Tobacco smoke in the workplace: an occupational health hazard

Neil E. Collishaw,* MA
John Kirkbride,† MD, MSc
Donald T. Wigle,‡ MD, PhD, MPH

Tobacco smoke, which contains over 50 known carcinogens and many other toxic agents, is a health hazard for nonsmokers who are regularly exposed to it while at work. Involuntary exposure to tobacco smoke annoys and irritates many healthy nonsmokers. Serious acute health effects are probably limited to the one fifth of the population with pre-existing health conditions that are aggravated by exposure to tobacco smoke. The consequences of long-term exposure include decreased lung function and lung cancer. Existing air quality standards for workplaces do not directly specify an acceptable level for tobacco smoke. The evidence on the composition of tobacco smoke and on the health hazards of involuntary exposure suggests that there may not be a "safe" level for such exposure.

La fumée du tabac, qui renferme plus de 50 substances cancérogènes connues et de nombreux autres agents toxiques, met en danger la santé des non-fumeurs qui y sont régulièrement exposés au travail. L'exposition involontaire à la fumée du tabac représente une source d'ennuis et d'irritation pour de nombreux nonfumeurs en bonne santé. Des malaises aigus graves sont vraisemblablement limités au cinquième de la population accusant des problèmes de santé préexistants que l'exposition à la fumée du tabac aggrave. Parmi les conséquences de l'exposition à long terme notons la diminution de la fonction pulmonaire et le cancer du poumon. Les normes actuelles de qualité de l'air aux lieux de travail ne s'adressent pas directement à la question d'un niveau acceptable de la fumée du tabac. Les données sur la composition de la fumée du tabac et sur les risques de l'inhalation involontaire pour la santé laissent entendre qu'il se peut qu'il n'y ait pas de niveau "sûr" d'exposition à la fumée du tabac sur les lieux de travail.

From the Department of National Health and Welfare, Ottawa

*Chief, policy analysis, Bureau of Tobacco Control, Health Protection Branch
†Director, Occupational Health Unit, Medical Services Branch
‡Chief, Non-Communicable Disease Division, Bureau of Epidemiology, Health Protection Branch

Reprint requests to: Neil E. Collishaw, Bureau of Tobacco Control, Health Protection Branch, Department of National Health and Welfare, Tunney's Pasture, Ottawa, Ont. K1A 0L2

There are standards for limiting occupational exposure to many airborne toxic substances. Threshold limit values (TLVs) are the maximum time-weighted average concentrations to which workers can be exposed in a normal 8-hour workday or a 40-hour workweek. The American Conference of Governmental Industrial Hygienists Inc. (ACGIH) has published the TLVs for over 500 toxic substances,[1] including several that are in tobacco smoke, but none for tobacco smoke per se.

The inhalation by nonsmokers of air contaminated by tobacco smoke is referred to as involuntary exposure. Such air contains the same toxic chemicals present in "mainstream smoke" (that inhaled by a smoker during a puff). However, the main source of tobacco smoke contaminants is "sidestream smoke" (that emitted by the burning tip of a cigarette, cigar or pipe), which contains much higher concentrations of many toxic and cancer-causing chemicals than does mainstream smoke[2] (Table I).

It has been estimated that 63% of the labour force in the United States is exposed to tobacco smoke in the workplace.[3] The proportion in Canada is likely to be higher because of the higher per-capita consumption of cigarettes.[4,5]

The toxic substances in tobacco-smoke-polluted air are inhaled and, to varying degrees, absorbed by exposed nonsmokers. Tobacco smoke, both sidestream and mainstream, is a concentrated aerosol of very small particles measuring less than 0.6 μm in mass median aerodynamic diameter.[6] It has been predicted that 30% to 40% of these particles will deposit in alveolar regions and 5% to 10% in the tracheobronchial region.[7] Such particles contain many known carcinogens;[2,8] they may be engulfed by macrophages or transported to regional lymph nodes and may take days to months to clear from the lungs.[9]

Composition of tobacco smoke

Tobacco smoke is a complex mixture of particles and gases that contain at least 3800 different chemical compounds,[10] over 50 of which are known to be carcinogenic in animals or humans or both. Little is known about the health effects of most of the 3800 chemicals, and almost nothing is known about their interactive effects.

