Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) are pulmonary disease states associated with capillary leak, widespread pulmonary edema, and poor oxygenation. While activated neutrophils are the likely effector cells responsible for ALI and ARDS, the overall immunopathology of ALI and ARDS remains to be clearly defined. The Th-17 immune response (IL-17, -21, -22, -23) has been implicated in a multitude of inflammatory disease states. Specifically, Th-17 T-cells, such as γδ T-cells, play a major role in animal models of pulmonary inflammation and infection. Based on these experimental findings, we hypothesized that the Th-17 response also plays a role in the development of inflammatory and infectious pulmonary complications in trauma patients.

In our study, we evaluated the Th-17 response and immune cell populations in blood and BAL fluid from trauma patients with pulmonary complications. Thus far, we have enrolled 21 subjects from the Surgical ICU who were severely injured, mechanically ventilated, and undergoing a BAL for clinical care. We have analyzed cell phenotypes from blood and BAL in all 21 subjects as well as blood and BAL cytokine levels from the first 10 subjects. Additionally, we have examined blood cytokine levels and cellular phenotypes in 6 healthy normal volunteers.

In this seminar we will discuss the background, hypothesis, methods, and new results from our study. We will examine the relationships, if any, between the presence of pneumonia, the transfusion requirements, and the presence or absence of ALI. We will examine how these various clinical states may relate to blood cellular phenotypes and blood cytokine levels.