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## Breathing other people's smoke

Air contaminated with tobacco smoke is disagreeable to many non-smokers and, in small unventilated spaces, even to smokers themselves. The smoke may irritate the eyes and throat, and precipitate an asthmatic attack in particularly susceptible individuals. But, as well as these acute effects and the annoyance smoking causes, might more long-term hazards be associated with tobacco smoke in the environment?

Cigarette smoke contains over 1000 substances; and some, such as tar, carbon monoxide, and nicotine, are found in higher concentrations in the side stream smoke (given off between puffs) than in the main stream. Recently Brunnemann et al<sup>2</sup> have shown that nitrosamines (potent carcinogens in animals though not yet linked to human cancer) are present in side stream smoke at 50 times greater concentration than in the main stream. These investigators estimated that during one hour in a smoky room a non-smoker may inhale nitrosamines in quantities equivalent to smoking about 15 filter cigarettes; and the concentration of nitrosamines in the air of an indoor room polluted by tebacco smoke may be over 80 times higher than in the home of a non-smoker.

Benz(a)pyrene has been incriminated in causing cancer in man as well as in animals, and one cigarette may yield 100 ng of the substance into the air. Carcinogens are generally thought to exert their effect in direct proportion to the level of exposure: there is no safe threshold beneath which no effect occurs. So even the small amounts of carcinogenic substances released into the air from tobacco smoke can be assumed to cause at least some cases of lung cancer. Data on exposure to benz(a)pyrene in men who made coal gas and their subsequent risk of lung cancer suggest that regular and fairly heavy exposure to environmental tobacco smoke might increase the rate of lung cancer among people who have never smoked by about one-third—about two cases of lung cancer per 100 000 per year.

The concentration of carbon monoxide in the air of a room free of tobacco smoke is typically about 2 parts per million (ppm). Tobacco smoke rarely causes concentrations over 20 ppm, which would lead to a maximum carboxyhaemoglobin. (COHb) concentration of 3%. In practice, such concentrations are usually intolerable because of the irritation of the eyes and throat from other constituents of tobacco smoke. In normal conditions there is no change in COHb concentrations among non-smokers in smoky offices and restaurants, and in deliberately extreme conditions (a concentration of carbon monoxide in an unventilated room of 38 ppm) the COHb level of the non-smokers increased from 1.6 to only 2.6%.

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There is no evidence that carbon monoxide causes arterial disease in healthy adults, nor does exposure associated with COHb concentrations below 5% produce clear-cut deterioration in mental function. A COHb concentration of 5% is rare among non-smokers, even in buses or cars, where smoking may cause episodic rises in carbon monoxide concentrations to over 50 ppm. At this level of exposure it would take five hours to produce a COHb concentration of 5%, so that drivers of public vehicles are unlikely to have their performance jeopardised by other people's smoke. Patients with coronary heart disease, however, might be at risk from the carbon monoxide in smoky atmospheres, since COHb concentrations of about 3% may exacerbate exercise-induced angina pectoris.

Investigations using nicotine as a marker of tobacco smoke absorption have shown that the non-smoker inhales extremely small quantities. For example, inhalation by a non-smoker of the air in a cocktail lounge has been estimated to be equivalent to smoking less than 1% of a cigarette. Exposure to environmental tobacco smoke causes detectable urinary excretion of nicotine, but in amounts that are less than 1% of those excreted by smokers. These small amounts of nicotine inhaled by non-smokers are unlikely to have any medical importance.

Direct epidemiological studies of the health risks of exposure to tobacco smoke by non-smokers have been made only in children. The children of parents who smoke have more infections of the upper respiratory tract than do children of non-smokers.9 10 The early results of such studies were open to the criticism that the association between parental smoking and the children's respiratory symptoms might have been due to social class factors or to the higher incidence of chest infections in the smoking parents infecting their children more frequently. A more recent study showed, however, that respiratory infections during the first year of life were closely related to parents' smoking habits in a way that was independent of parental respiratory symptoms, social class, or birth weight.11 Furthermore there was a dose-response relation between parental smoking and infant respiratory infections, The implications of these studies may extend beyond childhood, since episodes of respiratory illness in the early years of life have been associated with the development of adult chest

For the moment most—but not all—of the pressure for people (including many smokers) to have the right to breathe smoke-free air must be based on aesthetic considerations rather than on known serious risks to health. The unpleasantness for non-smokers comes when they have to come into contact with

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