The Role of Context in the Development of Psychopathology: A Conceptual Framework and Some Speculative Propositions

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Despite the explosion of studies assessing relations between various contextual factors and various forms of psychological disturbance, about the only firm conclusion one can draw regarding the environment’s role in the development of psychopathology is that “bad” things have “bad” effects among some—but not all—people, some—but not all—of the time. We argue that extant research has confused two different roles of context and suggest that (1) environmental factors act as nonspecific stressors in the elicitation of psychopathology by provoking autonomic arousal, with specificity of expressed psychopathology governed by individual differences in endogenous factors; and that (2) context is specific in affecting the course of psychopathology by influencing the extent to which the behavioral, affective, or cognitive components of the pathology are repeated.

INTRODUCTION

Since the emergence of the field of developmental psychopathology several decades ago, interest in the etiology and developmental course of psychological disorder has grown significantly. Research has moved away from traditional medical models of disorder to more dynamic and multidisciplinary models that recognize the important influence of contextual factors (see Boyce et al., 1998). Despite the proliferation of studies that attempt to describe the relations between a wide array of contextual factors and various forms of psychological disturbance, however, there remains little agreement in the literature about how, to what extent, and through what mechanisms contextual experiences affect the development of psychopathology.

The search for links between specific contextual experiences and specific psychopathological outcomes has been especially disappointing. Although there has been no shortage of theories about the specific effects of certain types of experiences (e.g., loss and depression, harsh parenting and acting out, enmeshed family relationships and anorexia), research indicates that there is a surprising lack of specificity in the relation between contextual factors and psychological outcomes. Thus, for example, studies of the impact of some of the most commonly studied contextual conditions presumed to foster the development of psychopathology—marital conflict (Grych & Fincham, 1990), parental hostility (Conger et al., 1992), parental divorce or separation (Aseltine, 1996), peer rejection (Parker & Asher, 1987), neighborhood adversity (Gorman-Smith & Tolan, 1998), sexual abuse (Kendall-Tackett, Williams, & Finklehor, 1993), exposure to violence (Schwab-Stone et al., 1999), stressful life events (Brooks-Gunn, 1991), and economic stress (McLoyd, 1990)—point to a wide range of outcomes, with some children developing symptoms of internalized distress (e.g., anxiety, depression), others developing externalizing problems (e.g., aggression, conduct disorders), others developing substance abuse or dependency, others developing some combination of problems, and others showing no symptoms at all. Studies that employ cumulative and multiplicative models of risk also highlight that the likelihood of both internalizing and externalizing problems increases with the number of risk factors present, regardless of the specific contextual factors examined (e.g., Compas, Howell, Phares, Williams, & Giunta, 1989; Forehand et al., 1991; Rutter, 1978, 1979; Sameroff, Seifer, & Bartko, 1997). About the only firm conclusion one can draw about the role of context in the development of psychopathology is the obvious and unhelpful observation that “bad” things have “bad” effects among some—but not all—people, some—but not all—of the time.

In this essay, we argue that inconsistencies and weaknesses in the literature on environmental influences on psychopathology result in part from a lack of clarity in theories about contextual processes and psychopathological outcomes. In particular, we believe that extant research has failed, either theoretically or methodologically, to disentangle two very different roles of context: the role context plays in eliciting psychopathology and the role context plays in influencing the course of psychopathology (i.e., whether the disorder is maintained, diminished, or exacerbated). Although the empirical study of the development of psychopathology refers to the study of both the onset and course of disorder, no known theories have adequately outlined the role of context in each of
these processes or have included a comprehensive discussion of them. Instead, many existing theories insinuate the same factors as contributing to both the onset and the course of a disorder. In addition, due to the cross-sectional, correlational, and retrospective nature of most studies, it is not possible to discern contextual factors that cause from those that serve to maintain, diminish, or exacerbate a particular disorder. Accordingly, although it is the case that some contextual factors show relatively stronger (albeit modest) relations with one versus another type of psychopathology (e.g., exposure to violence and externalizing problems, Schwab-Stone et al., 1999), it is not clear whether this is because the contextual factor contributes to the onset of the problem (as is often claimed), or because it helps maintain or exacerbate the problem (as we suspect). The frequent failure of researchers to specify in their theoretical and empirical models whether their focus is on the elicitation versus the course of disorder has contributed to the muddied and unimpressive state of the literature.

