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Resilience and Developmental Psychopathology

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Humans vary to a great extent in their ability to adapt to adverse experiences. Resilience reflects the positive end of this spectrum of adaptation and maladaptation in response to risk exposure. Some individuals, despite being faced with the most pernicious of adversities, manage to avoid collapse and to maintain healthy adjustment. For decades, researchers and clinicians have searched for the correlates and causes of resilience, and several comprehensive reviews of these efforts are available (eg, [1–5]). This article provides an overview of resilience research and selectively summarizes recent advances in the field, with a particular focus on a developmental psychopathology perspective. This article highlights some of the challenges to resilience research and uses the example of maltreatment to illustrate some of these issues.

Developmental psychopathology: a brief overview

Developmental psychopathology has been described as an “integrative framework” for a coherent, interdisciplinary science of normal and abnormal development [6–8]. It is not a theory; rather, it is an overarching perspective that encompasses multiple theories and encourages investigations at multiple levels of analysis ranging from the molecular to the cultural [9,10]. What has distinguished the developmental psychopathology viewpoint from the traditional perspective of psychiatry is its conceptualization of mental disorder not as an inherent trait that resides in an individual, but as something that emerges from the dynamic interplay between intraindividual and extraindividual contexts [11,12]. Moreover, issues of time and timing are essential in understanding the confluence of an individual’s past experiences with present circumstances in predicting future adaptation.

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Patterns of continuity and discontinuity over development are of particular interest; change is always thought to be possible with the caveat that the likelihood and degree of change is constrained by prior history and the current context [11].

Resilience from a developmental psychopathology perspective

Definition

Over the past several decades, the resilience construct has undergone a major shift in its conceptualization. Initially, children of parents who have schizophrenia or children growing up in poverty who avoided a psychopathologic outcome and developed a healthy pattern of adaptation were deemed to be “invulnerable” or “invincible” [13,14]; resilience at one time was viewed as a special characteristic of the child. Contemporary developmental psychopathologists now define resilience as a dynamic process of positive adaptation in the context of significant adversity [1,2,15–18]. Instead, this perspective implies several things that resilience is not: It is not static, it is not a trait, and it is not a construct that can be directly measured. Resilience is a “superordinate” construct that is indirectly inferred from two component constructs subsumed under its definition: risk exposure and “good” adaptation [3,17]. Evidence of positive adaptation implies that important protective processes are operating to steer an individual away from the maladaptive developmental trajectory that typically ensues after exposure to adversity. Scientists and clinicians have engaged in a search for these protective processes that hold promise for elucidating theory and for informing intervention strategies.

Risk, protection, and positive adaptation

Risk is defined in terms of predicted statistical probabilities in that being exposed to a risk factor elevates an individual’s likelihood of a maladaptive outcome. Risk factors that increase liability for mental disorder or maladaptive development include all manner of psychosocial hardship, including poverty, loss of a family member, maltreatment, or other traumatic events. Risk factors also include nonpsychosocial factors, such as prenatal and perinatal insults, infectious agents, or other biological pathogens, that can compromise healthy development. Many individuals exposed to these risk factors seem to manage well and escape the corresponding deleterious consequences. However, among children at risk, there is heterogeneity in the extent to which adversity has been experienced. For example, although parental psychopathology is a distal indicator of increased statistical risk for maladjustment in offspring, there is no evidence that it necessarily coincides with risk-increasing events and circumstances that a child actually experiences [19]. Children of parents who have a mental disorder are most

at risk for psychopathology if they have also experienced inadequate caregiving [20]. Thus, the presence of a parental psychiatric diagnosis alone to designate high risk could identify children who are at low risk and hence could not be considered “resilient” even if they are well adjusted [21,22]. Therefore, for a more thorough and precise understanding of resilience phenomena, risk exposure must be carefully conceptualized and measured.

