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PGE_2-mediated repair of airway epithelium

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Keywords

Bronchial asthma, prostaglandins, wound closure

Context

Following inflammatory and physical injury, the airway epithelium needs to be repaired for normal lung function to be restored. Epithelial regeneration involves spreading and migration of cells at the wound edge into the denuded surface to repopulate the wound site. Several factors, such as growth factors, extracellular matrix components and eicosanoids, seem to participate in this airway repair process. Prostaglandins are known to participate in wound healing processes in tissues such as skin, corneal epithelium, and intestinal epithelium. In view of this, the possible role of prostaglandins in regulation of airway epithelium wound closure was examined in this study.

Significant findings

In this *in vitro* study, the authors found that prostaglandin E_2 (PGE_2) stimulated wound closure in a dose-dependent fashion. Inhibitors of both cyclo-oxygenase (COX) 1 and 2 which convert arachidonic acid to prostanoids, reduced the rate of wound closure. PGE_2 receptors are called E prostanoids 1-4 (EP1-EP4). Using agonists and antagonists, it was observed that PGE_2 mediated its stimulatory action on wound healing through EP1 and EP4 but not through the EP3 receptor. Time course studies showed that prostaglandins are critical in the early stages of repair. Metabolites of 5-lipoxygenase did not seem to participate in this wound healing process. This study suggests that $PGE_2$is important for airway epithelial wound healing, and assumes significance in light of the known actions of arachidonic acid and its metabolites in cell protection, growth, angiogenesis and extracellular matrix production.

Comments

This study suggests that PGE_2 plays a critical role in wound closure in airway epithelium. Nonsteroidal anti-inflammatory drugs (NSAIDs) are frequently used by patients with asthma and other respiratory ailments, and these drugs are known to produce gastrointestinal mucosal damage. From the results of this study, it is clear that NSAIDs may also interfere with airway wound healing, since NSAIDs inhibit COX. Their use, therefore, needs to be monitored in patients with respiratory conditions. It remains to be seen whether NSAIDs interfere with wound healing induced by growth factors and how various growth factors and prostaglandins interact. More importantly, could inhalation of prostaglandins be used to enhance wound closure in the airway epithelium?

Methods

Human and feline primary tracheal and bronchial epithelial cells, immunoblotting for COX enzymes

Additional information

References

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