Commentary

Biological Psychiatry

Elucidating Neural Mechanisms of Poverty on Child Development Leads Back to Psychosocial Mechanisms

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The finding that experiences of poverty early in life are among the most powerful and prevalent drivers of negative developmental outcomes in children has been well known for many decades (1). This effect, evident in the behavioral developmental literature since the early 1970s, has been further validated and specified in the neuroscience literature over last 2 decades. Numerous independent studies have demonstrated the negative impact of childhood poverty on brain structure and function (2,3). Remarkably, these strong findings have done far too little to impact public policy and related prevention efforts. While there have been several social and political barriers that have contributed to this inaction, some have argued that a key question that must be addressed to facilitate the feasible implementation of widespread prevention is a greater understanding of the neural mechanisms of this process. If neural mechanisms were clarified, key factors in the risk pathway could be more specifically targeted.

In the current issue of *Biological Psychiatry*, Tian *et al.* (4) take an important step in this direction by linking early disparities in socioeconomic status to alterations in integrated cortisol secretion based on intensive sampling of diurnal salivary cortisol. Tian *et al.* then show that the alterations in this hypothalamic-pituitary-adrenal (HPA) activity act as mediators of the effects of low socioeconomic status on several measures of brain function subserving the processing and regulation of negative emotions. More specifically, Tian *et al.* show that children facing poverty have reduced daily cortisol secretion. This general finding has been previously documented in children facing stress and trauma, with effects dependent upon the nature of the stress experience and the age of exposure (5). These hormonal alterations mediated the effects of poverty on several aspects of amygdala prefrontal circuitry known to be involved in emotion processing that were found to be enhanced—a finding the authors interpret as suggestive of the need for increased prefrontal cortex activity when processing a negative emotion owing to inefficient cognitive control. This finding is consistent with and supported by a recently reported association between poverty and functional brain activation during a cognitive control of emotion reappraisal task in a magnetic resonance imaging scanner. This related study offers a mechanistic pathway between brain activation during cognitive control of emotion and impaired emotion regulation in children who are exposed to poverty (6).

These findings advance the literature and are consistent with studies that investigate the effects of adversity on each component of the outlined risk trajectory independently (e.g., dampened cortisol in response to trauma/stress and increased amygdala reactivity to negative stimuli and connectivity with prefrontal cortex in those experiencing stress/adversity). In this way, these findings provide an important link between the well-established adversity-related alterations in the stress hormone system and in brain function during negative emotion processing. However, what is equally evident from these novel results is just how much more work is yet to be done to unravel this risk pathway to clearly and tangibly inform the design of feasible universal prevention strategies. This is because modifying HPA axis activity is difficult to achieve without minimizing stressful experiences or maximizing external support, two well-known yet seemingly unreachable goals on a broad scale.

One key domain that the current results suggest should be studied further is the role that sleep and circadian rhythms play in the impact of adversity on HPA axis hormones and emotion function. One novel aspect of the Tian *et al.* (4) study design was the dense sampling of cortisol during the diurnal cycle (as opposed to a during a stress response). This raises the question of whether chronodisruption (a lack of sufficient restful and restorative sleep and/or irregular sleep cycles), a finding observed in children living in poverty (7), plays a mechanistic role in alterations in the stress system, brain function, and related emotion regulatory behavior. The effects of sleep on child development and emotion regulation is of great interest for two reasons: first, sleep is a modifiable factor that is well known to impact neural systems and to have restorative and synaptic homeostasis effects during slow wave sleep, and second, sleep has well-established effects on learning and memory consolidation (8). In addition, reciprocal relationships between sleep and the HPA axis have been demonstrated, with poor sleep being associated with both elevations and eventually flattening of diurnal cortisol rhythms (9,10). Children living in adverse environments that often lack structure are subject to more noise and external disruptions and are at risk for less regular sleep patterns and chronodisruption as well as poor-quality sleep. This might suggest that a logical next step in investigations of poverty and brain function should include measures of sleep, circadian rhythms, and their effect on diurnal cortisol and brain function.

While studies of the neural mechanisms of adversity are critically important, it is equally critical that they be linked to modifiable behavioral and psychosocial correlates. Until brain circuitry and correlated alterations in neurodevelopmental processes related to adversity are specifically and individually

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mapped to inform direct brain-based interventions (e.g., transcranial magnetic stimulation therapies)—a milestone our field is many decades away from—our interventions, particularly in children, will be focused on behavioral and psychosocial factors. Sleep and circadian rhythms are modifiable factors that have significant impacts on brain and behavioral development but for which the specific mechanisms remain underexplored. This makes this domain an ideal potential target for prevention if its causal role can be established.

While the search for neural mechanisms by which poverty negatively impacts brain development are critical, their close association to behavioral and psychosocial mechanisms remain critical to translate these findings into feasible public health interventions. As in this case, the elucidation of more specific neural pathways can lead us back to psychosocial pathways, which are often safer and more feasible targets for change during development.

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Article Information

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