

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

Dylan G. Gee

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 (1). 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 (2).

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 (3). 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 (3).

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 [reviewed in (4)]. 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 [reviewed in (5)]. 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 [reviewed in (5)]. 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 (1). 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 (6). 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 (6). 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 (7). 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 (8). 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

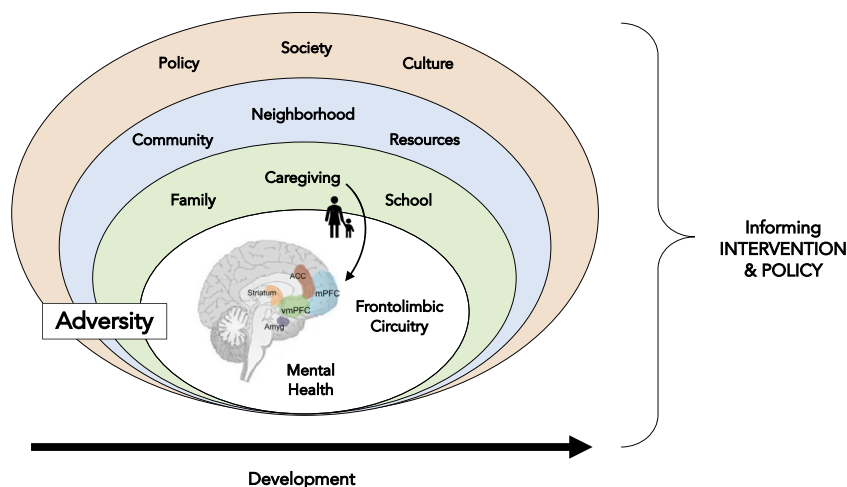


Figure 1. Interacting systems related to risk and resilience following childhood adversity. Childhood adversity occurs in the context of dynamic and interacting systems including psychological and neurobiological functioning, families, schools, communities, and broader society. Resilience to mental health disorders following childhood adversity depends on interactions and cascading effects across these systems (1). At the individual level, connections between the prefrontal cortex and subcortical structures such as the amygdala (Amyg) play a central role in emotion regulation and cognitive control and provide a mechanistic link between adversity and mental health. Supportive caregiving can buffer the impact of adversity on mental health (even when the adversity occurs outside of the family context), and cross-species evidence suggests that these protective effects may occur via modulation of frontolimbic circuitry. Psychosocial intervention can effectively target caregiving, and given the cascading effects of risk

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).]

relates to resilience during specific developmental stages, as well as examining how other sources of social support such as peers may modulate frontolimbic circuitry and contribute to resilience during adolescence.

Research on the effects of early caregiving adversity has had a significant impact on societal practices regarding vulnerable youths. Studies such as the Bucharest Early Intervention Project (9) revealed the profound effects of institutionalized care as well as delineated sensitive periods in neurodevelopment, demonstrating the importance of early intervention and playing a pivotal role in societal shifts from institutionalized care toward foster care. Recently, developmental science was critical for shaping policy related to the detention and separation of migrant families at the U.S./Mexico border resulting from the U.S. government's "zero tolerance" policy on immigration. Guided by a wealth of evidence demonstrating the devastating and lasting effects of forced parent-child separation on brain development and mental health, and the stress-buffering effects of caregivers, developmental scientists significantly contributed to international discussion about this humanitarian crisis (10). Coupled with research on the importance of early intervention, these findings informed policy to guide reunification and access to mental health care for separated families.

Foundational theories of resilience suggest that an individual's capacity for resilience is highly dependent on multiple levels of interacting systems in society (Figure 1). Consistent with this idea, evidence shows that intervening at the family, community, or broader societal level is often most effective for enhancing individual resilience. Positive changes in behavior and mental health are thought to propagate across levels, and even across generations, through "developmental cascades" (1). The field of developmental neuroscience can delineate and provide mechanistic insight into these cascading effects. Research has shown that living in poverty can

negatively affect brain development, and cross-species evidence shows that scarcity of resources disrupts caregiving in ways that increase unpredictability in early environments and alter frontolimbic circuitry in offspring [reviewed in (3)]. Such insights can inform the types of interventions that will most effectively promote well-being, as well as their timing. For example, programs that target cognitive development or caregiving factors could have the greatest positive impact during early childhood (9). Ensuring access to health care, affordable housing, effective schools, high-quality childcare, and paid family leave (1) may have cascades of effects that influence youth well-being. Understanding these effects on youths is essential to shaping policy and to breaking down structural inequities that disadvantage youths from lower-income and minoritized racial and ethnic backgrounds.

