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Title: Interleukin-17A in the pathogenesis of elastase-induced emphysema in mice

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Body: Background: Recent studies show that interleukin (IL) -17A is highly expressed in the lungs of patients with chronic obstructive pulmonary disease (COPD) and in the emphysematous lungs of mice after long-term cigarette smoke exposure. However, the role of IL-17A in the pathogenesis of emphysema is still unknown. In the present study, we examined the role of IL-17A in the development of elastase-induced emphysema using Il-17A gene-deficient (Il-17a$^{-/-}$) mice. Methods: Porcine pancreatic elastase (PPE) or phosphate buffered saline (PBS) was administered intratracheally in Il-17a$^{-/-}$ and wild-type (WT, C57BL/6J) mice on day 0. IL-17A mRNA expression in the lungs was assessed with RT-PCR. Lung inflammation was determined by differential cell count in bronchoalveolar lavage fluid. On day 21, we measured lung compliance by forced oscillation method. Emphysema was assessed by alveolar mean liner intercept (Lm) determined by computer-assisted morphometric analysis. Results: IL-17A mRNA expression was increased in WT mice lungs 6 hours after the administration of PPE. It was accompanied by neutrophilic inflammation in the lungs on day 2 – day 14, whereas neutrophil recruitment was significantly reduced in Il-17a$^{-/-}$ mice ($p < 0.05$). Lung compliance and emphysema (Lm) on day 21 in PPE-treated WT mice was significantly increased than in PBS-treated ones ($p < 0.05$). In contrast, Il-17a$^{-/-}$ mice administered with PPE showed significantly less increase in the compliance and Lm ($p < 0.05$, compared to WT). Conclusions: These results suggest that IL-17A contributes to the development of elastase-induced neutrophilic inflammation and emphysema in mice.