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Title: Chronic pseudomonas aeruginosa airway infection induces peribronchial lymphoid neogenesis in mice

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Body: Background: Lymphoid follicles are absent in normal lungs, but are described in lungs of subjects with severe COPD or bronchiectasis. A role of bacterial infection in the development of lymphoid follicles (lymphoid neogenesis) might be hypothesized. Aims: To examine if chronic P. aeruginosa (PA) airway infection could lead to lung lymphoid neogenesis. Methods: C57BL/6 mice were instilled intratracheally with PAO1 (5.10^5 CFU/mouse)-coated agarose beads, which produces chronic airway infection (Martin et al, Eur. Resp. J. 2011 38:939). Mice with no instillation (Baseline) or instilled with sterile beads (no bacteria) served as Controls. Mice were sacrificed at day (d) 1, d4, d7, d14 and d28 after instillation. Lung sections were immunostained for B (CD20+) and T (CD3+) lymphocytes and for high endothelial venules (PNAd+), and analyzed using quantitative morphometry. Results: At Baseline, mouse lung contained no lymphoid aggregate. Instillation of PAO1-coated beads induced CD20+ peribronchial lymphocytes recruitment at d1. At d4 and d7, CD20+ positive lymphocytes formed aggregates in bronchi where PAO1-coated beads and surrounding pulmonary infection were present. At d14 and d28, lymphoid aggregates contained areas of CD20+ lymphocytes, separate areas of CD3+ lymphocytes and PNAd+ structures. Lymphoid aggregates were absent in morphologically normal areas in the lung of PAO1-infected mice and in the lung of mice instilled with sterile beads. Conclusion: Chronic P. aeruginosa airway infection induces peribronchial lymphoid neogenesis in mouse lung. The role of airway epithelium in chemokine-mediated lymphocyte recruitment requires further studies.