Introduction:
Reorganization of endothelial barrier complex is critical for increased endothelial permeability implicated in the pathogenesis of acute respiratory distress syndrome. We have previously shown hepatocyte growth factor (HGF) reduced lipopolysaccharide (LPS)-induced endothelial barrier dysfunction. However, the mechanism of HGF in endothelial barrier regulation remains to be unclear.

Methods:
Recombinant murine HGF with or without mTOR inhibitor rapamycin were introduced on mouse pulmonary microvascular endothelial cells (PMVECs) barrier dysfunction stimulated by LPS. Then, endothelial permeability, adherent junction protein (occludin), endothelial injury factors (Endothelin-1 and von Willebrand factor), cell proliferation and mTOR signaling associated proteins were tested.

Results:
Our study demonstrated that HGF decreased LPS-induced endothelial permeability and endothelial cell injury factors, and attenuated occludin expression, cell proliferation and mTOR pathway activation.

Conclusion:
Our findings highlight activation Akt/mTOR/STAT-3 pathway provides novel mechanistic insights into HGF protective regulation of LPS-induced endothelial permeability dysfunction.
Figure 2. Western blot analysis of HGF on mTOR signaling pathway