehler (chapter 6) present an exquisite example of developmental process, wherein children with established problem behavior develop associations with deviant peers, which then creates a progressive amplification or cascade of antisocial problems. The work is important for the field both because it illustrates the importance of understanding normal developmental phenomena (here, friendships and peer networks) and because such change in problem behavior cannot be readily assimilated to a simple causal model. A true transaction is illustrated. There is no question that antisocial children increasingly associate with deviant peers, that such friendships operate in distinctive ways, and that over time they promote increased individual problems, which in turn consolidate deviant peer associations. A simple defect model is not adequate because level of social skills

and the capacity for mutual influence seem equivalent for deviant and nondeviant peer friendships. But in this case mutuality, through mutual support of deviance, promotes escalating antisocial behavior.

Garber (chapter 7) recapitulated many of these themes in adopting a multilevel view of a particular disorder, depression. She illustrates clearly that depression in not appropriately considered as simply an organic disorder, but rather is best understood in light of physiological, cognitive, and social features. As Rutter (chapter 1) said, "a subdivision of mental disorders into those that are 'medical' and those that are 'social' is totally meaningless" (p. 5).

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A comprehensive overview of the potential role for every level of analysis in understanding pathways to psychopathology is presented by Cicchetti and Valentino (chapter 8). Levels considered range from molecular genetics, acoustical startle, and neuroendocrine regulation, to emotional regulation, attention, memory and language, to self-representations, social relationships, and community. Their data reveal co-actions, such as powerful joint effects on problem behavior of parental maltreatment and living in violent neighborhoods, and the interplay of maltreatment, neuroendocrine regulation, and depression. Such multilevel analysis is used to explain diverging pathways associated with maltreatment over time. Their work also reveals developmental mediation effects. For example, the link between maltreatment and later peer problems was mediated by assessed cognitive control or understanding of negative affect. Complex paths also were shown, wherein negative representations undermined emotion regulation, which then compromised peer functioning.

In light of these splendid chapters, which move the field forward so nicely, what more then is there for a commentator to say? In this case, only to go further and to do so in a particular way; namely, by adopting an even more thoroughgoing developmental perspective. The bulk of this final chapter is devoted to laying out what this would mean and what the implications of a more thorough developmental view would be for thinking about and understanding psychopathology. Before doing that, I make some initial comments about the nature of development and its place in explaining behavior.

## THE NATURE OF DEVELOPMENT

All students are now taught that development and all behavior, whether normal or abnormal, results from the interaction of genes and environment. This marks an important advance over simpler ideas that genes cause behavior or that the environment simply washes over a passive organism. And yet it does not really capture the full complexity of development.

There is a critical third ingredient that is not yet fully appreciated. It is not just genes and environment, but genes, environment, and past development that set the stage for future development.

The necessity of this altered viewpoint is readily illustrated by examples from prenatal development (Arms & Camp, 1987; Kuo, 1967). For example, consider an early stage in the development of the chick embryo, when buds that will become legs and wings are just emerging. If one surgically removes a tiny piece of tissue from an area that would otherwise develop into thigh tissue and places it at the tip of the wing bud, it can become a normal part of the wing tip. The surrounding cells, it is argued, "induce" it to become wing tissue. Timing is crucial; the tissue must not yet be "committed" to becoming leg tissue. This is, of course, another example of genes being turned off and on, as discussed by Rutter, Hanson & Gottesman, and Boyce (this volume). But because of the timing issue, one also already sees a role for past development. If one does this transfer too late, a glob of anomalous tissue at the tip of the wing results. More remarkably, if one does the transfer at a very particular point, not as early as in the first experiment and not too late, an amazing result is possible. One gets not thigh tissue at the tip of the wing and not normal-looking wing tissue, but a *claw*. How can this be? Apparently, the transferred tissue already was committed to (differentiated toward) becoming leg tissue, but not fully committed to becoming thigh tissue. Thus, the surrounding wing-tip tissues (the surrounding context) could not alter it away from becoming a leg part; yet the new context could induce it to become a tip, so it becomes the tip of a leg—a claw.

