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Title: The effect of lipopolysaccharide-contaminated ovalbumin on airway inflammation and remodeling in a chronic murine asthma model

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Body: Background: The contaminated lipopolysaccharide(LPS) in commercial ovalbumin(OVA) may affect the immune response to OVA in a murine asthma model. We hypothesized that LPS in commercial OVA can decrease allergen induced eosinophilic inflammation and airway remodeling. Methods: BALB/c and C57BL/6 mice were sensitized with OVA emulsified with aluminum hydroxide on day 0 and 14. Phosphate buffered saline, commercial OVA or LPS-depleted OVA was instilled intranasally two times a week for six weeks from day 21. At 24hrs after the last OVA exposure, airway hyperresponsiveness was measured. Bronchoalveolar lavage fluids(BALF) and lung tissues were collected 48hrs after the last OVA exposure. Goblet cell hyperplasia and subepithelial fibrosis were quantified by immunohistochemistry and histomorphometric techniques. Enzyme-linked immunosorbent assay was performed to measure IL-4, MUC5AC, and MMP-9 levels in BALF. Results: Airway hyperresponsiveness, total cell numbers in BALF, and goblet cell hyperplasia were not different between commercial OVA and LPS-depleted OVA group. However, in the commercial OVA group, BALF neutrophils were greater and eosinophilis were lower than in LPS-depleted OVA group in both BALB/c and C57BL/6 mice. In BALB/c mice, subepithelial fibrosis and IL-4 and MMP-9 levels in BALF were significantly less in commercial OVA group compared with the LPS-depleted OVA group. Conclusion: The LPS contamination in commercial OVA decreased the influx of eosinophils in the airways in BALB/c and C57BL/6 mice and inhibited subepithelial fibrosis in BALB/c mice after chronic OVA exposure.