A developmental psychopathology framework offers several suggestions for improving upon the notion of risk in relation to resilience. First, one basic tenet of developmental psychopathology is that developmental “turning points” can provide especially fertile opportunities for change toward greater vulnerability or greater strength [7]. Therefore, investigating the developmental timing of exposure can yield important insights into “sensitive periods” during which the effect of a risk factor is especially strong [23]. A growing body of research points to development early in the life span, including the prenatal period, as a time during which adversity can have a lasting impact on the life course. For instance, longitudinal studies have shown that high levels of stress, anxiety, and depression in mothers during pregnancy [24] and in infancy [25] predict behavioral and emotional problems in children many years later. Research by Essex and colleagues [26] suggests that elevations in cortisol may help account for this link. In a longitudinal design, they found that preschoolers who had been exposed to high levels of maternal stress and depression only in infancy or only concurrently did not show elevated cortisol levels. However, children who had been exposed to high maternal stress in infancy responded to maternal stress in the preschool period with significantly higher cortisol levels compared with control subjects, and high cortisol in preschoolers was associated with high levels of mental health problems in first grade. Assessing a child’s response to stress exposure at only one point in time would have obscured the observation regarding the sensitizing effect of early and lasting exposure to later stress. By clarifying the conditions under which stress exposure contributes to vulnerability, an examination of the developmental timing of risk exposure can provide a more thorough understanding of adaptive processes.

Second, there is increasingly a need to understand how multiple risk factors during multiple developmental periods work together to bring about maladaptation or provide opportunities for resilience processes to unfold. Recent work by Essex and colleagues [27] indicates that risk factors may operate differently over development in families from high versus low socioeconomic backgrounds. Children at risk for internalizing and externalizing psychopathology from high socioeconomic backgrounds may be identified as early as infancy on the basis of a family history of psychopathology, which generates high levels of maternal distress and child dysregulation in early childhood. Children at risk from low and middle socioeconomic backgrounds may not be easily identified until the preschool period when high levels of maternal and child distress begin to be more predictive of later symptomatology. The way in which risk factors operate can depend upon

moderators such as family socioeconomic status (SES) and the developmental period during which they occur.

Third, any single risk factor is likely to explain only a small proportion of the variance in outcome, and many studies have documented the multiplicative effects of cumulative risks on child adjustment. For instance, Rutter and colleagues' [28] classic Isle of Wight Study examined six significant predictors of child psychopathology: severe marital discord, low socioeconomic status, large family size, parental criminality, maternal mental illness, and out-of-home placement. They found that children who had only one of these risks were at no greater disadvantage than children who had none. By contrast, the presence of two risk factors quadrupled negative effects on adjustment, and four or more risk factors increased maladjustment 10 fold. The demonstrated association between cumulative risk and adjustment has direct implications for how resilient individuals are identified. Evidence of positive adaptation to any one of these risk indicators is likely to be common, whereas evidence of positive adaptation in response to several risk indicators is likely to identify a rarer, and arguably more valid, exemplar of resilience.

Protective factors are variables that mitigate the impact of risk factors on developmental outcomes. Variables that moderate the link between a risk factor and an outcome can provide clues regarding protective and vulnerability processes. Protective moderating effects can operate in several ways that are illustrated by the model of neural plasticity, the ability of neural circuitry to change [29,30]. First, resilience may come about from an innate resistance to brain injury in response to challenges presented by adverse events. In other words, the presence of the protective attribute lends a robust capacity to maintain balance or homeostasis. Second, the degree of neural plasticity can determine the degree of recovery after injury. Third, neural plasticity can compensate for damage caused by adversity. Although the original loss of function may not be restored, other related brain mechanisms may develop and grow strong enough to compensate for the loss. These models of protective processes are not limited to thinking about neurobiologic mechanisms; they may also be applied toward thinking about the impact of psychosocial protective factors. For instance, in a neurobiologic sense, plasticity may result from appropriate and adaptive responses of the autonomic nervous system, the hypothalamic-pituitary-adrenal axis, and cardiovascular, metabolic, and immune systems [31]. In a psychosocial sense, the personality trait of ego-resiliency may operate in a similar way by generating flexible and suitable psychological responses that resist, repair, or compensate for the consequences of adversity [32].