Developmental neuroscience has an important role to play in informing policy to reflect the state of the science on healthy brain and behavioral development. Embracing that role will ensure that our research extends beyond the laboratory to guide policy and practices that protect youths from harm, facilitate coping, and provide opportunities for healthy development. Despite the remarkable capacity for resilience that children and adolescents can show, public policy must prioritize the well-being of youths and ensure that the burden of coping with adversity does not fall disproportionately on particularly vulnerable youths and their families.

Acknowledgments and Disclosures

This work was supported by a National Institutes of Health Director's Early Independence Award (DP5OD021370), a Brain and Behavior Research Foundation (National Alliance for Research on Schizophrenia and Depression) Young Investigator Award, a Jacobs Foundation Early Career Research Fellowship, and The Society for Clinical Child and Adolescent Psychology (Division 53 of the American Psychological Association) Richard "Dick" Abidin Early Career Award and Grant.

The author reports no biomedical financial interests or potential conflicts of interest.

Commentary

Article Information

From the Department of Psychology, Yale University, New Haven, Connecticut.

Address correspondence to Dylan G. Gee, Ph.D., Department of Psychology, Yale University, 2 Hillhouse Ave, New Haven, CT 06511; E-mail: dylan.gee@yale.edu.

Received May 1, 2020; revised and accepted Jul 8, 2020.

References

1. Sapienza JK, Masten AS (2011): Understanding and promoting resilience in children and youth. *Curr Opin Psychiatry* 24:267–273.
2. Casey BJ (2019): Healthy development as a human right: Lessons from developmental science. *Neuron* 102:724–727.
3. Gee DG, Casey BJ (2015): The impact of developmental timing for stress and recovery. *Neurobiol Stress* 1:184–194.
4. McLaughlin KA, Lambert HK (2017): Child trauma exposure and psychopathology: Mechanisms of Risk and resilience. *Curr Opin Psychol* 14:29–34.
5. Feder A, Fred-Torres S, Southwick SM, Charney DS (2019): The biology of human resilience: Opportunities for enhancing resilience across the lifespan. *Biol Psychiatry* 86:443–453.
6. Callaghan BL, Gee DG, Gabard-Dumam L, Telzer EH, Humphreys KL, Goff B, *et al.* (2019): Decreased amygdala reactivity to parent cues protects against anxiety following early adversity: An examination across 3 years. *Biol Psychiatry Cogn Neurosci Neuroimaging* 4:664–671.
7. Butterfield RD, Silk JS, Lee KH, Siegle GS, Dahl RE, Forbes EE, *et al.* (2020): Parents still matter! Parental warmth predicts adolescent brain function and anxiety and depressive symptoms 2 years later [published online ahead of print Feb 25]. *Dev Psychopathol.*
8. Brody GH, Gray JC, Yu T, Barton AW, Beach SRH, Galván A, *et al.* (2017): Protective prevention effects on the association of poverty with brain development. *JAMA Pediatr* 171:46–52.
9. Nelson CA, Zeanah CH, Fox NA, Marshall PJ, Smyke AT, Guthrie D (2007): Cognitive recovery in socially deprived young children: The Bucharest Early Intervention Project. *Science* 318:1937–1940.
10. Gee DG, Cohodes EM (2019): A call for action on migrant children's mental health. *Lancet Psychiatry* 6:286.
11. Macdonald AN, Goines KB, Novacek DM, Walker EF (2016): Prefrontal mechanisms of comorbidity from a transdiagnostic and ontogenic perspective. *Dev Psychopathol* 28(4pt1):1147–1175.