

Session/Poster#

Presenter

B06

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Phospholipid Transfer Protein Deficiency Enhances Lung Inflammation In Mice

Phospholipid transfer protein (PLTP) facilitates the transfer of phospholipids from triglyceride-rich lipoproteins into HDL. PLTP activity is subdued in the lungs of emphysema patients which results in elevated inflammation responses.

Alpha-1 antitrypsin (AAT) protein can protect extracellular PLTP from proteolytic degradation. Serine proteases (cathepsin G and neutrophil elastase) degrade PLTP protein in the airways.

Both AAT and PLTP reduced neutrophil degranulation and superoxide production, partially through their inhibition of the Src tyrosine kinase, Hck.

Deficiency of AAT could contribute to reduced lung PLTP activity and elevated neutrophil signaling associated with lung disease.

Here we present data on Pltp deficient mice administered LPS and AAT and look at several key inflammatory responses. Equally, we look at PLTP activity in AAT deficient mice during LPS-associated inflammation.