The conceptualization or operational definitions of resilience have not required that risk or protective factors be causally related to a particular outcome. The more we can document true causal effects that are environmentally (rather than genetically) mediated, the greater promise results regarding resilience hold for informing theory and intervention. For obvious ethical reasons,

most studies investigating the causes of resilient development cannot use experimental design involving random assignment to risk versus no-risk conditions. In lieu of this, researchers using observational studies can strengthen their conclusions regarding *environmental* risk and protection by increasing their awareness of possible genetic confounds. Research designs based on “natural experiments” are coming into focus that allow for possible causal inferences using correlational data [33]. Twin and adoption studies, longitudinal designs, migration studies, and intervention studies are a few of the methods that can be used to test for environmental causation. Extreme caution is necessary before conclusions regarding causality can be made in the absence of true experimental design, but if a number of specific conditions are met, preliminary causal associations can be suggested [33].

Broadly speaking, “good” adaptation has been described as success at achieving stage-salient developmental tasks or the absence of mental disorder or both [2,5]. Many risk factors can have an impact on a number of different domains of functioning. For instance, child maltreatment increases the risk for conduct problems and depression [34,35]. Therefore, the domains of psychological outcomes assessed must be carefully selected to correspond with the risk factor under study. Otherwise, resilient outcomes can be misidentified. Luthar and colleagues [2] noted that when the risk is especially deleterious for a particular outcome, it should be given priority [2,36]. They also suggest that consideration must be given to whether multiple or single outcomes should be measured. Investigators have begun to question the utility of global, combined measures of outcomes in studying resilient processes [5,37]. Luthar and colleagues [2] proposed that if the outcomes examined represent largely discrete constructs, it is most meaningful to examine them separately. Just as comorbidity of psychiatric conditions signals greater severity of illness [38], the co-occurrence of manifest resilience across a number of domains would logically indicate a greater degree of health and adaptational success.

The role of gene–environment correlations and interactions

Decades of family history and behavioral genetic research have revealed that most behavioral phenotypes, including psychiatric disorders, temperament, and measures of the family environment, show substantial genetic influence [39]. Heritable influences are likely to pervade risk and protective factors and to play some part in the development of resilience [40–42]. One implication of the heritability of psychological measures is that correlations among variables measured in biologically related family members cannot necessarily be interpreted to reflect environmentally mediated influences [43]. They may represent a gene–environment correlation, which refers to genetically mediated influences on environmental exposure. Thus, in thinking about associations between a risk factor such as maternal depression and an outcome such as child antisocial behavior, true environmental

effects should be tested and documented [44]. The same point can be made regarding associations between putative protective factors and resilient outcomes.

Evidence is building regarding genetic processes in promoting resilience. In a study of 5-year-old twins, behavioral and cognitive resilience to SES deprivation showed significant genetic and nongenetic influences [42]. Children's behavioral resilience was correlated with maternal warmth, and approximately half this covariation was explained by genetic effects, indicating that the possible protective function of maternal warmth on children's behavioral development could not be explained entirely by a gene-environment correlation. These findings help support the notion that parenting quality may perhaps be of top importance in protecting children's development against the effects of adversity [3].

In recent years, a growing body of evidence has demonstrated that specific genes are involved in predicting resilient functioning by modifying the effect of environmental risks on behavioral outcomes. These gene-environment interactions ($G \times E$) should not be surprising given that a fundamental purpose of polymorphic genes, or genes that vary across individuals, is to facilitate adaptation to a variety of environmental contexts [39,45]. Caspi and colleagues [46–48] have published three notable $G \times E$ findings using data from the Dunedin Longitudinal Study. First, a polymorphism in the gene encoding the monoamine oxidase A (MAOA) enzyme moderated the impact of childhood maltreatment on the development of antisocial behavior [48]. Second, a polymorphism in the gene encoding catechol-O-methyltransferase (COMT) moderated the association between early cannabis use and psychosis [47]. Third, a functional polymorphism in the serotonin transporter (*5-HTTLPR*) gene significantly moderated the association between stressful life events and depression [46]. In the context of stress, individuals carrying two copies of the “long” *5-HTTLPR* allele were at relatively lower risk of depressive symptoms, a major depressive episode, and suicidality compared with counterparts carrying one or two copies of the “short” allele. Several independent investigations have replicated this finding [33,49–51], including a study by Kaufman and colleagues [33] in which maltreated children were at decreased risk of depression if they carried two copies of the long allele. A notable finding in Kaufman and colleagues' study is that in maltreated carriers of the “vulnerable” short allele, perceived social support from a caregiver protected these children from developing depressive symptoms. This study is the first to demonstrate that social support can buffer the emotional health of maltreated children with a genetic vulnerability toward depression.

