Abstract

Recessive autosomal disease cystic fibrosis (CF) is caused by a mutation in the CFTR gene ("regulator of cystic fibrosis transmembrane conductance"), which encodes the same named chloride channel. This mutation leads to incorrect ion transport, which causes the formation of an excessively viscous mucus on the surface of the airways and subsequently to the susceptibility to bacterial diseases. This disease mainly affects the respiratory system, where infections are associated with various causes of death in patients with CF. The most common pathogen causing infections is *Pseudomonas aeruginosa* (PA), which uses many virulence factors, such as pili or adhesins. Lectin PA-IIL, from the group of PA adhesins, is characterized by a high affinity for L-fucose, so it contributes to the adhesion of PA to the low sialylated epithelium of CF patients.

In this work the interactions between PA-IIL and lung epithelium were investigated. The cell lines CuFi-1 (CF patient) and NuLi-1 (healthy individual), which were examined *ex vivo*, were used. A part of these cell lines were exposed to neuraminidase. The PA-IIL lectin was isolated from the *E. coli* cell line *pET25_PAIIL* and subsequently fluorescently labeled with DyLight 488. The activity of mentioned lectin was verified by red blood cell agglutination. The labeling procedure did not alter the binding affinity of the lectin-saccharide. A spectrofluorimeter (Tecan Infinite M200 Pro) and a FACS fluorescence flow cytometer (BD LSR II) were used to investigate the interaction between lectin and cell lines.

Lectin PA-IIL has been shown to bind more likely to a neuraminidase-treated cell lines. These results confirm that the lectin PA-IIL contributes to the adhesion of *Pseudomonas aeruginosa* to the lung epithelium with reduced sialylation.

Key words: cystic fibrosis, *Pseudomonas aeruginosa*, PA-IIL, cell lines