

13). Camner, et al. (BP 8) reported on clearance rates in 17 young and middle-aged ex-smokers who had stopped smoking for 3 months. These workers noted that mucociliary clearance of 6 μ m. fluorinated ethylene propylene (Teflon 120) particles tagged with Tc^{99m} . measured at 2 hours post-inhalation had improved at 3 months post-quitting in 11 of 17 patients. Mean retention of particles was significantly higher prior to stopping smoking than at 3 months ($P < .05$), and also was higher at 1 week post-cessation compared to 3 months post-cessation ($P = .005$). In this study, the volume of inhaled aerosol was not controlled. In addition, coughing after inhalation of the particles was reported to be conspicuously absent or rare, whereas in the Thomson study (BP 67), the effect of coughing during the study was not discussed.

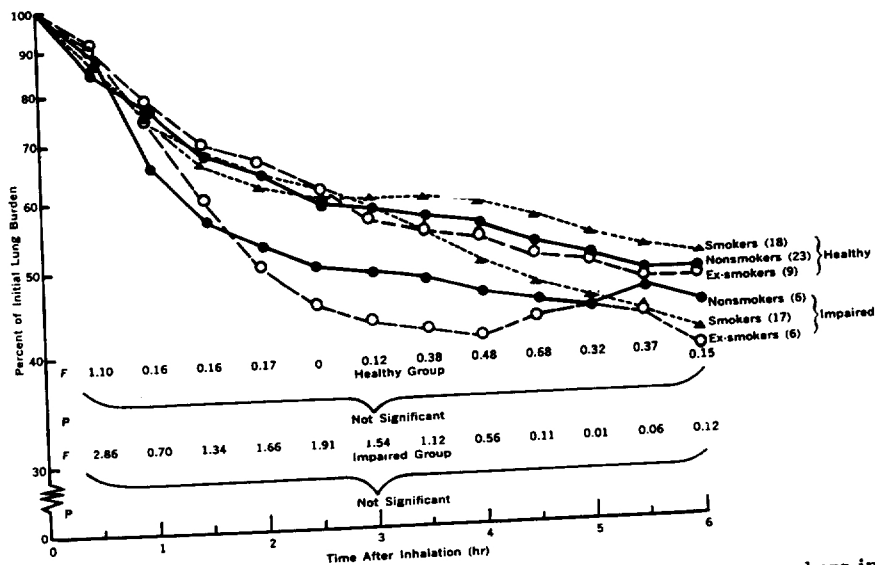


FIGURE 13.—Mean clearance curves for smokers, ex-smokers, and nonsmokers in the healthy group and the group with respiratory impairment. F=Snedecor's F; the values required for significance at the 5 percent level are 3.19 for the healthy group and 3.35 for the impaired group.

SOURCE: Thomson, M. L., Pavia, D. (BP 67).

The question of whether the short-term effect of cigarette smoking on enhancing mucociliary transport is specific to cigarette smoke, or is due to a nonspecific reaction of the tracheo-bronchial tree, was investigated by Camner, et al. (BP 7) who found that inhalation of inert carbon particles by 8 normal subjects (including 2 smokers) resulted in similar or enhanced

pulmonary clearance rates compared to the control states. These results suggest that the increased mucociliary transport effected by short-term exposure to cigarette smoke may be a nonspecific reaction.

Westergaard and Olsen (*BP 68*) studied ciliary activity in biopsy specimens from the larynx and carina in 20 patients and found that there was no ciliary activity in epithelial cells from these sites in the entire group of 16 moderate and heavy smokers, while in 3 nonsmokers and 1 cigar smoker normal ciliary activity was observed.

