Erythromycin inhibits Cl secretion

We read with interest the report of erythromycin reducing chloride secretion in canine tracheal epithelium [1]. However, we would like to caution against extrapolating from dog to man. Canine trachea is virtually unique in its ion transport properties predominantly secreting chloride, a process which is stimulated by endogenous prostaglandin synthesis. The short-circuit current of human airways principally relates to absorption of sodium and chloride secretion represents only a small component of this current [2]. Furthermore, it is presently unknown whether endogenous prostaglandins have any role in influencing these processes. Thus, similar studies in a sodium absorbing epithelium will be needed to assess the likelihood of the hypothesis that erythromycin alters ion and hence water transport in man. Finally, at present there is no direct evidence that we know of which demonstrates that altering airway ion transport affects the depth of the sol phase of airway surface fluid and the rheological properties of mucus to alter mucociliary clearance. Such studies with amiloride [3, 4] are suggestive that there may be such effects, but these tenets have yet to be rigorously explored.

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References

REPLY TO THE LETTER

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We would like to thank Drs. Alton and Geddes for their comments. As the reference indicated, Cl secretion in human airways represents only a small component compared with canine airways. However, with regard to the pathogenesis of cystic fibrosis, this issue does not necessarily negate the physiological importance of human tracheal Cl channel functions. Although there is no direct evidence for the exact relation between ion transport and mucociliary clearance, this has been believed for many years that Cl secretion and the resulting water secretion may, in theory, hydrate airway mucus and hence alter mucociliary transport. To extrapolate our data to human more conclusively, evidence that Cl and water secretions are abnormally elevated in patients with airway inflammation would be needed.

It is uncertain whether endogenous prostaglandins play a role in ion transport processes in human airways, but indirect evidence may support this possibility; 1) human tracheal epithelium spontaneously produces PGE2 and PGF2α [2]; 2) this production can be stimulated by inflammatory mediators such as bradykinin and platelet-activating factor [3]; 3) both PGE2 and PGF2α increase short-circuit current [4]; and 4) the bradykinin-induced increase in short-circuit current is inhibited by indomethacin [2].

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References