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Airway biofilms: implications for pathogenesis and therapy of respiratory tract infections.

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Abstract

The differentiation of bacterial biofilms in the airway environment, the pathogenesis of airway biofilm, and possible therapeutic methods are discussed. Biofilm diseases that characteristically involve the respiratory system include cystic fibrosis (CF), diffuse panbronchiolitis (DPB), and bronchiectasia with *Pseudomonas aeruginosa* (*P. aeruginosa*) infection. There is evidence to suggest that almost all strains of *P. aeruginosa* have the genetic capacity to synthesize alginate, a main matrix of biofilms, when ecological conditions are unfavorable for their survival. The bacteria inside the mature biofilm show increased resistance to both antibacterials and phagocytic cells, express fewer virulence factors because of their stationary state of growth, and are less stimulatory to the mucosa because of the 'sandwich binding'. These factors facilitate both the colonization of bacteria and their extended survival even under unfavorable conditions. Since the biofilm limits colonization to a latent form, the clinical symptoms in this situation are unremarkable. However, the clinical progression of both CF and DPB proceeds in two characteristic directions. The first is an acute exacerbation caused by planktonic bacteria that have germinated from the biofilm. The second is a slow progression of disease that is induced by harmful immune reactions. The harmful reactions are mediated by alginate, which induces antigen antibody reactions around the airways, as well as formation of circulating immune complexes that are deposited on lung tissue. Furthermore, the highest titer of bacterial permeability increasing anti-neutrophil cytoplasmic autoantibodies (BPI-ANCA) is observed in association with highly impaired pulmonary function in patients with CF and DPB, as well as in patients with a lengthy period of colonization with *P. aeruginosa*. BPI-ANCA subsequently makes chronic airway infection even more intractable. The long-term use of 14- or 15-ring membered macrolides results in a favorable clinical outcome for patients with DPB and in some patients with CF. In the last 10 years, an increasing number of studies have reported secondary actions of macrolides that include effects on both airway and phagocytic cells, as well as an anti-biofilm activity. The 14- or 15-ring membered macrolides inhibit: (i) the alginate production from *P. aeruginosa*; (ii) the antibody reaction to alginate, which leads to a decrease in the immune complex formation; and (iii) the activation of the autoinducer

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