Gas phase

About 90% by weight of tobacco smoke is in the gas phase. The major toxic element by weight is carbon monoxide. The visible smoke from the tip of a burning cigarette contains relatively high concentrations of toxic chemicals that become slightly diluted as the smoke drifts horizontally for distances up to 3 m. Nonsmokers who are located close

← For prescribing information see page 1303

to smokers are exposed to the irritants formaldehyde and acrolein at concentrations of up to 110 and 70 parts per million (ppm) respectively.[11]

The gas phase also contains other irritants (e.g., ammonia, nitrogen oxides and pyridine), ciliatoxic agents (e.g., hydrogen cyanide) and several potent carcinogens (e.g., *N*-nitrosodimethylamine). The toxic substances in the gas phase are not removed by standard air filtration systems.

Particulate phase

This is a highly concentrated aerosol containing about 5×10^9 particles per millilitre of mainstream smoke.[8] Particles of sidestream smoke have a smaller geometric mean diameter (0.20 μm) than those of mainstream smoke (0.36 μm).[12] Thus, a higher proportion of sidestream particles would be expected to deposit in alveolar regions.

Risks of involuntary exposure to tobacco smoke

The serious health consequences of smoking have been well known for many years and have been summarized in recent reports of the US surgeon general.[2,7,8,13] However, the effect of tobacco smoke on the health of nonsmokers is a matter of growing concern; it was reviewed in some detail in the most recent report of the US surgeon general[7] and merits closer examination here.

Physiological changes and subclinical toxic effects

Eye irritation, the most common complaint of healthy people exposed to tobacco smoke, and the rate of eye blinking increase with increasing amounts or duration of exposure to smoke.[7] After 1 hour's exposure to smoke-related carbon monoxide at concentrations as low as 1.3 ppm, well within those measured under realistic conditions, eye irritation and the rate of blinking increase significantly.[7]

Brief exposure of nonsmoking adults to high concentrations of tobacco smoke under carefully controlled conditions in exposure chambers results in a small but significant impairment of lung function, including maximal airflow at 50% and 75% of forced vital capacity, $\dot{V}max_{50}$ and $\dot{V}max_{75}$.[7]

Involuntary exposure to tobacco smoke increases the heart rate and blood pressure in patients with angina pectoris.[14]

Relatively low concentrations of carbon monoxide, similar to those in confined, smoky areas, significantly impair one's driving (e.g., by reducing braking time and delaying recovery of vision following glare).[15]

Symptoms

The symptoms reported by nonsmokers exposed to tobacco smoke include eye irritation, nasal congestion, headache, cough, sore throat, hoarseness, nausea, dizziness, "general annoyance", loss of appetite and Raynaud's phenomenon.[16–23]

Aggravation of pre-existing conditions

The results of the Canada Health Survey indicate that 21% of Canadians have a health condition that is aggravated by exposure to tobacco smoke.[24] These conditions include heart disease, acute respiratory disease, emphysema, asthma and hay fever. Persons who wear contact lenses also experience eye irritation, but the number of such persons in Canada is not known.

Table I—Concentrations of the major toxic and tumourigenic agents in cigarette smoke and their ratio in mainstream smoke (MS) and sidestream smoke (SS)[2]

