≈ POSTER PRESENTATIONS ≈

PP - 001

NLRP3 inflammasome activation is essential for paraquat-induced acute lung injury

Zhenning Liu, Min Zhao

Shengjing Hospital of China Medical University

Abstract

Objective: The innate immune response is important in paraquat-induced acute lung injury, but the exact pathways involved are not elucidated. The objectives of this study were to determine the specific role of the NLRP3 inflammasome in the process.

Methods: Acute lung injury was induced by administering PQ intraperitoneally. NLRP3 inflammasome including NLRP3, ASC and caspase-1 mRNA and protein expression in lung tissue and IL-1β and IL-18 levels in BALF were detected at 4, 8, 24 and 72 h after PQ administration in rats. Moreover, rats were pretreated with 10, 30 and 50 mg/kg NLRP3 inflammasome blocker glybenclamide respectively 1 h before PQ exposure. At 72 h after PQ administration, lung histopathology changes, NLRP3, ASC and caspase-1 protein expression, as well as secretion of cytokines including IL-1β and IL-18 in BALF were investigated.

Results: The NLRP3 inflammasome including NLRP3, ASC, caspase-1 expression and cytokines IL-1β and IL-18 levels in PQ poisoning rats were significantly higher than that in the control group. NLRP3 inflammasome blocker glybenclamide pretreatment attenuated lung edema, inhibited the NLRP3, ASC and caspase-1 activation, decreased IL-1β and IL-18 levels in BALF.

Conclusions: In conclusion, the NLRP3 inflammasome is required for paraquat-induced acute lung injury and the NLRP3 inflammasome inhibition can partially protect the lung against inflammatory injury induced by PQ in rats.