In this example, we of course continue to see an important role for genes. After all, it does not become a fin. We also see a crucial role for the environment (here, surrounding tissues) and for the interaction of the two. But there is a critical role for development as well. The intervening event has a notably different impact depending on when it happens; that is, depending on the prior development of the organism.

These examples from prenatal life are not simply a metaphor for the nature of development. Rather, this is the way development always works. There is no reason to think that things progress any differently whether we are talking about the development of tissue, the brain, a cognitive capacity, temperament, or the personality. Once we realize the importance of the time dimension, of development itself, we can never see genes and environment in the same way again. Except perhaps for a micromoment in time in the very beginning of development, it is not gene–environment interaction that concerns us when we study whole, living systems. In the embryonic example just cited, it really was tissue–environment interaction. Most often, in developmental psychology, it is organism–environment interaction or person–environment interaction (Sroufe, Egeland, Carlson, & Collins, 2005). The organism is, after all, Genes × Environment × Time. Once

time is entered, there is organism, and it is the organism that then interacts with environment. Following birth, with development, there is the person, and it is the person that interacts with the environment. The cumulative history of the person in part determines the environment (through what the person engages, reacts to, elicits, and processes) and even, at least in part, the genes that are active and inactive at a given time. "Child effects" are not gene effects but person effects. Person–environment correlations come to the fore, not gene–environment correlations. All of this derives from the inclusion of development as not just an outcome but as also a critical feature of a causal system.

This embryology example not only makes clear the three ingredients of development, but it reveals the very nature of development as well. Development is lawful and orderly in a particular way; namely it is "hierarchically integrated" (Werner, 1948) or *cumulative*. This means that development always entails everything that went before and something more. The "something more" is the emerging complexity resulting from the co-actions of organism and environment. The emerging complexity is not specified by prior features, yet it is founded on them (Sroufe et al., 2005). Acquired capacities are retained yet changed in meaning when new capacities are acquired and organized with them into more complex wholes. Future development, however complexly transformed, builds on what was already there. The tissue, organism, or person reacts differently to what appears to be the same environmental circumstance at different points in development. And individuals react differently because of their individual histories. As Freud is reported to have said: "The human mind is from start to finish incapable of separating itself from its own experience but can only build upon that" (Rosen, 1989, p. 126).

Development is an ongoing transactional process—the organism (person) as developed to that point in time engages the environment in particular ways (selecting, "interpreting," reacting to, and eliciting), while at the same time the current context (including the engaged environment) transforms the organism. In a cyclical way, the further developed person now engages the environment in altered ways and continually is further influenced by the engaged environment.

Our evolving view of development is that although genetic endowment is never lost, it is never the same following the early organizations of the organism. Likewise, although any inborn physiological and behavioral tendencies are not lost, they are never the same once the person begins interacting with the surrounding environment. As Werner (1948) argued:

> The development of biological forms is expressed by an *increasing differentiation* of parts and an increasing subordination or *hierarchization*. Such a process of hierarchization means that for any organic structure the organization of the differentiated parts is a closed totality, an ordering and grouping of parts in terms of the whole organism. (p. 41)

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## IMPLICATIONS OF A DEVELOPMENTAL VIEWPOINT

Adopting a thoroughgoing developmental viewpoint has widespread implications for the study of psychopathology, altering all aspects of our enterprise. It would change our language—the way we describe phenomena and conceptualize the origins and course of disorder. It would change how we interpret and explain research findings. And it would profoundly change the research agenda.

### The Language We Use

Linear, reductionistic thinking is deeply ingrained in all of us, and we readily slip into describing cause in terms of individual traits rather than developmental systems. At the outset, I want to adopt the curved finger of accusation and say that attachment theorists, such as myself, are equally vulnerable to this problem. Frequently, we slip into using terms such as "securely attached child" when we know that attachment is really a relationship term, and the proper description would be "a child with a history of a secure relationship with the primary caregiver." We don't do it because this is unwieldy, and I think that often explains why we use, as well, terms such as "inherent," "predisposition," and "largely genetic." Such terms are not developmental constructs and although, generally, we don't mean it, these terms imply a linear, not thoroughly developmental view. We know genes are modified by environment and that temperament is subject to transformation and is developing from the start, but in our language we sometimes gloss over this.

