Epithelial signatures in respiratory disease

Throughout the entire human airways, the surface is covered by epithelial cells, which constitute the airway epithelium. Similar to the skin, the airway epithelium forms a tight barrier to protect us against potential harm from inhaled agents such as allergens, viruses, and pollutants. Additionally, the airway epithelium produces mucus, trapping viruses and other particles, which is then transported up and out by motile cilia. Furthermore, the airway epithelium communicates with other cell types, including cells of the immune system, by mediators such as cytokines and chemokines.

In the respiratory diseases asthma and COPD (chronic obstructive pulmonary disease), the airway epithelium is affected and its functionality is altered. This can be due partially to the ongoing inflammation in these diseases. However, the type of inflammation may differ between patients, which may be one reason why not all patients respond to the currently available treatments. For this reason, there is a need to increase the knowledge about processes and mechanisms related to the different types of airway inflammation, which will enable development of new improved treatments and biomarkers.

In this thesis, a primary cell-based model was used to study how the airway epithelium responds when stimulated with inflammatory cytokines that represent types of inflammation that may be found in asthma and COPD. Three types of epithelial signatures were established in response to the cytokines: gene expression, the extracellular vesicle proteome, and miRNA expression. These signatures indicated markers and processes specific for the different types of inflammation, as exemplified in the papers within this thesis. The results increase the current knowledge of disease-driving mechanisms that may ultimately lead to novel treatments and improve the lives of patients with respiratory disease.

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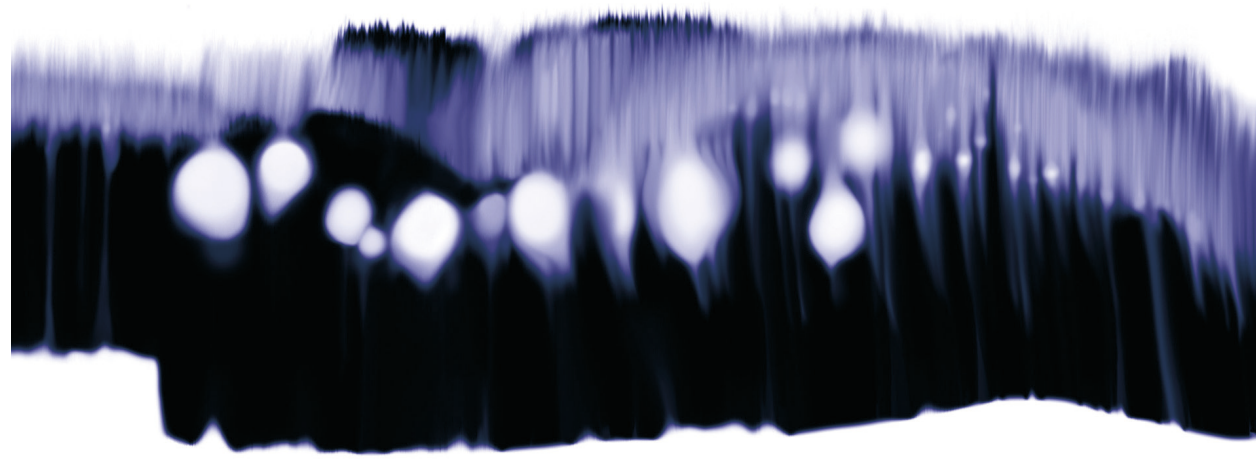

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