We offer here a series of observations and hypotheses about the multiple roles of contextual factors in the development of psychopathology. We use the term “context” broadly, referring to factors in the child’s physical or social environment that may contribute to his or her development. Our argument is that context plays a very different role in eliciting than in influencing the course of psychopathology. In particular, we hypothesize that the role of context in the elicitation of psychopathology is as a general stressor whose specific outcomes are extremely difficult, if not impossible, to anticipate on the basis of contextual assessment alone. In contrast, we argue that the types of contextual factors that affect the course of psychopathology are often specific to particular disorders.

Before turning to the central argument advanced in this essay, a few introductory comments are in order. First, as the space limitations of this essay preclude an extensive review of the literature, we provide only an overview with exemplary citations for the purpose of prompting alternative research questions and directions.

Second, we use the term “psychopathology” in its broadest sense, to refer to symptoms, syndromes, and clinical diagnoses (cf., Compas, Ey, & Grant, 1993). While we recognize that there are important differences between these levels of disorder, our intent is to formulate some general propositions about context and psychopathology that apply across these definitions.

Third, given our interest in contextual influence, we focus on those forms of psychopathology that are presumed to have a substantial environmental contribution, such as those studied under the broad head-

ings of “internalizing” and “externalizing.” We do not focus on disorders that are now believed to have very substantial genetic loadings, such as bipolar disorder, schizophrenia, autism, ADHD (attention deficit/hyperactivity disorder), and Tourette’s syndrome (for a recent review of genetic evidence, see Rutter, Silberg, O’Connor, & Simonoff, 1999b).

Finally, we recognize that the distinctions we draw between context and biology are to some extent artificial. The individual’s biological makeup is not a fixed set of innate characteristics but a genetically inclined set of predispositions that are malleable in response to environmental experience. By the same token, the individual’s context is not solely an objective reality that is completely independent of the person, because the environment is perceived, selected, and modified by the individual. Nonetheless, it remains useful to distinguish between influences generally presumed to be endogenous (e.g., anatomy, physiology, neurochemistry) and those presumed to be exogenous (e.g., physical settings, significant others, sociocultural conditions). Just because context and biology influence each other does not mean that they are one and the same thing.

THE ROLE OF CONTEXT IN ELICITING PSYCHOPATHOLOGY

For much of the 20th century, the study of context and its relation to psychopathology attempted to identify specific environmental forces that elicit (i.e., cause, precipitate, trigger, predispose, or potentiate) specific types of dysfunction. The particulars of disorder-specific theories of context have changed with the times and with the vicissitudes of popular psychological theories, for example, the “double-bind” theory of family communication and schizophrenia (Bateson, Jackson, Haley, & Weakland, 1956), physical punishment and childhood aggression (Bandura & Walters, 1959; Deater-Deckard & Dodge, 1997), “enmeshed” family communication and anorexia (Blair, Freeman, & Cull, 1995; Minuchin, Rosman, & Baker, 1978), maternal “overprotectiveness” and childhood anxiety (Parker, 1983; Waldrum, Shrier, Stone, & Tobin, 1975), maternal “insensitivity” and borderline personality disorder (Masterson, 1972, 1976), and so on. The more general attempt to link particular types of contextual stress with specific types of outcomes, however, has been a longstanding, but largely unsuccessful, quest in research on the development of psychological disorder. Indeed, the few studies that have examined the specificity of risk factors for different types of psychopathology indicate little specificity of contextual factors. More interestingly, these studies have suggested
that specificity inheres in factors that reside within the individual (e.g., individual differences in self-esteem or self-consciousness, Lewinsohn, Gotlib, & Seeley, 1995, 1997). The absence of strong support for context-specific theories of psychopathology leaves us with two challenging questions: (1) How can it be that a specific contextual phenomenon can result in such different expressions of dysfunction (i.e., multifinality)? and (2) How can it be that the same expression of dysfunction can have as its antecedents such markedly different contextual causes (i.e., equifinality, see Achenbach, 1990)?

