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A Brief Note on Epithelial Cells and Adhesion Molecules

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DESCRIPTION

Asthma is a complicated inflammatory condition of the airways that involves the interaction of multiple distinct cell types and several different mechanisms are implicated. However one mechanism, which has been receiving a lot of attention, is the attraction and retention of inflammatory cells at the site of inflammation. Understanding the processes by which these cells interact has become an interesting area of constantly expanding worldwide scientific interest and is thought to include adhesion molecules. The immunoglobulin superfamily, integrins, selectins, and cadherins are the four primary families of adhesion molecules.

These compounds are essential in the displacement of leucocytes through the endothelium wall, which is a prerequisite for cell buildup at the site of inflammation. The first stage is regulated by selectins, which stimulate the rotation of leucocytes along the endothelium wall, whereas subsequent stages, such as sticking and diapedesis, are controlled by integrins and members of the immunoglobulin superfamily. Adhesion molecules. On the other hand play a crucial function in the retention of leucocytes at the site of inflammation. It is now obvious that bronchial epithelial cells, which were previously regarded to be just a barrier preventing harmful chemicals from entering the lung, play an active part in the inflammatory response.

These cells can interact in the cellular network by synthesizing and releasing a variety of mediators such as eicosanoids and cytokines. A growing body of evidence suggests that bronchial epithelial cells can play an essential role in asthma by attracting and retaining inflammatory cells in the airways through adhesion molecules. The causes underlying these variances are unknown although the diverse approaches utilized are unlikely to account for the variations in results. In addition, inflammatory cells invading the airways may be able to influence the expression of these adhesion molecules. In asthmatics, eosinophils have been found infiltrating the bronchial mucosa and becoming activated. Furthermore, adding eosinophil-derived proteins to human nasal epithelial cells, such as major basic protein and

eosinophil cationic protein, increases ICAM-I expression considerably. The mast cell is another type of cell that can be seen between epithelial cells, especially in asthma. These cells appear to be somewhat degranulated, and it has been discovered that bronchial epithelial cells have histamine receptors. It is though unknown what function ICAM-I expression plays in asthma.

While elevated expression of this marker is believed to be involved in the cellular infiltration that characterizes this illness, particularly by eosinophils, ICAM- might also have a role in viral infections. ICAM- has been shown to be the principal surface receptor virus for rhinovirus and it is thought that a viral infection is implicated in around half of all children asthma attacks. Because Haemophilus influenza endotoxin and Para influenza virus type 2 have been demonstrated to increase the expression of ICAM-I on human airway epithelial cells, it's probable that a positive feedback network is involved in viral attachment to epithelial cells.

Increased expression of adhesion molecules on epithelial cells may potentially play a role in inflammatory cells' retention in the airways. As a result of contacts between inflammatory cells and extracellular matrix or epithelial cells, adhesion molecules are important not only in the extravasation of inflammatory cells at the endothelium level but also in the persistence of these cells at the inflammatory site. Anti-adhesion therapy may potentially find a role in the treatment of asthma if ICAM-I expression is interpreted as an indicator of inflammation in asthma, it has been proven that anti-ICAM- antibodies prevent neutrophil adherence to human airway epithelial cells. Anti-histamines such as pyrilamine and ranitidine, as well as loratadine and nedocromil sodium inhibit the histamine-induced expression of ICAM-I on bronchial epithelial cells, according to certain studies, although more information on the relevance of these studies in asthma is needed.