Studies in Animals

Binns and Clark (*BP 1*) described a new experimental model for testing the short- and long-term effects of cigarette smoke on pulmonary physiology. By using male cynomolgus monkeys which were fitted with a specially designed smoking device, these authors demonstrated marked increases in total pulmonary resistance in animals smoking approximately 12 cigarettes per day, for 5 days per week. These changes stabilized at about 20 weeks of exposure and extended through the 6-month test period. The changes in pulmonary resistance were statistically significant ($P < .001$). After 6 months, no changes in tidal volume, respiratory rate, or dynamic compliance were noted. Histologic sections of lungs from smoking monkeys showed clumping of pulmonary alveolar macrophages containing pigmented granules and foamy cytoplasm. These nonspecific cytologic changes have been observed in other animals exposed to cigarette smoke.

Previous editions of this report (1972, 1973) have described experimental evidence concerning the production of emphysematous changes in rat and guinea pig lungs by exposure to nitrogen dioxide (NO_2), one of the gaseous components of cigarette smoke. Freeman, et al. (*BP 18*) described experiments whereby low (10 to 15 p.p.m.), intermittent doses of NO_2 administered over the normal life span of rats resulted in more severe changes of the pulmonary parenchyma than those previously reported; these changes included fibroblastic proliferation, epithelial hypertrophy, loss of cilia in the respiratory bronchioles, fibrosis of alveolar ducts, destruction of alveolar walls, and enlargement of alveolar air spaces. These authors calculated a 29 percent loss of ventilatory surface in the NO_2 -exposed rats, occurring in a panlobular distribution. The lungs of the NO_2 -exposed rats had greater residual volumes than the controls, and these rats suffered from hypoxemia, hypercarbia, and acidosis, as well as a compensatory polycythemia. Thus, by administering lower doses of NO_2 intermittently, survival of these rats was prolonged (compared with survival of rats receiving continuous NO_2), and the

development of a full-blown picture of emphysema similar to that seen in humans was produced. The relative role of NO_2 in the causation of emphysema in humans is still unknown.

In another series of experiments, Giordano and Morrow (*BP 21*) studied mucociliary clearance rates in female rats exposed continuously to low doses of NO_2 (6 p.p.m.) over a period of 6 weeks. They found a significantly decreased rate of clearance in rats exposed to NO_2 than in nonexposed rats ($P < .02$). In those animals with a decrease in mucociliary activity, the effect of NO_2 was reversible within 7 days following this long-term low dose exposure.

Goldstein, et al. (*BP 22*) reported on the effects of low level NO_2 exposure on bactericidal activity of the mouse lung. They first infected mice with radioactively labelled *Staphylococcus aureus* and then exposed them to different concentrations of NO_2 for 4 hours. The authors then measured pulmonary radioactivity and bacterial concentrations. They found that at concentrations of 7, 9.2, and 14.8 p.p.m. NO_2 the level of radioactivity was unchanged, but bacterial counts were greater in the NO_2 -exposed mice ($P < .05$), and they concluded that the bactericidal activity of the NO_2 -exposed animals was significantly less than that of control animals at these concentrations of NO_2 . In a series of experiments where mice were first exposed to 1.0, 2.3, and 6.6 p.p.m. NO_2 for 17 hours and then infected with the labelled *Staphylococcus aureus*, pulmonary bactericidal activity was decreased in the mice exposed to the latter two concentrations of NO_2 ($P < .05$ and $P < .01$). In both sets of experiments, the physical removal rates of bacteria by the pulmonary tree (as measured by the degree of remaining radioactive label) was not influenced by NO_2 . These experiments suggest that the retardation of pulmonary bactericidal activity was due to dysfunction of the cellular elements of the pulmonary defense mechanism (i.e., pulmonary alveolar macrophages [PAMs]) in the NO_2 -exposed mice.

Fenters, et al. (*BP 15*) exposed four monkeys to low dose NO_2 (1 p.p.m.) for 16 months and infected these animals with influenza virus. Three control monkeys were exposed to the virus, but not to the NO_2 . The NO_2 -exposed animals had higher hemagglutination-inhibition and serum neutralizing antibody responses against the virus than the nonexposed animals. Pathologic examination of the lungs of these animals demonstrated slight to moderate emphysema in the NO_2 -exposed and virus-infected monkeys, along with thickening of the bronchial and bronchiolar epithelium, and no such changes in those animals only infected with virus.

