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Title: Identification of helicobacter pylori VacA in human lung and its effects on airway epithelial cells

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Body: [Background] Association of gastro-esophageal reflux and microaspiration with various respiratory diseases has been reported. Chronic infection with Helicobacter pylori (H. pylori) is major cause of gastroduodenal disease. We hypothesized that vacuolating cytotoxin, VacA, produced by H. pylori could be aspirated into the lung through gastro-esophageal reflux and stimulate the secretion of inflammatory cytokines by lung epithelial cells. [Methods] Immunohistochemistry of VacA was performed with lung tissue from 38 patients with interstitial pneumonia who underwent surgical lung biopsy. Pepsin levels in the bronchoalveolar lavage fluid were measured in the same patients by enzyme-linked immunosorbent assay (ELISA). Airway epithelial A549 cells were stimulated with different concentrations of VacA. The transcription and secretion of IL-8 by A549 cells was measured by real-time PCR and ELISA, respectively. [Results] VacA was detected in the bronchial epithelial cells and in the mucus in the bronchioles in 4 out of 38 patients (10.5 %) by immunohistochemistry. The pepsin levels in the bronchoalveolar lavage fluid were significantly higher in the VacA-positive group than in the VacA-negative group. Incubation with 120 nM VacA for 24 hours resulted in vacuolation of A549 cells, and significantly induced mRNA expression and protein production of IL-8 in a dose-dependent manner. [Conclusion] We demonstrated here that VacA produced by H. pylori was present in the lung and induced IL-8 production in airway epithelial cells. VacA could play a part in respiratory diseases by inducing the secretion of IL-8 by airway epithelial cells.