In a developmental viewpoint, we would not use the term *heritable* as though we had explained the cause of something, even in part. Heritability estimates would not be taken to imply genetic cause. Following Turkheimer (1998), we know that all we really can conclude is that H squared is not 0. When we say something like, "The heritability of ADHD is .70" and imply or say that this problem is then largely genetic, we are forgetting about development. Heritability estimates always would fluctuate depending on the variation in environments sampled. With one member of monozygotic twin pairs reared in a group-care Romanian orphanage and the other in a stable, supportive family, with an extensive social support network, heritability of almost any psychological characteristic would be very low. Moreover, the genetic term in these computations also includes the interaction with environment, and generally it is not possible to extract the effect that is purely due to genes. As Piaget (1952) said regarding cognition and affect, the workings of genes and environment are "non-dissociable." Finally, in the case of ADHD, heritability estimates very widely, being about .70 for

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parent report, .50 for teacher report, and .20 for observational data. Our own prospective data on onset and course of ADHD (e.g., Carlson et al., 1995) make clear that there is a critical role for environment in this problem (see Research Agenda, later in this chapter). Genes are important. Environment is important. We cannot say one is more important than the other.

Cicchetti and Cannon (1999) make this same point when they say, "No component, subsystem, or level of organization possesses causal privilege in the developmental system . . ." (p. 377). Yet we often do slip into granting privileged causal status to biological features. Authors who at times provide elegant statements regarding the co-active, integrative, and systemic nature of disturbance will at the same time speak of genetic *influences,* speak of biological *predispositions,* and use temperament as a causal construct. We seem to more readily accept circular statements such as, "She is shy because she has a behaviorally inhibited temperament" than "She is shy because she has an unresponsive caregiver." We treat neurobiological explanations as more basic, fundamental, and important. They are basic, fundamental, and important, but not more so than other levels of explanation.

One concluding example of how use of language impacts our thinking comes from the important work by Boyce (chapter 3). He describes the compelling and clearly developmental idea that children are differentially sensitive to stressful environments; that is, that there is an interaction between individual stress reactivity and context. And all would agree that biological features would be an important consideration in such sensitivity. However, choosing to describe the phenomenon as "biological sensitivity to context" has the consequence of moving his exquisite developmental view into and out of focus. On one hand, he points out that "biological" sensitivity to context may be itself an outcome of early social contextual influences and that "*all* children are vulnerable in settings of . . . deprivation" (p. 62). These statements make clear his understanding of the transactional, systemic nature of development. On the other hand, in the same chapter, he refers to "inherent individual susceptibility" and to "dandelion children" who "survive and even thrive in whatever circumstances they encounter (p. 61)." They are "resilient," he argues, because they are low on biological sensitivity to context. In fact, there are no dandelion children in this sense. Resilience is a product of a complexly evolved foundation of prior development and presence of current supports that offset challenges (Sroufe et al., 1999; see also Egeland, chapter 4, this volume). Studies of high early risk, such as the Rochester study (Sameroff, 2000), show that when eight or nine of the potent risk variables are present, there are no resilient children. Resilience, like sensitivity to context, develops, and defining resilience in terms of inherent characteristics moves us away from developmental analysis. I clarify this further in discussing research findings.