As will become clear, our approach to explaining multifinality and equifinality does not diminish the role of context in the elicitation of psychopathology. However, it seems to us, based on the accumulated evidence, that the impact of the environment on the elicitation of psychopathology must be nonspecific, and that the specificity of expressed psychopathology (both in form and severity) is likely governed not by context but by endogenous biological factors. In effect, our argument is that the same contextual factors can have decidedly different outcomes across individuals who differ from each other biologically. Well-documented gender differences in disorders like depression and conduct disorder, as well as well-established patterns of certain types of comorbidity (e.g., depression and anxiety), are consistent with this view of biological propensities and may be easily assimilated into this model.

As an illustration, consider the time-honored belief that loss precipitates depression. Loss may well precipitate depression in individuals who have a biological diathesis that predisposes them toward this disorder. We hypothesize, however, that the very same sorts of loss will precipitate aggression, and not depression, in individuals whose biology points in this direction, and it will have no significant impact on individuals who lack a biological predisposition toward disorder. By the same token, a lack of predictability and structure in the environment will contribute, as is typically assumed, to the development of conduct problems in children and adolescents who are biologically predisposed toward externalization, but the very same conditions may well provoke anxiety in individuals whose biological predisposition points toward internalization. We wish to make it clear that our view is not one of wholesale biological determinism. Indeed, we believe, consistent with a contextual perspective on psychopathology, that it remains essential to assess such factors as loss, family conflict, or environmental unpredictability in research on the development of psychopathology. We contend, however, that the assessment of such factors alone or in combination will do little to predict the specific form that the psychopathology will take in the absence of information about the individual’s biological makeup. More important, we are confident that thinking about contextual influence as nonspecific will help foster better theories of why and in what ways context matters.

Our position, of course, is a version of the more general “diathesis–stress” model of the development of psychological dysfunction (e.g., Rosenthal, 1970). But the version we propose differs from previous versions of this general model in four important ways. First, in earlier versions of the diathesis–stress model, stress was not seen as nonspecific in its outcomes (e.g., in diathesis–stress models of depression, the presumed stressor is some sort of loss). Second, earlier diathesis–stress models did not distinguish between objective and perceived stresses; however, as we explain below, a central tenet of our framework is that the impact of stress on the development of psychopathology is mediated by the individual’s level of subjective stress. Third, unlike most diathesis–stress models, the proposed model attempts to describe ways psychobiologic processes related to arousal and regulation mediate the link between contextual stress and specific expressions of psychopathology. Finally, the proposed model builds on new knowledge from the field of developmental neuroscience that permits a more detailed description of the particular types of diatheses that give rise to particular types of disorder.

**Psychopathology, self-regulation, and arousal.** If one accepts the proposition that contextual factors influence the emergence of psychopathology in a largely nonspecific fashion, and that the specificity of psychopathology is determined mainly by biological forces, one must have a theory about the general nature of the nonspecific contextual influence and the particular biological vulnerabilities that predispose individuals toward some forms of psychopathology. In our view, this question can be best addressed theoretically and empirically by building on the work of numerous writers who have focused on the constructs of self-regulation and arousal (e.g., Cole & Zahn-Waxler, 1992; Derryberry & Rothbart, 1997; Eisenberg et al., 1996; Fox, Schmidt, Calkins, Rubin, & Coplan, 1996; Schore, 1996). Within these and related perspectives, differences in arousal refer primarily to individual differences in autonomic reactivity, whereas differences in regulation refer to both individual and developmental differences in the higher-order control processes that are used to moderate arousal.

We share with numerous other writers the belief that many forms of psychopathology can be usefully cast as problems of self-regulation in the face of high arousal. In our model, psychobiologic arousal is pre-
sumed to result when, in the subjective experience of the child, one or more of the child’s basic needs—for contingent responsiveness, structure, safety, and so on—is threatened (see Boyce et al., 1998). The notion that psychobiologic arousal is mediated by the subjective appraisal of the individual leaves open a number of possibilities for individual differences in the development of psychopathology in addition to those that have their roots in individual differences in self-regulation.

