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# Identification of pendrin as a common mediator for mucus production in bronchial asthma and chronic obstructive pulmonary disease.

Nakao I, Kanaji S, Ohta S, Matsushita H, Arima K, Yuyama N, Yamaya M, Nakayama K, Kubo H, Watanabe M, Sagara H, Sugiyama K, Tanaka H, Toda S, Hayashi H, Inoue H, Hoshino T, Shiraki A, Inoue M, Suzuki K, Aizawa H, Okinami S, Nagai H, Hasegawa M, Fukuda T, Green ED, Izuhara K *J Immunol* 2008 May 1 **180**(9):6262-9 [abstract on PubMed] [citations on Google Scholar] [related articles] [order article]

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Richard L. Stevens Brigham and Women's Hospital, United States of America IMMUNOLOGY

Hypothesis
New Finding
Controversial
Novel Drug Target

Nakao and coworkers report a prominent role for the iodide-channel protein pendrin/solute carrier family 26, member 4 (SLC26A4; GenBank Gene ID 5172) in the interleukin (IL)-13-dependent expression of **mucus in the lung.** Excessive production of airway mucins occurs in patients with bronchial asthma and chronic obstructive pulmonary disease (COPD), as well as in varied animal models of these disorders. Because it has been known for some time that IL-13 induces the expression of mucins in airway epithelial cells, the authors exposed human tracheal epithelial cells in vitro to IL-13 for 3-21 days and then carried out a microarray analysis to identify those transcripts that were most significantly altered. That hunt resulted in the identification of SLC26A4. The authors then carried out a series of complementary in vitro and in vivo studies that led them to conclude that SLC26A4 has a prominent role in airway mucus production. Mutations in the SLC26A4 gene lead to the Pendred syndrome of deafness but no one has reported a role for this iodide-channel protein in the lung. The mechanism by which pendrin regulates mucus expression was not determined in this study and no abnormal lung phenotype has been reported in SLC26A4-null mice. Nevertheless, the study identifies a potential new candidate protein in the pathogenesis of asthma and COPD; it also raises the possibility that an imbalance of iodide or another anion in airway epithelial cells is a contributing factor in excessive mucus production.

Competing interests: None declared Evaluated 29 May 2008 NEW

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