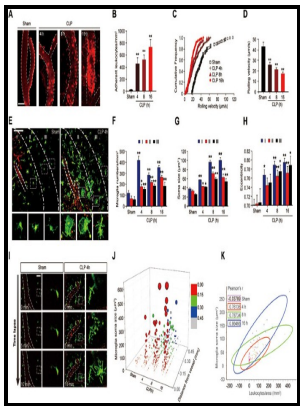
Role of CD18 dependent neutrophil adhesion in septic acute lung injury

- - Contribution of Neutrophils to Acute Lung Injury



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Group Ps demonstrated a significant increase in plasma TNF activity by 30 min 2. However, active PMNs that migrate back to systemic circulation may disseminate inflammatory responses to remote organs and tissue. Little, however, is known regarding the mechanisms underlying reconstitution of a damaged glycocalyx.

The Role of Adhesion Molecules in Acute Lung Injury

LTB4 induces PMN release of neutrophil elastase NE , which in turn cleaves endothelial JAM-C and subsequent damage of endothelial junction and promotes PMN rM.

The Role of Adhesion Molecules in Acute Lung Injury

In select experiments, we maintained mice at 60% FiO 2 in a hyperoxia chamber. Thus, we defined the role of neutrophils and specifically neutrophil iNOS in a murine model of septic ALI.

Specific role of neutrophil inducible nitric oxide synthase in murine sepsis

Data are represented as means ± s. Neutrophil emigration into the lungs is not sufficient to cause ALI; neutrophil activation is also required. The hydrodynamically relevant endothelial cell glycocalyx observed in vivo is absent in vitro.

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Recovery of cremasteric ESL thickness occurs 72 h after local high-dose TNF-α injection ; it is unclear whether this recovery period is a function of constitutive GAG turnover with reconstitution occurring after cessation of the inciting inflammatory insult or reflects an induction of endothelial repair processes. Notably, we observed no CLP-associated heparanase induction in the cremaster, a tissue with stable ESL thickness during endotoxemia. Both authors read and approved the final manuscript.

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