Title	Pseudomonas aeruginosa pyocyanin reduces cytokine levels in respiratory cell culture
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PSEUDOMONAS AERUGINOSA PYOCYANIN REDUCES CYTOKINE LEVELS IN RESPIRATORY CELL CULTURE

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Pseudomonas aeruginosa (PA) is the most difficult and versatile pathogen which chronically infects the airways of patients with bronchiectasis and cystic fibrosis. Once it is acquired, it is virtually impossible to eradicate despite intensive antibiotic treatment. PA probably exerts its effects via production of numerous exotoxins some of which have been found to cause mucosal damage. Pyocyanin, a blue phenazine pigment produced by PA has been shown to slow ciliary beating and causes ultrastructural damage to respiratory mucosa. However, the effects of PA exotoxins in the production of pro-inflammatory cytokines, an essential pathogenic component in bronchiectasis, has never been investigated. We have therefore studied this using human respiratory epithelial cells obtained from brushing the inferior turbinates of healthy subjects. We exposed these cells, suspended in medium 199, to purified pyocyanin (courtesy of Professor G Taylor of the Royal Postgraduate Medical School, Imperial College, UK) at a concentration of 20 μg/ml for 24h at 37°C in a humidified atmosphere containing 5% CO_2. Supernatant of the medium 199 was obtained and stored at -70°C before analysis of tumour necrosis factor (TNF) and interleukin(IL)-8 levels using ELISA techniques (n=7). After incubation, the mean (±SD) supernatant levels of TNF in medium containing pyocyanin was 23.5±5.7 pg/ml which was significantly lower than that in control (30.9±16.7; $p<0.05$). Similarly, IL-8 level was also significantly lower in the presence of pyocyanin (2731.6±2037.1 pg/ml) compared with control (7724.8±7304.1; $p<0.05$). We conclude that pyocyanin might play a significance role in the persistence of PA in the bronchiectatic airway by reducing the production of pro-inflammatory cytokines leading to less inflammation in the lungs of these patients. PA is therefore allowed to persist as a less inflammatory agent and our in vitro findings could help explain the PA persistence in the bronchiectatic airways.

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SERO-PREVALENCE OF HELICOBACTER PYLORI CAG A IN BRONCHICTASIS

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Bronchiectasis is a common respiratory disease in Hong Kong which accounts for significant morbidity. We have previously shown that HP IgG correlates with sputum volume production in bronchiectasis (*AJRCCM* 1998;158:723-7). As Cag A is a virulent factor for HP in the pathogenesis of peptic ulcers and gastric carcinoma, we have therefore evaluated the sero-prevalence of Cag A in Chinese patients with bronchiectasis. HP Cag A-specific IgG was measured by using ELISA techniques on subjects with steady bronchiectasis (n=100; mean age/SD 55.1 yrs/16.7; HP IgG 76% +ve) and healthy asymptomatic volunteers (94;54.6/7.6; HP IgG 54.3% +ve). Briefly ELISA plates coated with 17/12 (recombinant fragment) fusion protein was used to determined the serum level of Cag A IgG by measuring the optical density as described previously (Ching et al., 1996). In the bronchiectatic group, 24% of subjects were HP Cag A sero-positive, which was marginally significantly higher than that of the control (12.8%, $p=0.05$). Regression studies showed that HP Cag A IgG level had no correlation with FEV_1, FVC, sputum volume, presence of respiratory symptoms and upper respiratory gastrointestinal symptoms ($p>0.05$). This sharply contrasted our previous finding that HP IgG correlated with sputum volume production in the same cohort of bronchiectasis patients. Our results suggest that HP Cag A, despite a virulent factor for gastric ulcerogenesis and carcinogenesis, bears no relationship to bronchiectasis. Our findings are of clinical importance in further evaluation of the possible pathogenic role of HP in bronchiectasis.