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**How We Interpret Research**

A thoroughgoing developmental approach would not just change our descriptive language, but the way we interpret research findings. One recent example is the important research by Caspi et al. (2002) on an interaction effect between maltreatment and the MAO-A gene on conduct problems. It should, of course, be obvious with any interaction that both components must matter; yet it is striking how often this finding is interpreted to mean (and only mean) that maltreatment has an effect *only* on genetically vulnerable children. Oddly, it is often suggested that maltreatment per se does not matter; that is, it only matters if the genetic *defect* is present. It is not similarly concluded that genes do not matter. This widespread interpretation is doubly ironic. First, it is ironic because the graphed data reveal a classic crossover interaction. Thus, it is just as valid to conclude that the genetic variation only has its negative effect when children are maltreated, and even that the same genetic anomaly may have a positive effect in a nurturing environment (see also Suomi, 2002, for more compelling data on such phenomena with monkeys). The idea of a genetic "defect" should be called into question and the more developmentally friendly concept of genetic "feature" should be put forward. The frequent, overly simplified explanation is also ironic because a larger replication study found no main effect for gene but did find evidence for an interaction, *and* for a main effect for maltreatment(Foley, 2004). The implication that maltreatment only matters sometimes was misguided, especially given that it is a risk factor for diverse problems beyond conduct disorders. Maltreatment *always* matters (as do genes, even if their contribution is indirect).

Werner's classic, vitally important work (e.g., Werner & Smith, 1992) on resilience provides another example. In virtually every textbook, and in other scholarly writing, one conclusion almost always put forward from this study is that temperamental robustness enables some children to be resilient in the face of even overwhelming adversity. By this it is meant that some children have inherent, endogenous qualities that promote resilience. A close reading of the Werner work, however, reveals that significant results were for *one* (of numerous) temperamental variable at *one* age period, and this variable was the mother's description of her child as "loveable" at age 2 years. It is a stretch to interpret this variable, assessed in this way, as reflecting inherent child variation. Such a variable plausibly reflects more than endogenous child characteristics. It could be viewed as reflecting parental perceptions and attitudes as well, and is most prudently interpreted as a complex developmental outcome itself. Although this is an obvious case, showing how readily temperament as cause is accepted, the same considerations apply to other cases as well.

It is when temperament constructs are used causally (as endogenous variation that explains later functioning), rather than descriptively, that developmental thinking erodes. Many constructs currently discussed by temperament researchers are vitally important to developmental psychopathology. These certainly include "effortful control" and "emotion regulation." Such characteristics are central in current definitions of disorder (Cole et al., 1994), and they are deeply important developmental constructs. But that is the point. Effortful control and emotion regulation are capacities that develop, as do sensitivity to stimulation, biological vulnerability, and other important characteristics subsumed by temperament researchers. That such capacities may be linked to measures of brain functioning goes without saying. How could it be otherwise? Whatever the role of environment or of past development, such capacities would be reflected in brain activity. This is reductionism in the good sense that Rutter (chapter 1) describes. But such a link does not remove the need to understand development, and it does not mean that variations in such capacities are genetic or inborn.

Temperament as a term used to described individual variation has an important place in developmental study, but temperament as a causal concept obfuscates developmental understanding. When complex constructs such as emotion regulation are defined as temperamental differences, as though that explains something, we have left development behind and moved ourselves further from understanding. What we want to understand are the array of co-actions involving numerous levels across time that lead to differences in such capacities. There is at present extraordinarily little evidence, based on direct observations made early in the first year, that any temperamental dimension taken by itself has long-term predictive significance for psychopathology. In our own study, unprecedented with regard to its starting point, density of observations, comprehensiveness, and long-term follow-up, we do find evidence of interaction effects involving early emerging individual variation and experiential variables, but virtually no main effects for temperament (Sroufe et al., 2005).

There is a general tendency in the field to take "child effects" as implying causality based on inherent, endogenous, physiological variation. "Bidirectionality" of effects is a reality; children impact as well as are influenced by their parents. But demonstrations that different children elicit different reactions from parents and others, rather than giving an answer about the role of biology, raises a question about development. How do children come to do this? When the phenomenon is looked at developmentally, one discovers that child effects actually increase with age, which could be explained in a variety of ways (Sroufe et al., 2005). The child is an increasingly powerful influence with development. Also, one may predict varying environmental reactions to different children ("child effects") from knowl-

edge of varied prior experience, as well as or better than from knowledge of early temperament (Sroufe et al, 2005). Furthermore, there is a dramatically greater predictability of later disturbance from assessed child behavior after age 3 years than before age 3.

