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Abstract

Effect of Cigarette Smoking and Honey on Lung Inflammation by Lipopolysaccharide (LPS) in Mice.

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Background: Cigarette smoke (CS) is a risk factor of pulmonary diseases. Honey is used as a traditional medicine for colds, skin inflammation. It is known that Lipopolysaccharides (LPS) induces lung inflammation. We previously reported that CS inhibited the immune functions. However, it is unclear the effect of CS and honey on LPS induced lung inflammation. In this study, we investigated whether CS and honey affect LPS-induced lung inflammation. **Methods:** Mice were exposed to CS for 10 days. After expose to CS, mice were inhaled 60μg of LPS by intranasal administration. Mice were inhaled 600 μg of Japanese honey and 1 day later, mice were inhaled LPS. After 1 day, broncho-alveolar lavage cells were obtained. Expressions of TLR4, CD14 surface antigen and reactive oxygen species productions were measured by FACS. Cytokines and NF-κB mRNA expressions were assayed by RT-PCR. **Results:** Neutrophils were significantly increased with LPS inhalation. Expression of TLR4 in neutrophil was significantly decreased by CS. Hydrogen peroxide production from neutrophil was significantly increased by CS. IL-1β, TNF-α, CXCL1 and NF-κB mRNA expressions of neutrophil were not difference by CS. Honey inhibited infiltration of neutrophils to the lung , IL-1β and CXCL1 mRNA expressions. **Conclusions:** These results suggest that the recognition for bacteria of neutrophil is inhibited by CS. This inhibition may be resulted in increase of pulmonary infection and cause to exacerbation by infection. Honey indicated anti-inflammation activity via the suppression of infiltration of neutrophils to the lung. Honey may be a candidate as anti-inflammatory drug in pulmonary.