JAMA Network Open

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Childhood Adversity as a Plasticity Factor That Modifies the Association Between Subsequent Life Experience and Psychopathology

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Exposure to childhood adversity—whether poverty, maltreatment, or another life stressor—is one of the most potent social determinants of poor mental and physical health across the life span. From decades of research, it has been well established that childhood adversities at least double the risk of mental disorders that begin not only during childhood or adolescence, but even during adulthood. The consequences of childhood adversity on mental health are also transdiagnostic, spanning psychiatric diagnostic boundaries. Given these associations—and the fact that exposure to adversity and trauma is near ubiquitous worldwide—there is an urgent need to identify strategies to mitigate the negative consequences of adversity, in the service of ultimately preventing the onset of mental disorders and reducing their overall burden.

However, the field's approach to tackle this challenge has been generally limited to date. Most childhood adversity researchers too often adopt a binary approach, defining individuals as either vulnerable or resilient to psychopathology after adversity exposure. Too few studies have examined both vulnerabilities and positive adaptations after exposure to childhood adversity, particularly in large population-based samples. Even fewer have considered how the experience of childhood adversity can shape sensitivity to subsequent life experiences not only for worse, but also, when paired with environmental enrichments, for the better. As a field, we have not yet considered the full spectrum of adversity-associated plasticity, or its possible implications for prevention and intervention.

Albott et al[1] used data from one of the best US-based epidemiologic studies of adults to test a differential susceptibility model, positing that a history of childhood adversity could make individuals respond more unfavorably to harmful environments, but also more favorably to protective ones. By examining changes in stress and psychopathology symptoms during a 3-year period, the authors were able to demonstrate a stronger association between high levels of recent stress and psychopathology symptoms among adults exposed to childhood adversity. More uniquely, however, they also found evidence in support of the differential susceptibility model, whereby reductions in the number of recent stressors were associated with lower levels of psychopathology symptoms, especially among adults with a history of exposure to childhood adversity. This study appears to be the first to demonstrate such findings in a population-based sample.

These results reminded me of work by Martin H. Teicher, MD, PhD, on the mental health consequences of exposure to child maltreatment, which is one the most common types of childhood adversity. In 2014, Teicher and Samson[2] argued that individuals with a history of childhood maltreatment represent a clinically and biologically distinct subtype among individuals who experience psychopathology, showing that a history of child maltreatment (vs no history) is often linked to an earlier onset of disorder, as well as a poorer course and psychiatric treatment response. The work by Albott and colleagues[1] nicely extends these findings, emphasizing the distinctness of adversity as a unique phenotype, but in this case identifying ways in which differential susceptibility or plasticity can be advantageous.

Albott et al[1] acknowledge that the magnitude of difference between the groups exposed and unexposed to childhood adversity was much greater at the stress addition end of the spectrum—

meaning among individuals who recently increased their stress exposure—than the stress reduction end—meaning among individuals who experienced fewer stressors over time. However, this interpretation views these findings from a high-risk approach to disease prevention, rather than a population-based approach.[3] As articulated by Rose,[3] a high-risk approach to disease prevention only seeks to target individuals at the extreme end of a given risk factor, such as those with the most exposure to childhood adversity. In contrast, a population-based approach to prevention seeks to shift the underlying distribution of risk among all people; this goal is accomplished by changing the social determinants of health across the entire population, producing greater gains at a population level. In other words, small reductions in risk when distributed across many people can have more of an effect than large reductions in risk among a small number of people. Through the lens of this population-based perspective, and when coupled with knowledge about the ubiquity of childhood adversity, these study results suggest that efforts to reduce levels of childhood adversity—especially when paired with strategies to reduce overall stress exposure and create more protective communities—would go a long way in promoting the mental health of the entire population.

Should these results replicate in other independent samples, a critical next step will be to extend these findings to explore other dimensions of plasticity as they are associated with childhood adversity as a differential susceptibility factor. For example, are there age stages when children are especially vulnerable or sensitive to experiences of adversity? The issue may not simply be a case of whether someone is exposed to childhood adversity, but rather *when* they are exposed. Through efforts to identify whether and when such sensitive periods in development occur,[4] we may be able to understand more deeply the mechanisms giving rise to such plasticity and ultimately tailor prevention efforts in ways that capitalize on such insights, which ideally could yield more effective and efficient interventions.[5]

In addition, the question of whether all experiences of childhood adversity are created equal in shaping differential susceptibility patterns remains. In addition to the timing of adversity, do the type and dosing of adversity matter in determining how people respond to subsequent stressors and life experience? Although summing the number of childhood adversities experienced is often a good first step, efforts to move beyond these simple counts are needed.[6] Such efforts will be key to inform insights regarding disease mechanisms and intervention strategies.

Finally, how does childhood adversity—as a plasticity factor—interact with more commonly studied plasticity factors? To date, genetic variation has been the most commonly studied plasticity factor examined in association with the differential susceptibility model.[7] It may be that some people have greater plasticity, owing to their experience of childhood adversity and their genetic background.

The study by Albott et al[1] provides a strong foundation for future work to evaluate the role of childhood adversity as a possible plasticity factor. In addition to helping to better understand disease mechanisms and opportunities for intervention, such work has the potential to raise awareness of the psychological benefits that could come from reducing stress and promoting positive environments in our entire population, and especially among our country's most vulnerable citizens.

ARTICLE INFORMATION

Published: November 30, 2018. doi:10.1001/jamanetworkopen.2018.5358

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Conflict of Interest Disclosures: None reported.

Funding/Support: This work was supported by grant R01MH113930 from the National Institute of Mental Health.

Role of the Funder/Sponsor: The funding source had no role in the preparation, review, or approval of the manuscript and decision to submit the manuscript for publication.

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