Phase and agent(s)	MS level	SS/MS ratio*	Phase and agent(s)	MS level	SS/MS ratio*
Gas			**Stigmasterol**	**53 μg**	**0.8**
Carbon dioxide	**10–80 mg**	**8.1**	**Total phytosterols**	**130 μg**	**0.8**
Carbon monoxide	**0.5–26 mg**	**2.5**	**Naphthalene**	**2.8 μg**	**16**
Nitrogen oxides	**16–600 μg**	**4.7–5.8**	**1-methylnaphthalene**	**1.2 μg**	**26**
Ammonia	**10–130 μg**	**44–73**	**2-methylnaphthalene**	**1.0 μg**	**29**
Hydrogen cyanide	**280–550 μg**	**0.17–0.37**	**Phenanthrene**	**2.0–80 ng**	**2.1**
Hydrazine	**32 μg**	**3**	**Benz(*a*)anthracene**	**10–70 ng**	**2.7**
Formaldehyde	**20–90 μg**	**51**	**Pyrene**	**15–90 ng**	**1.9–3.6**
Acetone	**100–940 μg**	**2.5–3.2**	**Benzo(*a*)pyrene**	**8–40 ng**	**2.7–3.4**
Acrolein	**10–140 μg**	**12**	**Quinoline**	**1.7 μg**	**11**
Acetonitrile	**60–160 μg**	**10**	**Methylquinoline**	**6.7 μg**	**11**
Pyridine	**32 μg**	**10**	**Harmane**	**1.1–3.1 μg**	**0.7–2.7**
3-vinylpyridine	**23 μg**	**28**	**Norharmane**	**3.2–8.1 μg**	**1.4–4.3**
***N*-nitrosodimethylamine**	**4–180 ng**	**10–830**	**Aniline**	**100–1200 ng**	**30**
***N*-nitrosoethyl-methylamine**	**1.0–40 ng**	**5–12**	***o*-toluidine**	**32 ng**	**19**
			1-naphthylamine	**1.0–22 ng**	**39**
***N*-nitrosodiethylamine**	**0.1–28 ng**	**4–25**	**2-naphthylamine**	**4.3–27 ng**	**39**
***N*-nitrosopyrrolidine**	**0–110 ng**	**3–76**	**4-aminobiphenyl**	**2.4–4.6 ng**	**31**
Particulate			***N*-nitrosonornicotine**	**0.2–3.7 μg**	**1–5**
Total	**0.1–40 mg**	**1.3–1.9**	**4(methylnitrosamino)-1-(3-pyridyl)-1-butanone**	**0.12–0.44 μg**	**1–8**
Nicotine	**0.06–2.3 mg**	**2.6–3.3**	***N*-nitrosoanatabine**	**0.15–4.6 μg**	**1–7**
Toluene	**108 μg**	**5.6**	***N*-nitrosodiethanolamine**	**0–40 μg**	**1.2**
Phenol	**20–150 μg**	**2.6**	**Polonium 210**	**0.03–0.5 pc**	**NA**
Catechol	**40–280 μg**	**0.7**			

***NA = not available.**

Persons with angina have a substantially increased susceptibility to exercise-induced attacks when they are exposed to low concentrations of tobacco smoke.[14] Persons with asthma experience attacks (wheezing and difficulty in breathing) when exposed to tobacco smoke;[25,26] it is not clear whether tobacco smoke acts as an allergen, an irritant or both.

Acute illnesses

Available evidence on the role of involuntary exposure to tobacco smoke in acute respiratory tract infections is based on studies of children so exposed in their homes. Bronchitis, pneumonia and other respiratory illnesses have been found to occur more frequently among infants and children up to 2 years of age who have one or two parents who smoke.[7] Several of the studies reviewed by the US surgeon general revealed an exposure–response relation between the amount of parental smoking and the risk of respiratory illness. Young children may generally be more susceptible than adults, but it seems likely that exposure to tobacco smoke in the workplace would also increase the risk of acute respiratory illnesses in adults, particularly those with predisposing health problems, such as heart disease or allergies.