Dalhamn (*BP 13*) conducted experiments on 40 live rats, exposing them to cigarette smoke of different chemical compositions. The cigarette smoke was analyzed for "tar", nicotine, pH, acrolein, nitrogen oxides (NO), acetaldehyde, hydrogen cyanide (HCN), and carbon monoxide (CO). The author found an inverse correlation between the number of puffs required to produce ciliostasis of the tracheobronchial tree and the amount of acrolein, HCN, CO, "tar", and nicotine found in the cigarette smoke. The data appeared to indicate that the majority of the effect was caused by the "tar" and acrolein content of the cigarette smoke.

Gairola and Aleem (*BP 19*) studied the effect of the water soluble and insoluble fractions of tobacco smoke on rat liver mitochondrial function. These investigators found that both fractions were effective in inducing a decline in energy production by the mitochondria, but to differing degrees, thereby suggesting some difference in their mechanisms of action on mitochondria.

Snider, et al. (*BP 59*) exposed rats to 0.1 percent cadmium chloride solution by aerosol, and were able to demonstrate centrilobular emphysema in these animals after 10 days. Since cadmium has been found in cigarette smoke, and smokers and patients with emphysema have been shown to have elevated tissue levels of cadmium at postmortem, further studies defining the role of cadmium in the development of pulmonary emphysema in man would be useful. In Snider's experimental protocol, animals exposed to 1 percent of CdCl₂ developed a severe hemorrhagic necrotizing chemical pneumonia, and the lower dose of CdCl₂ also elicited a hemorrhagic response, although no evidence of such an inflammatory response was evident 10 days post-exposure.

CYTOLOGIC AND HISTOLOGIC STUDIES

Experimental evidence indicates that cigarette smoke can impair the function of pulmonary alveolar macrophages (*BP 53*). Pulmonary macrophages appear to be the primary defense against bacterial invasion of the pulmonary parenchyma and also serve to remove particulate contaminants from inspired air. In recent experimental work, Powell and Green (*BP 53*) investigated the mechanism of action of cigarette smoke on macrophage function. By using histochemical staining techniques, these workers found that the filtered gas phase of cigarette smoke (FGP) inhibited aldehyde dehydrogenase activity in rabbit pul-

monary alveolar macrophages (PAMs). This effect of FGP was inhibited by prior addition of cysteine to the medium. The loss of enzyme activity correlated with the loss of macrophage phagocytic function (which was also prevented by the prior addition of cysteine). No other enzyme was inhibited by cigarette smoke (except for G6PD in those preparations of cells which adhered to glass). By using crystalline glyceraldehyde 3-phosphate dehydrogenase, the authors demonstrated that FGP inhibited activity of this enzyme, and the degree of inhibition was directly related to the period of incubation (most of the inhibition occurring within the first five minutes). This enzyme was not inhibited by FGP in the presence of cysteine. When enzyme activity was assayed in cell preparations, glyceraldehyde 3-phosphate dehydrogenase activity was reduced by FGP. This inhibition was dose-related to FGP. FGP did not influence G6PD or LDH activity in the pulmonary alveolar macrophages. These experiments suggest that FGP inhibited the glycolytic pathway within the pulmonary alveolar macrophages concurrent with the impairment of phagocytic activity of these cells. The authors postulated that FGP may act as a sulfhydryl agent (thereby explaining the protective effect of cysteine) in the disruption of the activity of glyceraldehyde 3-phosphate dehydrogenase.

York, et al. (*BP 72*) showed that incubation of sheep pulmonary macrophages with tobacco extract resulted in an initial stimulation, then inhibition of macrophage oxygen consumption. When cigarette smoke extract was incubated with the macrophages, a continuous decrease in oxygen consumption was observed, proportional to the incubation period and concentration of smoke extract (figure 14). The enzymatic mechanism of the inhibition of macrophage respiration was not examined in these experiments.

