

**Kuhlman, Kate R.**

**Abstract**

Approximately 12% of youth will be exposed to severe childhood adversity which has been linked to more than double the risk for psychopathology across the lifespan, increased annual health care costs, early mortality, and lower socioeconomic achievement. Childhood adversity may be linked to this panoply of health disparities through alterations to key physiological systems, including one component of the body's physiological stress response system, the HPA axis, and the innate immune system. Indeed, cortisol and inflammation, the resulting outputs of these systems have profound effects on the body and the brain. In particular, inflammation and cortisol have implications for positive valence domains, such as those related to motivation for and consumption of rewards, as well as cognitive control, including inhibitory control. These cognitive and affective domains have cross-cutting implications for decision-making, risk-taking, learning, and maintenance of a supportive social network. This is particularly relevant during adolescence when risk for psychopathology increases dramatically. The purpose of this pilot study is to examine the associations between childhood adversity severity and functioning in positive valence and cognitive control domains in a sample of adolescents (ages 11-17) over-sampled for exposure to adversity. Additionally, we will examine the association between childhood adversity exposure and circulating, cellular, and intracellular markers of inflammation or physiological stress. Finally, we will examine whether biological markers of stress and inflammation mediate the association between childhood adversity and cognitive-affective functioning. To do this, we will recruit a sample of 30 adolescents (ages 11-17) exposed to high (4+ adverse childhood events) and low (0-3 adverse childhood events) adversity. During a laboratory visit, youth will complete clinical self-report and experimental measures of cognitive control and reward motivation and undergo a structured-psychosocial stressor. Throughout this laboratory assessment, we will collect blood and saliva to measure systemic, cellular, and intracellular markers of inflammation and HPA axis functioning. We anticipate that data from this study will support our hypothesis that childhood adversity is associated with diminished cognitive control and increased reward motivation and decreased consummatory value of reward under conditions of stress in these adolescents. Completion of this study will identify provide preliminary data for several modifiable risk factors (physiological stress reactivity, inflammation, reward motivation, and cognitive control) for health disparities in at risk youth, and provide preliminary evidence to support an R01 (or similar) application examining cognitive and behavioral strategies that may mitigate risk for psychopathology in youth exposed to adversity who go on to develop psychopathology.