Measurement of Inflammation and Oxidative Stress in Patients with Post-Deployment Dyspnea

Emily Chung and Andrew Gow
Rutgers, The State University of New Jersey

Military personnel were exposed to burn pit smoke and other respirable toxicants (e.g. geological dust, chemical hazards, regional air pollution) during deployment in Iraq and Afghanistan. Post-deployment, a particular subset of U.S. veterans has presented with new-onset dyspnea, while unusually still maintaining regular pulmonary function. Particulate exposure increases cellular production of reactive oxidants. The resulting oxidative stress is capable of compromising endothelial functionality, either directly through cellular damage, or indirectly through consumption of nitric oxide (NO). We hypothesize that these novel symptoms of dyspnea are caused by a decrease in both pulmonary and systemic endothelium function, and, consequently, a reduced ability to regulate vascular tone in these areas. Physiologically, this would present as either poor flow/perfusion (V/Q) match in the pulmonary circuit, or impaired hypoxic vasodilation in the systemic circuit, both of which could contribute to poor oxygen delivery to the tissues. Veterans will be recruited as subjects to test exercise-induced responses in pulmonary blood flow and systemic arteriole resistance. Capacity for hypoxic vasodilation will be measured using flow-mediated dilation (FMD) in the brachial artery. Additionally, we will take sputum and blood samples to assess inflammatory status and NO production. Due to NO's short half-life, measurements of nitrite (NO_2^-) and nitrate (NO_3^-) will be used as indications of NO bioavailability. Subjects with the highest circulatory inflammation are predicted to have the worst FMD values and lowest overall NO production. Higher ratios of non-functional NO_3^- in comparison to functional NO_2^- would indicate greater oxidative stress, and therefore greater NO consumption. If these results are observed, we can conclude that the mechanism of symptomology is rooted in a state of elevated inflammation throughout the circulation. Supported by ASPET SURF, NIH R25ES020721, NIH ES005022, and the East Orange VA Medical Center.