

College of Science Neuroscience Faculty Candidate

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Tuesday, October 13, 2015
310 Kelly Hall
3:00pm – 4:00pm

“Prefrontal cortical circuitry integrates behavioral and homeostatic stress responses”



Alterations in the processing of stressful information are proposed to underlie psychiatric conditions such as mood and anxiety disorders. Additionally, stress frequently leads to somatic conditions, including cardiovascular disease. In fact, heightened reactions to acute psychological stress and chronic stress exposure are both associated with depressive illness and increased cardiovascular mortality. The prefrontal cortex is a brain region implicated in stress appraisal, regulating behavioral and homeostatic responses to stress. Thus, prefrontal activity may play a role in the consequences of stress exposure and account for the co-morbidity of mood disorders and cardiovascular disease. My presentation will discuss the overarching hypothesis that prefrontal glutamatergic circuits integrate behavioral, autonomic, and endocrine responses to stress. These studies, in rodents, have identified the posterior hypothalamic nucleus as a critical substrate linking prefrontal function with behavioral and physiological output. Further, site-specific viral genetics to chronically reduce glutamatergic outflow from prefrontal cortex indicate that glutamate release from prefrontal neurons is necessary to integrate the effects of chronic stress on depression-related behavior with cardiovascular and neuroendocrine stress reactivity. I will also discuss ongoing experiments that combine glutamate receptor pharmacology with optogenetic terminal leveraging to define the specific signaling mechanisms leading to enhanced autonomic responsiveness and behavioral dysregulation after chronic stress. Collectively, my presentation will highlight my current and future research efforts aimed at elucidating the neurocircuitry of stress-related illness.

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