Acute respiratory distress syndrome (ARDS), a severe form of acute lung injury (ALI), results in moderate to severe loss of lung function and is associated with significant morbidity and mortality. There are an estimated 190,000 cases per year just in the US and new cases are continually being reported, especially now with the COVID-19 pandemic. Platelets have been shown to play a role in the progression of ARDS/ALI, highlighting an interplay between inflammation and coagulation, although specific pathways are still not defined. The receptor TREM Like Transcript-1 (TLT-1), found in alpha-granules of platelets and megakaryocytes, is a membrane surface receptor that upon platelet activation, is rapidly brought to the surface of these cells and has been shown to play a role in platelet aggregation, hemostasis and more recently, inflammation. Our lab has shown that TLT-1 binds to fibrinogen and has a role in the innate immune functions of platelets. However, the underlying molecular mechanisms between fibrinogen and TLT-1 are still unknown. The main goals of my study are to identify and dissect the biological mechanisms of TLT-1 influence on hemostasis and inflammation in the lung with the long-term goal of developing improved treatment methods for ALI patients.