Multiple levels of analysis in risk and protection

Because risk and protective processes traverse multiple levels of analysis, resilience research stands to benefit from incorporating multiple disciplines

and levels of analysis including molecular, neurologic, behavioral, and cultural systems [9,10,52]. To move resilience research toward the next level of sophistication, variables from multiple domains need to be incorporated into simultaneous investigations to explicate gene-to-behavior pathways [53].

Genes involved in influencing behavior must operate via brain-based mechanisms. Pioneering work in the emerging field of developmental imaging genetics [54,55] has begun revealing structural and functional brain differences associated with variation in genotype. For instance, in a sample of healthy, adult volunteers, Meyer-Lindenberg and colleagues [54] reported that individuals carrying different variants of the *MAOA* promoter polymorphism differ significantly in structural and functional brain measures. Compared with carriers of the high-activity *MAOA* variant, carriers of the low-activity variant showed patterns of brain activation that are associated with greater impulsivity, poor cognitive control, and more sensitivity toward angry cues, which suggest intermediate processes by which genotype may be linked with a vulnerability toward aggressive behavior. Genetically influenced differences in brain morphology and function are promising avenues for identifying determinants of resilient (and vulnerable) patterns of responses to adversity [52,56].

Temperament has long been thought to play a major role in shaping resilient development. For instance, Werner and Smith [14,57] found that children who as infants were smiley, cuddly, and sociable were most likely to overcome the adversities associated with rural poverty on the Hawaiian island of Kauai. Other studies have identified an easy-going temperament in childhood to be associated with resilient outcomes in youths growing up in conditions of urban deprivation [58]. In a twin study of resilience in the context of socioeconomic adversity, an outgoing and sociable tendency to approach others was correlated with better-than-expected IQ scores [42]. Moreover, common genetic influences accounted for 71% of the covariation between an outgoing temperament and cognitive resilience in these children. Although the direction of effects cannot be determined in this cross-sectional study, findings suggest that children's genetically influenced temperamental traits may elicit the kind of attention and stimulation from others that help protect against the deleterious impact of poverty.

Numerous studies have highlighted the critical influence of parenting quality on children's resilient adaptation [3,59–61]. For instance, a secure attachment with a consistent caregiver is one of the most robust predictors of resilient functioning via a number of possible mechanisms. Recent research is shedding light on one possible mechanism by which a secure attachment relationship may protect against adversity and stress. In a study by Nachmias and colleagues [62], children who had a behaviorally inhibited temperamental style did not show significant elevations in cortisol if they were securely attached to their caregiver. However, children who were insecurely attached to a caregiver showed cortisol elevations even after

postinoculation. Protective factors at the family level can come in the form of an absence, rather than a presence, of a parental figure. For instance, Jaffee and colleagues [63] showed that children who had a familial liability toward developing antisocial behavior were less antisocial if their antisocial fathers were absent and spent less time providing care for the children.

Few studies have examined the processes by which cultural influences might affect resilient development. Studies that have been conducted on samples of Latino children have concluded that, by and large, patterns of resilience and vulnerability do not differ substantially from those of European American children [32,64]. Certain cultural influences, such as ethnic pride and biculturalism, are potential sources of protection, whereas other aspects of culture, such as clashes between two cultural value systems, are potential sources of vulnerability [65]. As such, resilience has been identified as a fruitful area in which to incorporate the study of culture and developmental psychopathology [66].

