Early-Life Trauma and Resilience: Insights From Developmental Neuroscience for Policy

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Childhood trauma increases the risk for later mental health problems. However, not all individuals who are exposed to early-life trauma go on to develop psychiatric disorders. Delineating factors that promote resilience in the face of adversity is critical for identifying mechanistic targets for intervention and for identifying youths who are at elevated risk. Increasingly, the field of developmental neuroscience is providing novel insights into neurobiological processes of resilience—that is, adaptive brain responses associated with favorable outcomes despite exposure to adversity. These discoveries are positioning developmental and clinical neuroscientists like never before to inform policy and practices that prioritize the fundamental human right to healthy development for all youths.

Research on early adversity and the developing brain has the capacity to inform interventions and practices that promote resilience in myriad ways. These include delineating the effects of risk and protective factors in the early environment, identifying targets for prevention and intervention, and developing and testing the efficacy of interventions for youths and families. Moreover, there is vast heterogeneity in the nature and developmental timing of early adversity, which are likely to contribute to differential neurobiological and mental health outcomes. Knowledge of the developing brain and sensitive periods is essential for determining the types and timing of interventions that will be most effective for youths at specific stages of development or following adversities characterized by particular features such as caregiver involvement.

Developmental neuroimaging has begun to identify neural markers associated with resilience. Among youths exposed to early adversity, stronger frontoamygdala functional connectivity and prefrontal control appear to be protective against internalizing symptoms. In the structural domain, adolescents with exposure to adversity but with lower levels of psychopathology show greater integrity of white matter tracts implicated in the regulation of emotion. Trait resilience, as self-reported by children and adolescents exposed to diverse stressors, is associated with alterations in dynamic resting-state functional connectivity among large-scale brain networks. Within a particular brain state, youths with higher trait resilience show lower connectivity between a network of regions involved in salience and emotion processing with the central executive network and default mode network. Taken together, these findings suggest that resilience may be characterized by both large-scale and circuit-specific neural processes that reflect a stronger ability to engage cognitive control over emotional processes. However, much remains unknown about the potential for such neural findings to contribute to the prediction of mental health outcomes following adversity, particularly over and above more readily obtained psychosocial and clinical information. Future longitudinal studies will be important for testing whether specific neural markers can be leveraged to better predict trajectories of resilience, potentially contributing to enhanced detection of risk and resilience earlier in development.

Delineating the neurobiological mechanisms by which key protective factors or interventions promote resilience among youths exposed to adversity is a major contribution toward understanding resilience and identifying targets for intervention. Having a close bond with a supportive and stable caregiver is one of the strongest protective factors against adversity-related psychopathology. Children show reduced amygdala reactivity and a more mature pattern of regulatory connectivity between the medial prefrontal cortex and amygdala in the presence of parental cues, suggesting a neurobiological mechanism through which caregivers buffer children’s stress reactivity. In line with this idea, stronger parental buffering of amygdala reactivity is associated with lower symptoms of child anxiety and more secure parent-child attachment. Although parental buffering is weaker on average among youths exposed to early parental deprivation, youths who show reduced amygdala reactivity to parental cues exhibit lower anxiety up to 3 years later. Thus, even within a group of youths at heightened risk for anxiety disorders, the capacity for parents to buffer stress and amygdala reactivity may have protective effects on mental health.

Consistent with the role of developmental science in informing the optimal type and timing of interventions, evidence on parental buffering also provides insight into the developmental specificity of parenting effects. Parental buffering of amygdala reactivity appears unique to childhood. However, research with adolescents demonstrates the continued importance of supportive parenting. Specifically, evidence suggests that parents continue to influence adolescents’ brain function in ways that reduce the risk for anxiety and depression. A randomized controlled trial of the Strong African American Families Program showed that enhancing supportive parenting for adolescents living in poverty may prevent reductions in hippocampal and amygdala volumes that are otherwise observed by young adulthood. These findings further indicate that protective effects of supportive parenting on mental health act through certain neural pathways (i.e., frontolimbic circuitry), which can be targeted effectively through psychosocial intervention. Future research will be helpful for delineating the ways in which parenting differentially

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and resilience across levels, interventions that occur at other levels can also have a robust influence on mental health. Development is marked by substantial changes in frontolimbic circuitry, relationships with caregivers and peers, and at all levels of interacting systems. Thus, leveraging knowledge from developmental science has the potential to optimize prevention, early risk identification, and intervention (e.g., for whom and when will interventions be most effective?), as well as to inform policies that promote youth mental health and development. ACC, anterior cingulate cortex; mPFC, medial prefrontal cortex; vmPFC, ventromedial prefrontal cortex. [Brain image adapted with permission from (11).]...
Commentary

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References