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The role of SIRT1 in LPS-induced lung endothelial barrier dysfunction and lung injury *in vivo*

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Introduction: It is not completely clear about the mechanism of the endothelial hyper-permeability in acute lung injury.

Objectives: The aim of this study was to determine the role of protein deacetylase SIRT1, one of the nicotinamide adenine dinucleotide (NAD+)-dependent intracellular silent information regulators, in lipopolysaccharide (LPS) induced lung endothelial barrier dysfunction and lung injury *in vivo*.

Methods: The cultured human pulmonary endothelial cells (HPECs) were exposed to LPS and C57BL/6 mice administered intratracheally, and were treated with SIRT1 activator SRT1720 or inhibitor EX527 after LPS exposure, respectively. The endothelial permeability was measured in transwell with the use of FITC-dextran. Lung injury was studied by measuring vascular permeability, histopathological examination, nature of infiltrating cells and inflammatory cytokine induction in the bronchoalveolar fluid.

Results: In cultured HPECs, LPS increased endothelial permeability in parallel with a decrease in SIRT1 expression. Consistent with this observation, SIRT1 activation with the potent sirt1 activator SRT1720 attenuated LPS-induced endothelial hyper-permeability *in vitro*, but increased by the SIRT1 inhibitor EX527. Intra-tracheal administration of LPS (5 mg/kg) in mice reduced SIRTs expression in lung tissue extracts, increased protein content and cell count in bronchial alveolar lavage fluid, and increased Evans blue dye infiltration into the lung. Pretreatment with SRT1720 reduced the lung injury in LPS-treated WT mice, and EX527 accentuated the lung injury.

Conclusions: We concluded that, SIRT1 plays a role in LPS-induced endothelial barrier dysfunction and that its activation attenuated the endothelial barrier dysfunction and lung injury.

Biography

Weizhong Jin got his degree from FudanUniversity in 2012 and has been working as a internal resident in Hangzhou First People's Hospital, China since graduation.

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