Abstract

Integration of behavior and autonomic function in prairie voles: a focus on mechanisms underlying the association of mood disorders and cardiovascular disease

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The research being conducted in my laboratory in the Department of Psychology focuses on using behavioral and physiological approaches to investigate the hypothesis that responses to the social environment and neural mechanisms involving oxytocin underlie the documented association between mood disorders and cardiovascular disease (CVD). Disorders relating to negative affect, such as depression and anxiety, are associated with dysregulation of the cardiovascular system and CVD, both in current cardiac patients and in individuals with no history of cardiovascular pathophysiology (Van der Kooy et al., 2007; Glassman, 2007; Grippo and Johnson, 2009). Psychological and physiological responses to environmental and social stressors may represent common mechanisms that underlie depressive signs and cardiovascular dysregulation. Furthermore, oxytocin is associated with stress-buffering effects, and mediates behavioral and physiological processes associated with stress and social behavior (Carter, 1998; Pedersen and Boccia, 2002). Therefore, the current series of experiments is focused on investigate these mechanisms in a rodent model, the socially monogamous prairie vole, as they relate to depression-relevant behaviors and dysregulation of the cardiovascular system. These studies will enhance our understanding of mechanisms underlying the association of mood disorders and CVD in humans.

This research involves a novel, integrative approach to studying common mechanisms that may underlie depressive behaviors and cardiovascular dysfunction, which can lead to the development of more comprehensive treatments for patients with comorbid depression and CVD. This research plan will investigate in the prairie vole behavioral, neuroendocrine, autonomic, and cardiac responses to a species-relevant stressor (social isolation), and will examine the hypothesis that oxytocin administration will prevent the behavioral and physiological changes associated with social isolation. Converging evidence suggests that the physiological responses of social mammals, including prairie voles, are particularly sensitive to social experiences. Further, preliminary data from my laboratory indicate that the autonomic nervous system in prairie voles has shared features with that of humans, including a high level of parasympathetic activity not previously observed in rodents. Therefore, the prairie vole model provides a unique opportunity to examine neurobiological mechanisms through which the social environment impacts behavior, brain function, and cardiovascular responses. The specific aims and associated experimental questions for the current proposal are as follows:

Specific Aim 1: To examine the hypothesis that social isolation induces behavioral, autonomic, and neuroendocrine changes that are relevant to depressive syndromes and cardiovascular dysregulation.

1a. Does social isolation lead to depression-relevant behaviors (anhedonia and behavioral despair), and does social pairing protect against these changes?

1b. Does social isolation lead to altered cardiac and autonomic function [heart rate, heart rate variability, vagal tone, and ventricular arrhythmias], and does social pairing protect against these changes?

1c. Does social isolation induce alterations in circulating and central nervous system stressor-reactive hormones and peptides, and does social pairing protect against these changes?

Specific Aim 2: To examine the hypothesis that oxytocinergic mechanisms underlie the behavioral, autonomic, and neuroendocrine responses to social isolation.

2a. Does long-term administration of oxytocin prevent the behavioral, autonomic, and neuroendocrine changes associated with social isolation?

2b. Does administration of an oxytocin antagonist block the effects of oxytocin on the behavioral, autonomic, and neuroendocrine changes associated with social isolation?