First, some individuals are temperamentally more autonomically reactive than others; these individuals require less “objective” threat to experience psychobiologic arousal than individuals who are less autonomically reactive (Rothbart & Derryberry, 1981). Second, while some experiences are inherently high in their probability of provoking autonomic arousal, others are less determinative. For example, whereas the threat of severe, painful physical punishment is likely to arouse most young children all over the world, the threat of failing an exam in school is unlikely to be universally experienced as stressful.

In the face of objective events that have strong universal “threat potential,” individual differences in arousal will result mainly from individual differences in underlying biology. In the face of less universally threatening events, however, contextual factors in interaction with biological predispositions determine the level of experienced threat. Because most circumstances do not have objective threat potential outside their cultural context, both contextual factors that define what is threatening, as well as individual differences in autonomic reactivity, influence individuals’ subjective experiences of threat and, therefore, their levels of psychobiologic arousal. It is not known how these moderating processes work, but it is known that similar experiences are experienced as differentially stressful as a function of the context in which they occur (e.g., Brown & Harris, 1989). How contextual variation influences threat perception (and, consequently, psychobiologic arousal) is an important but mainly unresearched question, especially among children and adolescents.

Examining contexts in terms of their contribution to regulation and arousal helps in articulating a more general theory of the role of context in the elicitation of psychopathology. Over extended periods of time, high-risk contexts provoke high levels of psychobiologic arousal and/or interfere with arousal regulation in the child. Conversely, protective contexts are those that support mechanisms of affective, behavioral, or attentional regulation, either by contributing to the child’s ability to self-regulate or, in the case of younger children, by providing adequate external regulation for individuals whose internal regulatory processes are still developing. This proposition provides a basis for the development of general models of contextual risk and protection that can be applied across a variety of different types of settings. For example, the observation that children fare better under conditions of support and structure—whether the setting in question is the home, the classroom, or the neighborhood—is consistent with the view that protective contexts facilitate self-regulatory competence.

Psychopathology and neuropsychological vulnerability. We have hypothesized thus far that specificity in the expression of psychopathology inheres in biological differences among individuals and not in individual differences in contextual experience. It is important to clarify what is—and more important, what is not—implied by this proposition. First, we do not equate biological differences with genetic differences. Although many differences among individuals in neuropsychological vulnerability are inherited, many other biologically based differences between individuals are acquired through experience.

Second, it is unlikely that the search for neuropsychological bases of individual differences in vulnerability to specific forms of psychopathology will reveal many one-to-one correspondences between specific, isolated anatomical or neurochemical vulnerabilities and specific behavioral problems any more than the search for genetic bases of individual differences in vulnerability to various psychopathologies has resulted in the identification of many disorders whose origins can be traced to single genes (Rutter, Silberg, O’Connor, & Simonoff, 1999a). Nevertheless, recent advances in neuroscience are beginning to shed light on a variety of potential leads worthy of systematic investigation, and the development of new techniques and technologies in the brain sciences have allowed scholars to pursue many of these possibilities. Recent work on the role of the amygdala and hippocampus in emotion regulation, for example, has focused attention on these structures and the role that vulnerabilities in these complexes may play in a wide range of disorders (e.g., Derryberry & Rothbart, 1997). This line of work may well reveal particular patterns of biological vulnerability that predispose individuals toward some disorders (e.g., disorders whose central feature is heightened anxiety) but not others (e.g., disorders whose central feature is poor attentional control).

Our guiding hypothesis is that certain sorts of biological vulnerabilities, in the presence of psychobiologic arousal, lead to the development of specific types of psychopathology. These anatomical or neurochemical vulnerabilities—excesses or deficiencies of specific types of neurotransmitters or receptors, faulty myelin-
processes of normal neurotransmission (e.g., “soft-
ware”) during periods of brain plasticity and with brain development (e.g., the development of “hard-
exposure when the right frontal cortical area is espe-
depression are more likely to result from stress prefrontal cortex is very plastic, whereas problems of more likely to be caused by stress exposure when the one might predict that problems of impulsivity are more likely to have an impact during development. This raises some that, within a given developmental period, regions of the brain that are more plastic at that point in development will more likely be adversely affected are those that have some vulnerability to begin with (i.e., where there are relatively weaker or less well-developed synaptic patterns). We suggest this based on some of the work on the impact of early stress on the hippocampus, amygdaloid nuclei, and corpus callosum (reviewed by Teicher et al., in press) as well as speculation and evidence regarding the impact of environmental experience on prefrontal cortical functioning (Davidson, 1994; Dawson, Hessl, & Frey, 1994; Dawson, Panagiotides, Klinger, & Spieker, 1997; DeBellis & Putnam, 1994; Schore, 1997; Teicher et al., 1997) and on the role of stress-induced increases in adrenal hor-
mones in the suppression of neurogenesis in the adult hippocampus (e.g., Gould, Cameron, Daniels, Wollo-

