

Abstract

Cystic fibrosis is an autosomal recessive disease caused by mutation in CFTR gene coding for a chloride channel in apical membrane of epithelial cells. This disorder leads to the change in ion transport causing the increase in mucus viscosity in airways as well as changes in glycosylation of saccharide structures on the cells. Because of that these cells are the target for bacterial adhesion. Chronic bacterial infections, which lead to gradual decline of lung function and damage of lung tissue, are the major cause of death of patients suffering with cystic fibrosis.

Pseudomonas aeruginosa is the main pathogen causing chronic infections in cystic fibrosis patients. This bacterium produces a biofilm protecting them from host immune system and antibiotics. Once the colonization with PA occurs, it is difficult to get rid of this pathogen. The prophylactic treatment with orally administered hen antibodies against the PA virulence structures could be a prevention of chronic PA infections.

In this work we tested the antibody against the bacterial lectin PA-IIL, which is suggested to be involved in the adhesion of the pathogen on epithelial cells. First, it was verified that the prepared antibody from egg yolks of a hen immunized with the bacterial lectin PA-IIL recognizes this antigen expressed recombinantly as well as naturally by *Pseudomonas aeruginosa*.

The influence of this antibody on the adherence of PA was studied on epithelial cells derived from a CF patient (CuFi-1) and a healthy individual (NuLi-1). The effect of antibodies on the PA adhesion was determined by means of spectrofluorimetry as ratio of fluorescence of bacterial and epithelial cells stained with fluorochromes PKH.

By using a luminescent strain of PA we demonstrated that fluorescent dying does not affect the viability of the bacterial cells and their adherence.

Reproducibly we found that the specific chicken anti-PA-IIL antibody effectively reduces adhesion of bacterial cells to lung epithelial cells up to 67% in both lung cell lines compared to PBS treated controls. This trend, which is concentration dependent, was observed for all bacterial strains used.

On the other hand, control antibody increases the binding of bacteria on lung cells. This may be caused by an interaction between bacterial lectins and antibody saccharides. Because of the dimeric structure of the antibody, the inter-linking of bacterial cells via the antibody can occur. The removal of resulting bacterial aggregates is limited during the lung cells washing.

Furthermore, we studied the influence of the recombinantly prepared PA-IIL lectin on the bacterial adhesion. Pre-incubation of lung cells with lectin PA-IIL did not affect the binding of the bacterial cells.

The significant reduction in amounts of bound bacteria in the presence of specific antibodies anti-PA-IIL suggests that hen antibodies would be a suitable tool of preventing bacterial infections caused by *Pseudomonas aeruginosa* in patients with cystic fibrosis.

Key words: cell line, yolk antibody, luminescence of bacteria, fluorescence labelling