C4.4: Smoker Vulnerability to Infection: Using Immune Cells in a Bench Science Approach

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**Abstract:**

**Introduction:** Smoker vulnerability to infection is a complex interaction that can be difficult to study in human subjects. Therefore the purpose of this study was to investigate the immune response of airway cells in vitro when exposed to cigarette smoke and a virus. Patients with lung
disease who are exposed to smoke are more prone to infection and exacerbation. The exact mechanisms of smoker vulnerability remain unclear. Lung epithelium is the first line of defense against inhaled particulates like cigarette smoke and subepithelial dendritic cells (DCs) survey the airway epithelium linking the innate and adaptive immune response. Therefore DC may play an important role in immune modulation and smoker vulnerability to infection.

Method(s): To determine the effect of smoke on airway immune cell function, experiments were performed with epithelial and DCs from human donors. Epithelial and DCs were exposed to cigarette smoke for 12 hours, stimulated with a viral analog (poly I:C) for 2 hours and cultured with T cells. Cells and supernatants were collected and analyzed for markers of immune function, including, DC maturation, cytokine secretion and T cell proliferation. Data were analyzed with paired t-tests.

Results: DCs exposed to cigarette smoke exhibited more markers of maturation compared to controls. In addition, smoke-exposed DCs exhibited increased T helper cell type 2 (Th2-allergic) cytokines and increased interleukin 10 (immunosuppressive). Despite evidence that smoke exposed DCs were mature, there was no increase in T cell proliferation, further indicating immune suppression. This study indicates that cigarette smoke and viral stimulation increase DC maturation without inducing a proliferative immune response while potentiating an allergic environment.

Discussion & Conclusions: These findings suggest that DCs of smokers may be less likely to mount a normal immune response to a virus providing evidence for smoker susceptibility to infection. In addition, these findings are particularly important in patients with allergic airway disease since increasing Th2 cytokines would increase the risk for disease exacerbation. This may lead to future clinical studies investigating the immune response and DC phenotypes of smokers experiencing exacerbation.

Abstract History:
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