Likewise, individual variation on any characteristic in and of itself does not mean genetic variation or inborn variation or inherent variation. The fact that some children show persistent, stable conduct problems, with early onset, is not best simply taken as evidence of neurobiological disorder but, rather, as posing a developmental question. How does this come about? At the least, in addition to endogenous variation, one would wish to examine how this pattern of adaptation elicits perpetuating feedback from environmental encounters in an ongoing way. This is what Dishion and Piehler show with their work on deviant peer associations. Prospective, early beginning studies show that, in fact, the demonstrated verbal deficits of these children in general emerge subsequent to initiation of the conduct problem pathway and do not precede it; nor do newborn assessments of temperament or neurological status predict it (Aguilar et al., 2000).

Our tendency to attribute causal status to temperament is most obvious in studies beyond infancy. Such studies can show how readily we lose hold of developmental thinking even in the midst of exquisite studies of developmental process. In their wonderful chapter in this volume, Dishion and Piehler demonstrated an intriguing interaction between self-regulation capacity and deviant peer group membership; namely, that for those assessed as high on self-regulation, deviant peer membership was less predictive of increased behavior problems. This is an important finding and would immediately lead to the question of how self-regulation develops and how we can promote this capacity. But not when self-regulation is simply defined as temperament (and implied to be endogenous). The quest for understanding is ended when temperament is taken as explanation. The case for doing so is not strong. Using an instrument, a form of which has been used in infancy, and constructs that other investigators label as "temperament," does not make something a measure of temperament in anything other than a descriptive sense. It is not even clear yet whether parental descriptions in infancy of these constructs reflect endogenous variation, and there is no data to support the stability of these constructs, independently assessed, from infancy to adolescence. It is a strength that Dishion and Piehler use both parent and child report (although these did not agree with their experimental measure) and that other data sources were independent. This often is not the case. The main point is that by defining "capacity for self-regulation" as temperament (and using the term causally), the investigators moved away from a thoroughgoing developmental view.

**Research Agenda**

The research agenda within a thoroughgoing developmental approach to disturbance would be quite different from the apparent agenda today.

> Within a developmental perspective, maladaptation is viewed as evolving through the successive adaptations of persons in their environment. It is not something a person "has" or an ineluctable expression of an endogenous pathogen. It is the result of a myriad of risk and protective factors operating over time. (Sroufe, 1997, p. 251)

Therefore, within this perspective, key research questions are focused on the factors that initiate and maintain maladaptive developmental processes.

The bulk of research in the current psychiatric literature is two-group research (those with and without some disorder), and the focus is on establishing correlates of disorder once established. Mostly, the search is for neurophysiological concomitants or, more recently, specific gene loci. The clear implication is that cause is understood once these correlates are discovered. Much of this work seems to spring from a belief in the inherent and fixed nature of disorder. The fallacy of this logic is obvious. The physiological differences may be results of the disorder, reflections of complex causal features that led to the disorder, or, if genuine antecedents, themselves the results of developmental processes. Now, even genetic effects are known to be subject to experiential influence through methylation, Moreover, establishing a role for genes still leaves open questions of mechanisms and process. Thus, the current agenda is restrictive.

The broader agenda of a developmental approach is concerned with at least three foci (Sroufe, 1997). The first focus is on understanding the array of factors and combination of factors that coalesce to initiate individuals onto a pathway that is probabilistically related to later disorder. Central here is discovering early patterns of adaptation that are precursors to disorder. They are themselves not pathological but are markers of pathways to disorder when subsequent developmental challenges are faced, if surrounding structures of support and liabilities are not altered. There will be critical clues here for prevention.