Long-term effects

A study of 2100 adults revealed impairment of small-airways function in nonsmokers who were employed for at least 20 years in enclosed work areas where smoking was permitted.[27] The loss of function was equivalent to that in persons who had smoked up to 10 cigarettes per day for at least 20 years. As well, the amount of particulate matter inhaled by nonsmokers is equivalent to that inhaled by persons who smoke up to 10 cigarettes per day and is sufficient to produce serious lung damage.[28]

A recent study of almost 8000 adults in France indicated that nonsmokers of either sex who were 40 years of age or older and whose spouses had smoked at least 10 g of tobacco (i.e., about 10 cigarettes) per day had impairment of lung function that was not explainable by social class, education, air pollution or family size.[29] The average degree of impairment was 16% for men and 6% for women. Although not related to workplace, it is interesting that impairment of small-airways function has also been observed in children whose mothers smoked.[30]

Several studies have shown an increased risk of lung cancer among nonsmokers who are married to smokers.[31–34] Indeed, the risk was up to 3.4 times that among persons not exposed to tobacco smoke. Another study revealed only a slight, statistically insignificant increase in the risk of lung cancer among women who did not smoke but were married to men who did.[35] However, this finding is weakened by the fact that marital status and the spouse's smoking habits are a poor index of exposure to tobacco smoke for American women since many of them work outside the home.[36] Repace[37] noted that the failure to adjust for exposure to tobacco smoke in the workplace probably resulted in an underestimate of the effect of involuntary exposure in the home.

Hirayama[38] reported standardized mortality ratios for nasal sinus cancer among women who did not smoke of 1.0, 2.3, 2.6 and 3.3 when their husbands were nonsmokers or smokers of 1 to 14, 15 to 19, or 20 or more cigarettes daily respectively.

Interaction between involuntary and occupational exposures

This topic was critically reviewed in the 1979 report of the US surgeon general under the general heading of smoking and occupational exposures.[8] The report noted that certain toxic agents in tobacco smoke may also be present in the workplace, thus increasing the likelihood of exposure to the agent. For example, hydrogen cyanide is present in cigarette smoke at concentrations of up to 1600 ppm. Other examples include carbon monoxide, methylene chloride, acrolein, arsenic, formaldehyde and polycyclic components. Given that the risk of lung cancer is greatly increased among workers who smoke and are exposed to asbestos,[39] it is highly probable that exposure to air contaminated by both tobacco smoke and asbestos is a greater hazard than exposure to asbestos alone for nonsmokers. This inference is based on the fact that all of the chemicals in mainstream smoke are also in sidestream smoke; indeed, the concentrations are often much higher in the latter.

Estimates of exposure to tobacco smoke among nonsmokers

Biologic indicators

The chemicals present in tobacco smoke, or their metabolites, have been repeatedly detected in samples of blood, urine, saliva and breast milk from nonsmokers.[40–46] Russell and Feyerabend[41] concluded that, as a result of involuntary exposure to tobacco smoke, most nonsmokers in urban areas have measurable amounts of nicotine in their body fluids for most of their lives. In one study nicotine levels were measured in saliva and urine samples from hospital employees after a morning at work.[42] The nonsmokers who had been exposed to tobacco smoke had significantly higher nicotine concentrations than those who had not been exposed. Mutagenic activity has been detected in cigarette-smoke-contaminated air and in urine samples from nonsmokers exposed to such air.[45] In another study trace amounts of nicotine and cotinine (formed in the body from nicotine) were detected in samples of breast milk from three nonsmokers who were exposed to tobacco smoke at work but not at home;[46] the concentration of these substances fell to unmeasurable levels on weekends.

Levels of tobacco smoke in ambient air

Using data from American time use and smoking surveys, Repace and Lowrey[3] estimated the proportion of nonsmokers who are likely to be chronically exposed to tobacco smoke at work or at home, or both, to be 86% (Table II). In the United States it was estimated that, in 1980, smokers constituted 34% of the adult population and smoked an average of 22 cigarettes per day.[13] In Canada in 1981 both the proportion of smokers and the average number

of cigarettes smoked daily were higher (39% and 27 respectively).[4] Canada has the highest rate of use of manufactured cigarettes among industrialized nations.[5] Moreover, it is likely that Canadians, who generally face more severe climatic conditions than Americans, spend more time indoors. Therefore, Repace and Lowrey's estimates of the proportion of American nonsmokers exposed to tobacco smoke indoors can be viewed as conservative estimates of the proportion of Canadian nonsmokers so exposed.