Invertase placed in a medium of calf's serum results in enhanced pinocytosis by sucrose-laden mouse peritoneal macrophages (monocytes). Schwartz, et al. (*BP 56*), utilizing this test system, reported that nicotine inhibited invertase-induced pinocytosis by sucrose-laden mouse peritoneal monocytes by 29 and 18 percent, depending on the concentration of nicotine added to the medium. The contribution of this type of pinocytosis to the bactericidal activity of these monocytes is unclear at present.

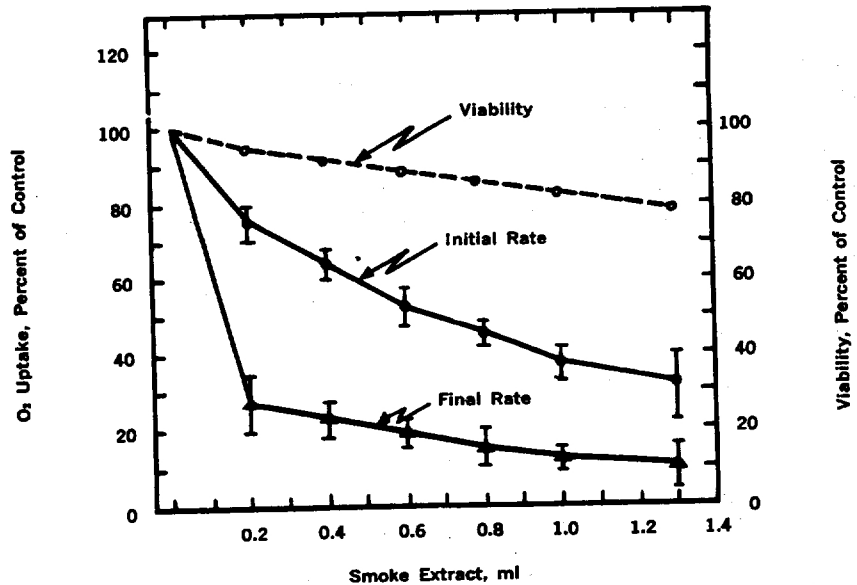


FIGURE 14.—Effects of aqueous cigarette smoke extract on initial oxygen uptake, final oxygen uptake, and cell viability of pulmonary macrophages.

SOURCE: York, G. K., et al. (*BP 72*).

SUMMARY OF RECENT NON-NEOPLASTIC BRONCHOPULMONARY FINDINGS

1. Results from epidemiologic studies on elderly populations demonstrate an increased prevalence of respiratory symptoms and impairment of pulmonary function among smokers of both sexes compared to nonsmokers.
2. Data from several recent studies indicate that standard pulmonary function tests and physical work capacity are impaired in apparently healthy smokers compared to nonsmokers.
3. Recent epidemiologic data suggest that smokers who retain their cigarettes in their mouths continuously while smoking ("droopers") have a higher prevalence of chronic bronchitis than those smokers who remove the cigarette from their mouths between puffs.
4. A recent epidemiologic study confirms the observation that cigarette smoke and air pollution act synergistically in the development of symptoms of respiratory disease.
5. Results from several recent studies indicate that cigarette smokers have a higher prevalence of functional abnormalities of the small airways than do nonsmokers.

6. Results from a recent study suggest that although a history of lower respiratory disease as an infant is related to the prevalence of cough at age 20, cigarette smoking is a far more important factor in the development of cough in young adulthood.
7. Data from a major retrospective study indicate that cigarette smoking is related to the development of bullous disease of the lung.
8. Experimental studies in animals have shown that exposure to nitrogen dioxide, a constituent of the vapor phase of cigarette smoke, results in emphysema-like changes in the pulmonary parenchyma, diminished mucociliary clearance, and impairment of bactericidal activity of alveolar macrophages.
9. Data from experimental studies have demonstrated that the filtered gas phase of tobacco smoke may effect changes in pulmonary alveolar macrophage metabolism through inhibition of the glycolytic pathway; cigarette smoke may also impair oxygen consumption and pinocytic activity of pulmonary alveolar macrophages.

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Part II

(Additional articles which were reviewed but not discussed in the text.)

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