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Title: Role of dendritic cell subsets in the early stage of cigarette smoke-induced lung inflammation

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Body: To investigate the role of DC subsets in the airways in an acute model of cigarette smoke exposure (CSE) to mice, animals were treated with expansion and/or depletion of DC subsets. CSE significantly increased the number of neutrophils and macrophages in the BAL-fluid and in the lung tissue. In the BAL fluid, CSE significantly increased KC, MIP-1α and IL-6, -1β, -17, -12, -10 levels. Modulation of DC subsets in the smoke exposed group decreased the number of inflammatory cells in the BAL fluid, with a significant reduction in the number of neutrophils and macrophages. In contrast, in the smoke exposed group, treatments significantly enhanced the number of macrophages and neutrophils in lung tissue. To find a clue behind these contrasting findings, we did an attempt to link the production of cytokines in the BAL fluid and CD11c+ CD11bhi positive cells (as a marker for leukocyte adhesion and migration) with the number of cells in BAL and lung tissue. No association could be found between the levels of KC, MIP-1α and IL-6, -1β, -12 and the number of BAL cells. However, interesting was the observation that the decrease in BAL cells with DCs modulation could be linked to a additional increase in IL-10 levels and an additional decrease in IL-17 levels in smoke exposed animals. The enhancement in neutrophils and macrophages in lung tissue of smoke exposed animals after DC subsets treatments could be linked to an additional increase in CD11c+ CD11bhi positive cells. This study shows that DC's may compartment-dependent influence the degree of inflammation. Shifts in subsets of DCs are probably not very important in changing the number of cells per compartment.