dysfunction in patients with chronic bronchitis after cessation of smoking has also been reported in a small prospective study (Camner et al. 1973b).

Thus, both patients with chronic bronchitis and healthy smokers exhibit an impaired mucociliary function. However, the magnitude of the impairment is not the same as suggested by Goodman et al. (1978), who demonstrated a greater impairment of tracheal mucociliary transport rates in smokers and nonsmokers with chronic bronchitis than in healthy smokers (Figure 2).

The consequences of airway mucociliary dysfunction have not been satisfactorily examined, but may include increased susceptibility to respiratory infections, airflow obstruction by excessive airway secretions, and increased risk of carcinogenesis resulting from prolonged contact between inhaled carcinogens and the respiratory epithelium (Matthys et al. 1983; Hilding 1957; Moersch and McDonald 1953).

Summary and Conclusions

1. Increased numbers of inflammatory cells are found in the lungs of cigarette smokers. These cells include macrophages and, probably, neutrophils, both of which can release elastase in the lung.
2. Human neutrophil elastase produces emphysema when instilled into animal lungs.
3. Alpha,-antiprotease inhibits the action of elastase, and a very small number of people with a homozygous deficiency of α1-antiprotease are at increased risk of developing emphysema. The α1-antiprotease activity has been shown to be reduced in the bronchoalveolar fluids obtained from cigarette smokers and from rats exposed to cigarette smoke.
4. The protease-antiprotease hypothesis suggests that emphysema results when there is excess elastase activity as the result of increased concentrations of inflammatory cells in the lung and of decreased levels of α1-antiprotease secondary to oxidation by cigarette smoke.
5. Cigarette smokers have been shown to have a more rapid fall in antibody levels following immunization for influenza than nonsmokers. Whole cigarette smoke has been shown to depress the number of antibody-forming cells in the spleens of experimental animals.
6. Cigarette smoke produces structural and functional abnormalities in the airway mucociliary system.
7. Short-term exposure to cigarette smoke causes ciliostasis in vitro, but has inconsistent effects on mucociliary function in man. Long-term exposure to cigarette smoke consistently
causes an impairment of mucociliary clearance. This impairment is associated with epithelial lesions, mucus hypersecretion, and ciliary dysfunction.

8. Chronic bronchitis in smokers and ex-smokers is characterized by an impairment of mucociliary clearance.

9. Both the particulate phase and the gas phase of cigarette smoke are ciliotoxic.
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304


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314


315


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316


318


319


323