A corollary of this view is that contextual stress will more likely have an impact during development-
al periods of high neuropsychological plasticity and that, within a given developmental period, regions of the brain that are more plastic at that point in development will be more likely affected. This raises some potentially interesting hypotheses about the timing and duration of contextual stress as a variable that affects the specificity of the psychopathology—that is, one might predict that problems of impulsivity are more likely to be caused by stress exposure when the prefrontal cortex is very plastic, whereas problems of depression are more likely to result from stress exposure when the right frontal cortical area is espe-
1 We condition the last two statements with the word “likely” in appreciation of the complexity of links among multiple brain systems. Even with appropriate levels of contextual stress, biological vulnerabilities will not definitively result in predetermined effects but in the greater likelihood of these effects.
or attention, not whether the pattern is reinforced or rewarded. Although it is axiomatic that rewarded patterns of behavior are more likely to be repeated (and thereby strengthened), our contention is that repetition— even in the absence of reinforcement— strengthens affective, behavioral, and cognitive patterns, too. This suggests that the main role of context in the maintenance, exacerbation, and diminution of psychopathology can be seen (and presumably measured) as a function of the frequency with which the context permits—not only encourages or discourages—the expression of the specific psychopathology in question to be repeated. A necessary corollary is that some contextual stressors may be related to the maintenance of more than one type of psychopathology, so long as the specific stressor allows for the repetition of behavior relevant to each of the disorders. For example, expressed emotion has been found to be related to the relapse of mood disorders, eating disorders, and schizophrenia (Bulfaiff & Hooley, 1998). As a construct involving both hostility and psychological control, it is reasonable to assume that family environments high in expressed emotion allow for the repetition of depressive-type cognitions (i.e., to the extent that exposure to hostility increases the likelihood that one will feel bad about oneself and one’s situation) and simultaneously serve to maintain a lack of self-control (as in eating disorders).

Contextual opportunities therefore can be studied in terms of their likelihood of increasing or decreasing the repetition of certain patterns of behavior. Consider the example of aggression in children. It has been consistently shown that a lack of structure in the environment is associated with the development of antisocial behavior. Whereas a lack of structure does not necessarily cause antisocial behavior, a lack of sufficient structure is more likely, for a child with an already-existing conduct disorder, to permit the expression of the disorder and to crystallize the disorder in a way that makes it less malleable over time than is likely within a more structured environment. Thus, individuals who are already inclined toward aggressive behavior are more likely to behave aggressively in contexts that are less controlled and more likely to be forced to inhibit their aggression in contexts that are more structured.

Because contexts are not randomly assigned to individuals (or vice versa), the odds are high that individuals will end up having experiences that maintain or even strengthen, rather than extinguish or weaken, existing patterns of behavior (e.g., Scarr, 1992; Scarr & McCartney, 1983). Thus, for example, depressed individuals have synaptic weightings that predispose them toward certain behaviors, emotions, or cognitions, which in turn predispose them toward contexts that allow for and encourage more depression (Hammen, 1991), and the experiences that these depressed individuals have are more likely to strengthen than to weaken these weightings. Aggressive individuals have synaptic weightings that predispose them toward behaving aggressively, and these predispositions, when realized, further strengthen the synaptic weightings. Consistent with this reasoning, several investigators (e.g., Cairns, Cairns, Neckerman, Gest, & Gariepy, 1988; Dishion, Patterson, Stoolmiller, & Skinner, 1991; Kendal, 1978) have shown that conduct-disordered adolescents actively affiliate with conduct-disordered peers, whereas other investigators (Hogue & Steinberg, 1995) have shown that individuals who report internalizing problems are more likely to actively affiliate with other youngsters with similar problems. Over time, association with conduct-disordered peers influences the development of further antisocial behavior, whereas association with depressed peers influences the further development of depression.