Air quality standards

In Canada the standards set by the ACGIH are usually used to judge air quality in the workplace.[1,47] However, to date the ACGIH has provided neither recommendations nor documentation pertaining to a TLV for tobacco smoke per se. TLVs and related documentation are provided primarily for identifiable chemical substances that are closely associated with industrial processes.

Tobacco smoke is a complex mixture of particles and gases, and the ACGIH suggests a formula for determining the TLVs for mixtures.[47] The air quality standard is deemed not to have been exceeded if

$$\sum_{i=1}^{n} \frac{Ci}{Ti} \leq 1,$$

where C is the observed concentration of the dangerous substance i, and T is the listed TLV for that substance. The TLV of a mixture of dangerous substances is to be applied only when the components have similar toxicologic effects and when the concentrations and sources of the dangerous substances are known. Unfortunately, the toxicologic effects of many of the chemicals in tobacco smoke are poorly understood. In the numerous investigations to determine the concentrations of various toxic substances in tobacco smoke in indoor air, the conditions under which the measurements were obtained differed greatly.[48] Therefore, the calculation of an exact TLV for tobacco smoke is difficult. Nevertheless, enough is known about the effect of tobacco smoke on indoor air for the US National Research Council to have concluded that "public policy should clearly articulate that involuntary exposure to tobacco smoke has adverse health effects and ought to be minimized or avoided where possible".[48]

When there are a number of harmful substances it is frequently only feasible to evaluate the hazards by measuring a single substance. In such cases the ACGIH[47] recommends that "the threshold limit used for this substance should be reduced by a suitable factor, the magnitude of which will depend on the number, toxicity and relative quantity of other contaminants ordinarily present". To determine a suitable TLV for tobacco smoke a more detailed examination is needed of the known health effects of and the recommended exposure limits for some of the substances in tobacco smoke.

In smoky areas with minimal ventilation the TLV for carbon monoxide may be exceeded, but even a modest increase in the amount of ventilation results in a rapid fall in the carbon monoxide level. The air concentrations of carbon monoxide and other contaminants from tobacco smoke are often measured at some distance from the nearest smoker and thus tend to be lower than those to which persons working close to smokers are exposed.[8] Carbon monoxide concentrations of up to 29 ppm were found in work areas where smoking was permitted;[27] this level is below the TLV (50 ppm) but well above the Ambient Air Quality Standard[48] (the US standard for outdoor air in cities) of 9 ppm.

There are at least 38 known or probable carcinogens in the particulate phase and another 16 in the volatile phase of tobacco smoke.[2,8,49] The eight volatile *N*-nitrosamines are largely retained by the smoke particulates in the glass fibre filters that are used to separate the two phases. The carcinogenicity of tobacco smoke particulates in animals exceeds that expected from a summation of the carcinogenicity of the individual known carcinogens.[2] This is probably due, at least in part, to the presence of many tumour promoters and cocarcinogens in the volatile phase. For example, catechol is a known cocarcinogen and is the main phenolic compound in tobacco smoke, its concentration being 20 to 460 μg per cigarette.[2]

Most of the cancer-causing and other toxic chemicals in tobacco smoke are formed in a pyrolysis–distillation zone just behind the heat-generating combustion zone.[2] The concentration of toxic chemicals is higher in sidestream than in mainstream smoke because the temperature of the burning tip of a cigarette or cigar that is not being smoked is lower; hence, combustion is less complete than during a puff.[2]

Table III lists the known and probable carcinogens in tobacco smoke. The ACGIH lists acrylonitrile and vinyl chloride as known human carcinogens with assigned TLVs of 4.5 and 10 mg/m^3 respectively.[1] Two other chemicals, 2-naphthylamine and 4-aminobiphenyl, are listed as known human bladder carcinogens to which "no exposure or contact by any route — respiratory, skin or oral, as detected by the most sensitive methods — shall be permitted".[47] Although these two substances are known to be present in very small quantities in