Illustrations from resilience in the context of maltreatment

Childhood maltreatment is a well characterized risk factor for the development of aggressive behavior and is involved in perpetuating the cycle of violence across generations [67]. The association between maltreatment experience and aggression might reflect a gene–environment correlation in two ways. First, the association could be explained by a passive gene–environment correlation such that the genes influencing a parent’s propensity to maltreat a child could be the same genes transmitted to the child that then increase his or her likelihood of engaging in aggressive behavior. Second, the association could be explained by an evocative gene–environment correlation such that a child’s genetically influenced traits, including aggressive tendencies, might elicit harsh and abusive treatment from a caregiver. Evidence suggests that this is not the case and that the association between maltreatment experience and aggression is one that is environmentally mediated and perhaps causal [35].

Although a substantial proportion of maltreated children become maltreating parents, this association is not direct nor inevitable [68]. Multiple circumstances and mediating mechanisms may explain how some maltreated individuals show resilience and do not abuse their own offspring or otherwise demonstrate adequate caregiving. First, an early study of women who had been reared in institutions found that the women who had managed to find supportive, stable spouses demonstrated good quality caregiving that was comparable to the caregiving provided by the noninstitutionalized comparison group [69,70]. The women’s ability to exercise “planning” was found to be the most important variable in explaining how they well they fared in their marriage and parenting roles, despite having been raised in an institution. Planning suggested a good ability for

foresight and control and kept these women from making hasty and poor decisions about marriage.

At a different level of analysis, recent research has demonstrated that another reason why maltreated children go on to have relatively better developmental outcomes may have to do with a specific genetic polymorphism, as mentioned above. Caspi and colleagues [47] reported that a functional variant in the gene encoding MAOA significantly moderated the impact of childhood maltreatment on males' risk for antisocial behavior in adolescence and adulthood. The primary function of MAOA is to selectively metabolize serotonin, dopamine, and norepinephrine [71], important neurotransmitters involved in the regulation of mood and behavior. The degree of activity of the *MAOA* gene varies across individuals and can be classified as being low or high in activity level. Caspi and colleagues' study [47] revealed that maltreated individuals carrying the high-activity version of the *MAOA* gene were significantly less likely than maltreated individuals carrying the low-activity version of the gene to engage in antisocial behavior, violence, and criminality. Among individuals not exposed to childhood maltreatment, the *MAOA* gene showed no significant association with antisocial outcomes, suggesting that the gene itself in the absence of the environmental risk has no impact on resilience or vulnerability. In the context of a childhood maltreatment history, the protective (and vulnerability) effect of the gene became apparent. To date, three positive replications [72–74] and three failures to replicate [75–77] this $G \times E$ interaction have been published, and a meta-analysis of these studies provided preliminary confirmation of the moderating effect of the *MAOA* gene on the association between childhood maltreatment and antisocial behavior [78].

With regard to evidence at the level of neurotransmitters, a recent study of rhesus macaques found that levels of serotonin in cerebral spinal fluid may help explain which maltreated rhesus macaques grow up to abuse their own offspring and which do not [79]. Previous work using a powerful cross-fostering design demonstrated that approximately half of the rhesus macaque females who had been physically abused by their mother (or adoptive mother) in infancy exhibited abusive behavior with their own offspring [80]. Monkeys who were abused in infancy and later became abusive mothers themselves had lower levels of cerebral spinal fluid serotonin than did abused monkeys who did not become abusive with their own offspring. These findings suggest a biologically mediated protective process, such that adequate levels of functioning in the serotonin system may play a significant role in escaping the intergenerational cycle of violence.

Summary

For decades, the study of resilience has been an important area of research within the developmental psychopathology framework. The study of resilience has provided special opportunities and challenges for

understanding the interplay of normal and abnormal development and the processes by which individuals cope successfully with life's "slings and arrows." Despite tremendous progress, much work is left to be done. Future decades will see further advances in understanding resilience from multiple levels of analysis, especially by incorporating genetics, neurobiology, and the relatively understudied area of cultural contributions to resilience.

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