The notion that disorder-specific contextual factors operate not in the genesis of psychopathology but in the exacerbation of the specific disorder through strengthening already established biological pathways helps to explain four related and widely accepted axioms about development (see also Caspi, 1998): (1) that people become more like themselves over time; (2) that, over time, the psychologically rich get richer and the psychologically poor get poorer; (3) that stress serves to strengthen, rather than transform, previously existing traits; and (4) that repetitive practice is the single best predictor of performance improvement. One important conclusion from this line of reasoning is this: in the absence of environmental intervention, individuals with a specific type of psychopathology will, over time, be more likely to continue along this psychopathological trajectory, because the psychopathology influences the sorts of experiences the individual has, and these experiences tend to strengthen existing behavior (Hammen, 1991; Rutter, 1997).

A comparable contextual process can be suggested to explain how forces in the environment can weaken a preexisting pattern of psychological disorder. The primary mechanisms through which nonbiological interventions operate are those that weaken synaptic weightings through nonuse of “maladaptive” circuits or through the strengthening of “competitive” circuits. This can be accomplished by directly changing the individual’s affect, behavior, or attention or by modifying the individual’s context (e.g., family, classroom, neighborhood) to reduce opportunities for repetition of the maladaptive behavior. For example, evidence from intervention studies suggests that af-
fecting the family and peer environments is critical in preventing the escalation of antisocial behavior during adolescence (Borduin, 1999; Dishion & Andrews, 1995). Other studies have shown that when contexts change for the better, behavioral and emotional problems diminish (Esser, Schmidt, & Woerner, 1990; Hetherington, Cox, & Cox, 1982).

Understanding contexts as providing opportunities for strengthening or weakening synaptic weightings through repetition may also help account for certain observations about the relative effectiveness of various (especially behavior-oriented) types of clinical intervention. This is why, for instance, there appears to be a dose–response relationship in cognitive/behavioral therapy (although the relationship is not entirely linear, more therapy usually means more repetition for the competing circuit); why intensive therapy is generally more effective (fewer opportunities to engage in maladaptive behavior between sessions); and why psychological checkups and booster shots are likely to be beneficial (opportunities to make sure that the desired pattern is repeated). We think this also accounts for the inconsistent findings concerning the benefits of early versus late intervention, because, while certain brain regions may be more plastic earlier than later, not all follow this pattern (Nelson, 1999). If, for example, brain plasticity in certain regions is greater around the time of puberty than before it, some interventions may be more effective during adolescence than prepubertally. More research is needed to better map the timetable of effective intervention.

CONCLUSION

Much of our failure to understand the role of context in the development of emotional and behavioral disorder stems from our tendency to confound two very different roles that context plays in psychopathology: as an eliciting factor and as an influence on the course of disorder. Theories about the role of context in the development of psychopathology do not always distinguish between these roles, and often overlook important differences between them. The most serious error in this regard is the assumption that contextual influences that affect the course of a disorder must therefore be the same as those that contribute to the disorder’s onset.

We readily acknowledge that the argument we have advanced here is speculative. Nonetheless, we believe that the argument is consistent with the extant literatures in both developmental psychopathology and neuroscience, and that the speculations advanced in this essay have potentially important implications for future research, among them the need to design prospective studies that differentiate between influences on the onset and influences on the course of disorder and the need to assess the contributions of both biology and context within the same research design.

In the meantime, what are contextually oriented researchers interested in developmental psychopathology to do? First, we should abandon the search for disorder-specific contextual factors that elicit psychopathology and focus instead on understanding how individuals perceive and experience stress. Second, we should devote more time to research aimed at identifying environmental mechanisms that attenuate or exacerbate existing conditions (e.g., Dishion, Capaldi, & Yoerger, 1999; Scaramella, Conger, & Simons, 1999). Finally, and most important, we must do more than pay lip service to the oft-pronounced but seldom followed directive to try to understand how biology and context interact (e.g., Eisenberg et al., 1999). If contextualists do not recognize that the future is in such collaborative efforts, our promise to contribute to the study of developmental psychopathology in the next decade will go unfulfilled.

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