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Sulphur Dioxide Exposure Effect on Ciliated Cells and Mucins

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Perspective

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ABOUT THE STUDY

Chronic bronchitis is characterized by cough and mucus hypersecretion. Airway obstruction and airway hyper-responsiveness are also common features of this disease. Animal models have been developed which mimic many of these aspects of human chronic bronchitis. In most of the studies, animals were exposed to SO_2 gas, at concentrations ranging from 200 to 500 ppm, 2-5 hours/day, for periods of up to 6 months. Most of the published data concern effects in rats and dogs.

The effect of SO_2 gas on ciliated epithelial cells

In rats and dogs, there is sloughing of collated cells following acute exposure to SO_2. After 2-4 days of exposure, ciliated cells are lost throughout the trachea and main stem bronchi and replaced by one or two layers of small flat cells, whereas peripheral airways and alveoli appear normal. The central but not peripheral effects of SO_2 are consistent with what could be expected from its highly water-soluble nature and with studies that have shown that SO_2 is removed from inhaled air primarily in the upper respiratory tract. Hence the lower airways and alveoli are skelly to receive a much smaller dose of SO_2 than the central airways. After 6 weeks of SO_2 exposure, the epithelial layer in the trachea and bronchi begins to regenerate, it becomes thicker than normal, and some of the cilia begin to reappear. In ferrets, chronic exposure to SO_2 leads to decreases in the number of cilia per cell, and causes widening of the intercellular spaces at the base of the epithelium. This widening may account for the increased epithelial permeability that has been reported in animals exposed to SO_2.

The effect of chronic SO_2 exposure on mucus secretion

In rats after chronic SO_2 exposure, mucus is sometimes present in sufficient quantities as to be apparent either by direct visualization or in histological sections. There are also increases in the amounts of acidic and neutral mucins extracted from the lungs and trachea," and elevations in the mucus content of broncho-alveolar lavage fluid. The amount of mucus recovered increases with increasing duration of exposure and may represent an adaptive response in that it protects the regenerating epithelial cells.

Mucus hypersecretion is likely to result from both increased mucus synthesis and decreased mucus clearance. The size of the tracheal mucous glands, the abundance of mucus glycoprotein mRNA, and the number of epithelial secretory cells increases in rats exposed chronically to high concentrations of SO_2. The secretory cells also extend into more peripheral airways. Mucus hypersecretion is also observed with chronic SO_2 exposure in dogs. Tracheal mucus flow rates are reduced by approximately 50% in rats after 4 weeks of exposure to SO_2 perhaps as a result of changes in the viscoelastic properties of the mucus as well as decreases in the number of ciliated epithelial cells.

The nature of airway mucus glycoproteins changes over the course of chronic SO_2 exposure in rats, cons tent with observations in human bronchitis. Exposure to SO_2 gas increases both acidic and neutral mucus glycoproteins in rat lung extracts, but the percentage increase is much greater for the neutral (PAS staining) mucins than for the acidic (Alcian Blue) staining mucins. Similar results are obtained with chronic metabisulfite exposure. The number of PAS compared to Alcian Blue staining bronchiolar epithelial cells also increases with 4-6 weeks of SO_2 exposure. Changes in the composition of mucus are observed with chronic SO, exposure in dogs. These changes in mucus composition correlate with changes in its rheological properties. Similar results are obtained with chronic cigarette smoke exposure in dogs.

The mechanisms accounting for mucus hypersecretion in SO_2-exposed animals are not known. One possibility is that neutrophils are involved, since neutrophils are present in the airways of animals with SO,-induced bronchitis and neutrophil proteases are potent mucus secretagogues. It is also possible that SO_2-induced airway injury renders the animals more susceptible to infection. Infections are an important component of human chronic bronchitis, and endotoxin exposure alone is known to induce mucus hypersecretion. Indeed in rats, mucus hypersecretion induced by chronic SO_2 exposure is amplified by bacterial infection.