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Mechanism of Bronchial Epithelial Cell Response to Antigens and Allergens

Leo Danyee*

Department of Public Health, Madda Walabu University, Robe, Ethiopia

Opinion Article

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***For Correspondence:**

Dr. Leo Danyee, Department of Public Health, Madda Walabu University, Robe, Ethiopia

E-mail: leodanye@gmail.com

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ABOUT THE STUDY

The bronchial epithelium is a complex structure involving goblet, ciliated and essential cells. In endobronchial dissections, airway epithelium appears as fragile halfway or fully scaled areas. There are increased epithelial cells in the broncho-alveolar lavage of asthmatic commodities, which reflects the capability for epithelial desquamation in the airway lumen. Some studies suggested that these studies epithelial derangement could be attributed to a sampling artefact, but current data suggest that there's increased epithelial fragility. This loss of mechanical and biochemical dynamic barrier can lead to posterior submucosal cellular activation, pertained to as an abnormal epithelial mesenchymal unit.

The mechanisms upholding the fragility of epithelium in asthma are still a matter of debate. Plasma exudation may ease the detachment of epithelium from the submucosa. A direct effect of pro-inflammatory intercessors similar as metalo-proteinases or tumour necrosis factor- Alpha can induce cell death by necrosis. Epithelial damage may lead to increased airway responsiveness by the reduction of relaxant factors and loss of enzymes degrading pro-inflammatory neuropeptides. The integrity of airway epithelium may impact the perceptivity of the airways to provoke stimulants by liberating a variety of broncho-active intercessors similar as lipoxygenase and cyclo-oxygenase deduced products.

Epithelial cells are recognized as crucial players of the inflammatory process by producing pro-inflammatory products, including cytokines and proteases and expressing various adhesion molecules. Different allergens, pollutants and microorganisms can spark epithelial cells. Epithelial cells can induce inflammatory signals that are suitable to induce structural and inflammatory cells. These signals can also increase leukocyte losses from the blood and allow longer survival of inflammatory cells within the bronchi by activating cell apoptosis.

The bronchial epithelial cells can immortalize activation, as shown by the overexpression of transcription factors similar as nuclear factor kappa B, which leads to a constant state of inflammation within the bronchial structures.

Both repair and inflammatory processes are present in the asthmatic bronchial epithelium. The number of goblet cells in the airway epithelium is increased in asthma. Hyper-secretion is a common endoscopic finding in asthmatic airways related to the over production of mucus. On the other hand, mucus clearance is altered, contributing to an excessive mucus accumulation, implicated in airway obstruction. This can be due in part to a diminution in ciliated cells or a different secretory phenotype with increased goblet cells and or decreased cilia viability. Mucins and many other products are dysregulated and contribute to the phenotypic modifications of the asthmatic bronchi. Infact, goblet cells hyperplasia would be the consequence of epithelial cells activation *via* the up-regulation of mucin genes. Various cytokines derived from the epithelium such as Il-4 and Il-3 can contribute to goblet cell hyperplasia as shown in animal and cellular models.

The epithelial cells release different mediators involved in regeneration, proliferation and differentiation. Activated epithelial cells are involved in every stage of the inflammatory reaction by the release of mediators including lipid derived pro-inflammatory molecules such as leukotrienes, prostaglandins and cytokines. Epithelial cells are also involved in the recruitment and activation of leukocytes release of extracellular matrix components and growth factors, and expression of adhesion molecules contributing to cell to cell interactions. The epithelial cells are at tge interface between airspace and internal milieu. They act as physical and biochemical barriers to ensure the best transition between compartments. Many stimuli activate epithelial cells including noxious agents, infections, allergens in atopic patients, air pollutants such as diesel particulates or ozone and cigaretter smoke. Once activated these cells produce many mediators leading to adaptive response which is important for neutralization, elimination and would healing.