The influence of bullying and victimization on the HPA axis and inflammation: A systematic review

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Background: Early life stress (ELS), such as bullying, may affect health through the body's physiological stress response systems, e.g., inflammation and the hypothalamic-pituitary-adrenal (HPA) axis. The purpose of this study was to gain a better understanding of current research findings on the influence of bullying on physiological stress biomarkers.

Methods: For the study, we conducted a systematic review using key search terms including child trauma, inflammation, and cortisol in Pubmed ($N = 28$ empirical articles) to assess the link between childhood adversity, the HPA axis, and inflammation. Then, we identified the studies that (1) look at some form of peer victimization or bullying, and (2) measured HPA or inflammatory biomarkers.

Results: We identified 28 published, empirical articles describing the association between childhood adversity that includes some form of peer victimization or bullying exposure and functioning of physiological stress response systems. Among these studies, 53.6% ($N = 15$) observed that childhood adversity was associated with elevated biomarkers of stress physiology, and 39.3% ($N = 9$) found that childhood adversity was associated with attenuated biomarkers of stress physiology. The methodological details of these studies will be presented.

Conclusion: A majority of results from the studies showed elevated biomarkers in the context of childhood adversity including bullying, but nearly 40% showed an attenuated association. More research is need to understand how bullying contributes to our understanding of emerging health disparities within the broader ELS literature.

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Physiologic reactivity to and recovery from acute social-evaluative stress: Differential associations with preadolescents' internalizing and externalizing problems

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Background: This study aims to identify phase-specific patterns of HPA-SAM co-activation during both reactivity to and recovery from acute stress that are differentially associated with preadolescent internalizing and externalizing problems.

Method: 149 preadolescent-parent dyads completed interviews and questionnaires in the afternoon (3–5 pm). Preadolescents also completed the TSST-M, starting 45 min after arrival to the lab. Seven saliva samples were taken over the course of the 95-min visit.

Results: Regression models explained 41% of the variance in parent-reported externalizing and 22% of child-reported internalizing problems. After accounting for puberty, medications, sex, and stress, salivary alpha-amylase (sAA) reactivity and cortisol (sC) recovery slopes each predicted externalizing, such that higher sAA reactivity and lower sC recovery were associated with more externalizing. The interaction between recovery sAA and sC significantly predicted externalizing—the highest levels of externalizing were in preadolescents with low recovery sC and sAA. sC reactivity and sC recovery slopes both independently predicted internalizing, such that higher sC reactivity and lower sC recovery were associated with higher internalizing. The interaction between reactivity sAA and sC significantly predicted internalizing—the highest levels of internalizing were associated with asymmetric activation–either low sC-high sAA or high sC-low sAA.

Conclusion: This study confirms prior research indicating that concurrent low HPA and SAM activation is associated with externalizing, and extends it by isolating this effect to the recovery phase. Similarly, we replicate prior findings that asymmetric HPA-SAM activation is associated with internalizing, and extend the research by isolating this effect to the reactivity phase.

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AGING AND COGNITIVE IMPAIRMENT

Increased cortisol reactivity predicts cognitive impairment not dementia during aging: a follow-up study

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Background: Individuals with Alzheimer's disease (AD) show high cortisol levels suggesting that biological mediators of stress may be involved in the neurodegenerative process of cognitive disorders. However, there is no consensus whether altered cortisol profile represents a risk factor for the development of cognitive impairment. We aimed to analyze whether cortisol concentration under basal and acute stress conditions could be associated with the incidence of cognitive impairment during aging.

Methods: One hundred-twenty nine individuals 50 years and older, with preserved cognitive and functional abilities were recruited in 2011 for assessment of cognitive performance and cortisol levels. Cortisol was determined in saliva samples collected as following: during two typical and non-consecutive days at morning, afternoon and night and during the exposure to an acute psychosocial stressor (Trier Social Stress Test – TSST). After five years of follow-up, 69 individuals were reassessed for cognitive impairment diagnosis, functional evaluation, memory complaints and depression.

Results: The incidence of cognitive impairment not dementia (CIND) was 26.1% and was associated with greater increase in TSST-induced cortisol concentration (responsiveness) [(95% CI = 1,001–1,011; $B = 0.006$), $p = 0.027$]. Moreover, participants with CIND had, five years before the diagnosis, greater responsiveness to TSST ($p = 0.019$) and lower waking cortisol response ($p = 0.018$) compared to those who did not develop CIND. Conclusion: These findings suggest that higher psychosocial stress responsiveness profile may represent a risk for development of cognitive disorders during aging.

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