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Title: Alteration of the immune response to nontypeable haemophilus influenzae (NTHI) during COPD exacerbation in mice

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Body: Background: Acute episodes of bacterial exacerbations mark the progression of chronic obstructive pulmonary disorder (COPD). These exacerbations result in an increased lung inflammation. NTHI is commonly isolated bacteria during these episodes. Mechanisms responsible for the increased susceptibility to this pathogen are unknown. Objectives: Our aim is to define the alteration of the immune response to NTHI infection by using a mouse model of COPD. Methods: C57BL/6 wild type mice were chronically exposed to cigarette smoke for 12 weeks. The mice were intranasally challenged with a sub-lethal dose of NTHI (5x107cfu) and sacrificed at day 2 post-infection. Inflammation and immune responses, bacterial burden and pathological changes were evaluated. Results: An increased bacterial load was observed in infected COPD mice as compared to air control mice. There was an increase in the levels of IL-1β, IL-6, TNFα and IFNγ in the lung and BAL of infected COPD mice. IL-22 levels were decreased in the infected COPD mice. There was an increased mobilization and activation of innate cells and T lymphocytes in COPD mice. This was associated with an increased production of IFN-γ and IL-17 in the NTHI-restimulated lung cells. There was a strong inflammation in the lung with mucus secretion and remodelling in infected COPD mice. Conclusions: An increased susceptibility to H. influenzae infection was observed in COPD mice. The decrease in IL-22 production might be involved in the increased susceptibility to infection by NTHI. In contrast, the inflammatory cytokine storm and the neutrophil recruitment might trigger exacerbation-induced progression of COPD.