Socioeconomic Risk for Psychopathology: The Search for Causal Mechanisms

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The influence of environmental factors on child psychiatric illness is terribly important. Garmezy and Masten¹ noted that, "...for at least four centuries stressful life experiences have been viewed as direct etiological agents of disorders" (p. 191). As acute (e.g., traumatic events) and chronic (e.g., low socioeconomic status [SES]) stressors have been established as important predictors of child and adolescent psychopathology, there has been increased effort to identify the pathways through which risk is heightened in some children exposed to these stressors but not in others. For the past decade, there has been particular emphasis on the study of genetic factors and how they interact with environment as a possible explanation for differential outcomes. It is postulated that genes and environment interact (G × E) to either promote or protect against psychopathology.² The search for such genetic mechanisms can be intoxicating, clarifying, and at times overblown. For example, Risch and colleagues³ report in their recent meta-analyses of 14 studies that there is no evidence for the interaction between serotonin transporter gene alleles and adversity as a risk factor for depression. Their analyses revealed that adversity alone explains the greatest proportion (40%) of depression risk. It may be that chronic adversity, negative outcomes, and genetic factors are correlated, and almost certainly, G × E research will yield important findings in the near future. However, in the interim, the results of the study by Risch et al.³ highlight the importance of studying the relative contributions of chronic and acute environmental adversity when attempting to understand the etiology of child psychiatric illness.

Among the more widely studied chronic environmental stressors in child and adolescent psychiatry are family so-

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cioeconomic factors (e.g., income, education). Research has shown that youths from low-SES families are at risk for a variety of problems, including conduct problems and substance use, relative to their peers with higher SES.4 Furthermore, patients from low-SES families often lack many treatment-facilitating advantages, such as the means to pay for medication and therapy, or schools and teachers with the resources to attend to children's mental health needs. Most mental health providers have probably wished for the power to change the neighborhood safety, family income, or school quality of their patients at some point. Acknowledging the risk associated with low SES allows for the targeting of specific populations of youths for prevention and intervention programs. However, the mechanisms and processes by which low SES influences psychological functioning must also be identified to understand how and why certain low-SES youths develop internalizing and externalizing problems, whereas others do not. As practitioners, we have little to no influence over the neighborhoods in which our patients live, or their parents' incomes, but we can facilitate change in factors that may determine how youths function within low SES contexts, such as a child's ability to evaluate safety and danger in his or her environment, or his/her coping skills.⁵

In this issue of the *Journal*, Amone-P'Olak and colleagues⁶ examine the role of life stressors as one pathway through which low SES confers risk for internalizing and externalizing problems in 2,149 adolescents ages 12 to 15 years. Interestingly, although both "person-related" (e.g., "dismissal from school," "romantic breakup") and "environment-related" (e.g., "parental unemployment," "chronic illness/handicap of family member") life stressors were related to internalizing and externalizing problems, only the number of environmentrelated life stressors mediated the association between low SES and psychopathology. This effect was particularly strong for internalizing disorders. The authors note that personrelated stressors may result primarily from factors other than SES, such as personality traits or psychiatric symptoms, whereas exposure to environment-related stressors is more likely to stem from the familial context, including socioeconomic position.

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The ramifications of these findings for the treatment of psychopathology among low-SES youths are striking. First, although treatment of childhood and adolescent anxiety and depression is typically conducted with the referred child, the results of the study by Amone-P'Olak et al. suggest that, for those patients from low SES backgrounds, intervening with the child's environment may be fruitful as well. For example, assessing and treating parental psychopathology, recommending regular physician's visits for all family members to prevent and treat serious and chronic illness, or referring clashing parents for couples therapy could help to alleviate the child's internalizing symptoms.

Second, the use of what have been termed *secondary control coping* skills (acceptance, distraction, cognitive restructuring, and emotion regulation) has been shown to mitigate the effects of environment-related stressors on psychiatric symptoms. These skills can be taught to low-SES children and adolescents as ways to manage their emotions, thoughts, and behaviors when facing stressors beyond their control, which Amone-P'Olak and colleagues suggest play a key role in the development of internalizing and externalizing problems.

These implications are preliminary, however, because this study used a cross-sectional design and retrospective reporting of life stressors, raising questions as to whether preexisting parental and/or adolescent psychopathology could have confounded the reporting of particular life events. As the authors discuss, there may also be alternative, more influential mechanisms by which low SES relates to psychopathology and wellness. For example, social support has been identified as a stronger contributor to happiness in low-SES compared with high-SES families.9 Furthermore, there may be specific life stressors that are more or less toxic than others. The death of a parent, for instance, could be more detrimental than the death of a pet. Similarly, the intensity and chronicity of life stressors needs to be explored in future studies. Chronic stressors such as recurrent parental psychopathology may play a different role in the link between SES and child mental health relative to time-limited events like divorce.

Finally, although the authors appropriately determined their use of number rather than severity of life stressors to be a strength of the study, as this method reduces subjectivity, this may also be a point to be expanded on in future work. Previous research has suggested that individuals' perception of life events influences whether and how these events relate to psychopathology. Therefore, a child's appraisal of the severity of his/her parent's illness, for example, could elevate or mitigate that child's risk for psychiatric disorders.

We eagerly anticipate additional lines of prospective, experimental, and epidemiological research that continue to examine the ways in which socioeconomic context confers psychiatric risk for certain children but not others. Such investigations will help to provide windows through which mental health providers can more effectively alleviate psychiatric symptoms and enhance wellness among children and adolescents living in stressful environments.

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