

Plasma Oxytocin, Immune Function and Depression in Women

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There is now substantial evidence that perceptions of available social support protect persons from pathogenic effects of depression and stressful events. However, little is known about the neurobehavioral pathways that link social support to improved physical and mental health outcomes. A growing literature suggests that negative affect is associated with change in immune function that may render the individual more susceptible to disease. In this regard, it is widely proposed that activation of the HPA axis and the SAM axis are biological pathways through which negative affect can influence immune function and, in turn, increase disease risk. In this proposal we begin to explore the possibility that one pathway by which social support can protect persons from the pathogenic effects of negative life events is via a reduction in HPA and SAM activity linked to activity of the hypothalamic neurohormone, oxytocin. This hormone is associated with affiliative behaviors in women and is known to down regulate the HPA and SAM stress axes. By reducing activation along these pathways, oxytocin may buffer the impact of stress on immune function and thus on disease processes, accounting for observed associations between social support and improved health outcomes in women. The proposed pilot study is designed to provide a preliminary examination of the role of oxytocin in women's stress sensitivity. Twenty-five normal cycling, depressed females aged 21-40 and 22 age-matched never-depressed controls will be recruited to participate in a 3-hour laboratory experiment designed to stimulate, measure and compare peripheral oxytocin release and basal oxytocin concentration within and between groups, and to examine whether peripheral oxytocin release is associated with a down regulation of activity along the HPA/SAM axes and a decrease in stress-induced immune modulation following an acute stress task.