Dyslipidemia Promotes Lung Injury
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Background: Obesity is considered an epidemic today; rates of affliction have more than doubled in adults, from 15% in 1980 to 33% in 2004. Obesity is not only associated with a decrease in the quality of life, but also with numerous diseases. ARDS, or Acute Respiratory Distress Syndrome affects about 200,000 patients in the United States and carries a high mortality rate. This results from numerous insults including infection or trauma, where leukocytes infiltrate the lung causing tissue damage, which can lead to respiratory failure. Recent studies have shown that obese patients may be more susceptible to developing. One hypothesis is that a ‘priming’ effect of the endothelium is created in obese mice via proinflammatory mediators, which causes increased leakage through the endothelium into the lung space.

Methods: We induced lung injury in lean and obese mice through inhaled lipopolysaccharide (LPS) to mimic ARDS. Albumin levels were examined at different time points from BAL using western blots as a way to measure inflammation and capillary leak in the lungs. Bone marrow neutrophils were isolated from lean and diet induced obese mice by discontinuous density gradient centrifugation. These neutrophils were labeled with indium-111 and then passed through a 5-micrometer pore filter, comparable to width of the pulmonary capillaries. The percentage of cells retained in the filter was determined by gamma-counting.

Results: After 6 and 24 hours of exposure, it was found that generally lean mice had higher albumin levels in their lungs, but results are currently inconclusive. In the filter experiment, we noted that 7% more fat mouse neutrophils were retained compared to lean.

Conclusions: We conclude that dyslipidemia may promote lung injury indicated by albumin levels inside of the lung; Filter experiments suggest that neutrophil sequestration and activation may drive this process.