



HOT TOPICS



The biological embedding of structural inequities: new insight from neuroscience

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Neuropsychopharmacology often fails to appropriately consider the impact of race-related structural inequities which perpetuates inequity in mental health research and treatment [1]. Although race is not a biologically meaningful construct, neuroscience studies often include racial categories in analyses without considering potential impacts of racial inequity on mental health. In the United States, structural inequities created and reinforced by structural racism - such as economic hardship, neighborhood disadvantage, and trauma exposure - disproportionately affect Black and Hispanic individuals [2]. Rather than reflective of inherent differences, race-related differences in neurobiology are likely due to biological embedding of structural inequities.

Trauma and stress-related disorders, such as posttraumatic stress disorder, are thought to be driven by dysfunction of core threat neurocircuitry, such as the amygdala, hippocampus, and prefrontal cortex (PFC) [3]. The amygdala is central to learning cue-threat relationships and threat response expression. Emergent work, however, suggests that structural inequities can lead to race-related variability in amygdala and physiological reactivity (e.g., skin conductance responses; SCRs) to threat. Black individuals exhibit lower threat-elicited amygdala reactivity and SCRs to threat compared to White individuals, but this difference is predominately driven by the greater exposure to violence, lower income, and more disadvantaged neighborhoods for Black individuals [4]. Thus, chronic exposure to structural inequities plays a role in shaping the amygdala's response to threat.

Inequity-driven modulation of threat-processing in the amygdala further appears to be important for understanding race-related variability in PTSD. In a large study of recent trauma survivors, Black and Hispanic individuals showed greater connectivity between the amygdala and salience network (e.g., insula) compared to White individuals [5]. Critically, race-related variability connectivity patterns were differentially associated with 3-month PTSD symptoms such that decreased connectivity was positively associated with PTSD in Black participants but *inversely* associated in Hispanic participants. These findings suggest that developing effective neuroscience-based PTSD treatments for all individuals will require careful consideration of structural inequities.

It remains unclear how structural inequities influence neurodevelopment. However, some insight was gained by leveraging the Adolescent Brain Cognitive Development Study. Race-related

differences within threat neurocircuitry were visible in children as young as 9 years old [6]. Across numerous metrics, Black children were differentially exposed to structural inequity compared to White children [6]. Lower volumes of the PFC and amygdala were associated with greater adversity. In turn, group differences in the PFC, but not the amygdala, were largely driven by the biological consequences of structural inequities. Thus, the impact of structural inequities is discernable across the life course and may contribute to neural susceptibility for trauma-related disorders.

Structural inequities appear to drive race-related differences in the neurobiology of trauma and stress-related disorders. While race is not a biologically meaningful construct, *structural racism* meaningfully influences neurobiology of trauma and stress-related disorders. Due to (warranted) fears of legitimizing racist beliefs, race-related differences in neurobiology are not frequently investigated or, if they are, not adequately interpreted. However, intentionally probing biological consequences of structural inequities and underlying mechanisms is necessary to ensure findings are accurately understood and to address mental health inequities.

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ADDITIONAL INFORMATION

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