The development of non-infectious pulmonary complications (ALI, ARDS) and multiple organ dysfunction syndrome (MODS) hampers the recovery of patients with severe traumatic injuries. While current evidence suggests that the activation of a pro-inflammatory cascade plays an important role in this pathogenic process, the identification of specific mediators has been elusive. Recently, a novel T-cell response, the Th-17 response, has been shown to be intricately involved in other non-traumatic lung diseases, as well as multiple extra-pulmonary inflammatory diseases. With the recent discovery of this novel T-cell response (Th-17) and the main effector cytokine, IL-17, there is the potential for further categorizing of the biologic mechanism(s) leading to ALI and ARDS.

We hypothesize that those patients with traumatic injury and the subsequent development of ALI and ARDS will have an increased Th-17 inflammatory response both systemically and locally. In our prospective clinical study thus far, 18 subjects from the SICU who have had severe traumatic injury and a pulmonary complication necessitating a bronchoscopy have been enrolled. Both their pulmonary and systemic inflammatory responses have been analyzed with regard to the Th-17 response, inflammatory makers and immune cell phenotypes. Our data to date shows that the majority of subjects with acute lung injury have an increase in the systemic level of the cytokines involved in the Th-17 response. However, pneumonia is a common precursor to ALI and ARDS and it may influence the ultimate cytokine profile independent of injury.

This seminar will open with a brief introduction, a review of the methods, presentation of new results, and finally a closer look at the local inflammatory response in the bronchoalveolar lavage (BAL) fluid of the enrolled subjects.