**The Effect of Paced Breathing on Autonomic and Immune Functioning**
***PI: Aric A. Prather, M.S.***

Emerging evidence indicates that the autonomic nervous system (ANS) plays an important role in modulating the immune system, particularly in regulating the magnitude of the innate inflammatory response. Specifically, animal models and preliminary human evidence suggests that low levels of parasympathetic activity can increase the magnitude of the inflammatory response, characterized by elevated production of the pro-inflammatory cytokines interleukin (IL)-6, IL-1β, and tumor necrosis factor (TNF)-α —providing evidence for a mechanism by which dysregulated autonomic-immune homeostasis can increase risk for inflammatory diseases, such as cardiovascular disease and rheumatoid arthritis. The goal of the proposed study is to further explore this pathway by employing a paced deep breathing intervention designed to increase parasympathetic (vagal) activity and determine whether this results in a decrease in pro-inflammatory cytokine production in response to an in vitro inflammatory challenge. To this end, we will recruit sixty healthy undergraduate volunteers, aged 18-40, to participate in a 1 hour laboratory session. Following a 20 minute resting baseline period, participants will be randomly assigned to either 20 minute resting control condition (n=20) or a paced deep breathing (6 breaths/minute) intervention (n=40). Parasympathetic activity, as indexed by spectral analysis of heart rate variability and baroreflex sensitivity, will be assessed continuously throughout the baseline and task periods. Blood samples will be obtained toward the end of both periods for determination of immune reactivity. We hypothesize that those in the paced breathing intervention will show an increase in parasympathetic activity and a related decrease in pro-inflammatory cytokine production when compared to those in the resting control condition. It is our hope that findings from this study may lead to the development of non-invasive interventions that may be of benefit in the clinical management of chronic inflammatory conditions. In the event that our hypotheses are confirmed, we will use these data as pilot findings to support an external grant proposal to further explore the role of parasympathetic activity in regulating inflammation among individuals with or at risk for chronic inflammatory conditions