The second and third issues concern understanding features of the developmental landscape that serve to maintain individuals on a pathway and guide them to one outcome or another (cf. Dishion & Piehler, chapter 6), and discovering factors and processes that help deflect individuals away from pathways toward disorder, back toward health or vice versa. It is understood that factors that initiate a pathway may not be the same as those that maintain or deflect individuals from the pathway. Discovering mechanisms and processes of change comes to the fore, and a more dynamic view of disorder results. When, for example, one sees ADHD in terms of a pathway a

child is *on,* rather than a condition a child *has,* change processes become central. The attention and control problems of these children, in fact, fluctuate (e.g., Sroufe et al., 2005), and changes in family stress and social support in part account for such changes (Carlson et al., 1995). Even with disorders that are quite stable, numerous developmental questions arise. What governs this stability? What determines when it becomes relatively stable?

The complete developmental question is not just whether some marker, characteristic, or experience is associated with later disorder, but how did this feature arise or have the impact it did and how does this process vary when other features are present. The discovery of particular genetic loci is important within this view as well, but in a very different way. Genes do not have the status of explanations, and they never will, but they may be starting points for developmental inquiry. Like other risk factors, they may serve to focus our work on developmental processes by pointing to key aspects of development that may go awry or at least by defining groups where more individuals might be expected to ultimately manifest the disorder in question. This would increase research efficiency. It is no accident that genetic work where there has been most promise concerns genes where something about developmental process is understood.

The agenda centered on understanding developmental pathways has radical effects on all aspects of our work. With all disorders, we want to know the differing array of features that are associated with onset at one point in development versus another. For example, early onset of depression and of conduct disorders is associated more with early adversity than is adolescent onset (Aguilar et al., 2000; Duggal et al., 2001).

A developmental approach also has implications for classification. Classification based on current manifest behaviors alone ignores the potential meaningfulness of different pathways to the same problem and different prognoses depending on foundations established before the period of difficulty. Moffitt's (1993) work represents an important beginning for this type of work. Dividing adolescents with conduct problems into those whose problems began very early and were persistent and those whose problems began in adolescence appears to have etiological and prognostic significance. Early onset is more heavily associated with harsh treatment than is adolescent onset, and adolescent onset cases more likely desist in early adulthood. These pathways considerations add to our understanding far beyond that granted by a symptom portrait in adolescence. In this case and more generally, developmental pathways considerations may help resolve the vexing problems of heterogeneity and of multiple disorders (comorbidity) that plague the current DSM system. We have yet to begin in earnest research in which we proceed forward in time from early patterns of adaptation and maladaptation and their later manifestations in disturbed

or healthy behavior, rather than proceeding backwards from presumed disorders in later life to antecedents.

## CONCLUSION

The chapters in this book are sophisticated and thoughtful, and in addition to underscoring the value of a multilevel approach, they point toward a comprehensive developmental perspective on psychopathology. When this perspective is taken further, and development is put fully at the forefront of our endeavors, several conclusions are reached.

First, development (of anything) is not just the product of genes and environment in interaction, but genes, environment, and past development. From the first cell division forward, there is no outcome that does not entail development.

Second, it more properly is the organism or person that interacts with environment or context, not genes. Given development, the interaction between genes and environment is only indirect. From this perspective, it actually makes more sense to talk about genes as part of the total context within which the person is acting.

Third, everything develops—including irritability, EEG asymmetry, stress reactivity, effortful control of attention, cognitive biases, resilience and psychopathology itself. None of them are givens. "There is no aspect, activity, function, or structure of the psyche that is not subject to development" (Spitz, Emde, & Metcalf, 1970, p. 417). Heterogeneity of reaction to the environment is not evidence of genetic effects, but of development (although genes, of course, are presumed to play a role). Parent reactions to children likewise are developmental outcomes.

Not only is it inappropriate to speak of gene or environmental effects as independent of one another, it also is inappropriate to speak of genes and environment as independent of development. To paraphrase the remarkable insight of developmentalist Rene Spitz, writing decades ago (e.g., Spitz et al., 1970): Where we once saw maturation and experience, genes and environment, brain and mind, body and psyche, now there is only development.

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