The Role of Apical Junctional Proteins in Epithelial Repair in Asthmatic Patients

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Abstract

In asthma, part of the abnormal response of the airway epithelium plays an important role in orchestrating the inflammatory response following interactions with multiple environmental factors (1).

Although asthma is an inflammatory disorder of the conducting airways involving T-helper type 2 cells, there is increasing evidence that the airway epithelium also plays an important role in orchestrating the inflammatory response following interactions with multiple environmental factors (1).

Hypothesis

In normal airway epithelium the apical junction complex (AJC) forms a barrier against the external environment which includes air pollutants, microorganisms and particulate matter.

In asthma, part of the dermal response of the airway epithelium is due to impaired barrier function caused by primary disruption of epithelial AJCs. This disrupted epithelial function allows basal substances to pass more easily into the airway lumen with mucin and inflammatory cells, which may also account for asthmatic susceptibility to air pollution and respiratory virus infection (2).

Methods

In vitro: RSV infection decreases TEpR in asthmatic airway epithelial cells whereas normal donors not suitable for transplantation and donated for mechanical damage.

Conclusions

Airway epithelial cell layers (AEC) were cultured on transwell systems and exposed to RSV infection. AEC monolayers were studied on Transwell (53 kΩ) or in culture with vehicle control and an intracellular pH probe (pA5). The study was performed in Edison, NJ. From the transwell system, AEC were stained for markers of the respiratory epithelium and then analysed by immunoblot or immunohistochemistry.

References