

Pseudomonas aeruginosa Infection and Glucose Homeostasis in Pulmonary Cystic Fibrosis

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Glucose can cross from blood to airway surface liquid (ASL) via transcellular and paracellular routes. When transcellular glucose transport occurs, intracellular glucose concentration that determine glucose metabolism usually exceed that of ASL glucose concentration. In normoglycemia, upon entering the cell, glucose is phosphorylated by hexokinase to glucose-6-phosphate and keeps glucose concentration low in the cell, thus, uptaking glucose is promoted over efflux into ASL. Nevertheless, this mechanism in hyperglycemia is yet unknown. To reduce the risk of respiratory tract infections, maintaining low glucose concentrations in ASL at around 0.4 mM in normoglycemic stage is necessary. During hyperglycemia, glucose concentrations in ASL can increase to 4 - 15 mM.

In pulmonary cystic fibrosis (CF), there is a lack of functional cystic fibrosis transmembrane conductance regulator (CFTR) that impairs respiratory bicarbonate and fluid secretion and produces an acidic, viscous ASL, easily colonized by *Pseudomonas aeruginosa*. These conditions contribute to defective anti-bacterial properties of the CF ASL and much changes in host-pathogen responses across the CF respiratory epithelium. There is relatively underreporting on elevated ASL glucose concentrations among CF-related diabetic (CFRD) patients. CFRD patients have an increasing risk of multiple antimicrobial-resistant *Pseudomonas aeruginosa* infection and more pulmonary exacerbations, compared to those without diabetes. In CF patients, there is elevation of ASL glucose concentrations to about 2 mM, compared to about 0.4 mM in normoglycemic persons. In CFRD patients, there is further elevation of ASL glucose concentrations to around 4 mM. A previous study on comparing the mechanisms influencing respiratory tract glucose homeostasis in CF and non-CF primary human bronchial epithelial monolayers, under normal conditions and in the presence of *Pseudomonas aeruginosa* filtrate demonstrated that elevation of basolateral glucose concentrations could increase *Pseudomonas aeruginosa* growth any CFTR-dependent effects. These study results indicate the significance of glucose concentration in influencing the promotion of the *Pseudomonas aeruginosa* growth, including other airway infections in patients with pulmonary CF.

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