Table II—Estimated daily annual average exposure of nonsmokers to tobacco smoke at work and at home[3]

Exposure site	Probability of exposure (rounded values, %)*	Estimated amount of particulate phase inhaled daily (mg)	
		Average	Probability-weighted average
Work and home	63 × 62 = 39	2.27	0.89
Neither work nor home	37 × 38 = 14	–	–
Work only	38 × 63 = 24	1.82	0.44
Home only	37 × 62 = 23	0.45	0.10
Total	100		1.43

sidestream smoke, studies of their concentration in ambient air containing tobacco smoke have not yet been reported.

The International Agency for Research on Cancer (IARC) lists arsenic, benzene, and soots, tars and oils, as well as "whole tobacco smoke", as known human carcinogens.[49] Both the ACGIH and the IARC consider benzo(*a*)pyrene, formaldehyde and hydrazine as probably carcinogenic to humans on the basis of repeated demonstrations of carcinogenesis in animals and the limited evidence of carcinogenesis in humans. The IARC also lists nickel and cadmium as probable human carcinogens. The ACGIH considers *N*-nitrosodimethylamine as probably carcinogenic and recommends "that exposures be avoided, insofar as possible, or otherwise be kept to an absolute minimum".[1] Brunnemann and Hoffmann[50] have detected *N*-nitrosodimethylamine at concentrations of 0.11 to 0.24 $\mu g/m^3$ in ambient air contaminated with tobacco smoke.

The ACGIH also lists TLVs for most of the other toxic agents in tobacco smoke shown in Table I. The TLVs are also given for many other toxic substances, such as nickel, cadmium and arsenic (Table III). However, because tobacco smoke is a mixture of substances all the TLVs should be reduced by an unknown amount.

The ACGIH has suggested a TLV of 5 mg/m^3 for respirable nuisance particulates.[1] However, this category was defined to include dusts that have a long history of little adverse effect on the respiratory system. Neither the IARC nor the ACGIH has made specific reference to the particulate phase of tobacco smoke.

Repace and Lowrey[3] have given the total suspended particulate concentrations for 56 indoor locations. In all 23 areas where tobacco smoke was present the US National Ambient Air Quality Standard, 75 $\mu g/m^3$,[48] and the Canadian National Air Quality Objective, 70 $\mu g/m^3$,[51] both *outdoor* air standards for time-weighted annual exposure to total suspended particulates, were exceeded. These 23 cases involved short-term time-averaged measurements of total suspended particulate concentrations, and the 24-hour standards were exceeded in only a few cases. However, Repace and Lowrey[28] have argued that in each of the 23 areas there would be repeated violations of the 24-hour standard and almost certainly repeated violation of the annual standard. In all 33 locations where tobacco smoke was not present the concentrations of total suspended particulates were well within both the United States' and Canada's standards. From these findings Repace and Lowrey[3] have estimated that nonsmokers are exposed to an average of 1.43 mg of tobacco smoke particulates per day (Table II).

In another investigation Repace and Lowrey[52] analysed data on the concentrations of tobacco smoke particulates in the workplace, the risk of such exposure and the effect of ventilation on indoor air quality. Standards of lifetime involuntary carcinogenic risk to the public that are used by the US Food and Drug Administration and the US Environmental Protection Agency range from 10^{-5} to 10^{-7}. Repace and Lowrey[52] assumed a 10^{-5} risk for lifetime involuntary exposure to tobacco smoke and calculated the resulting maximum permissible annual average of daily exposure to tobacco smoke particulates in the workplace to be 0.75 $\mu g/m^3$. The current published air quality standards do not list precise threshold limit values for tobacco smoke particulates. However, in average conditions the actual concentration of tobacco particulates in a typical office was estimated to be about 200 $\mu g/m^3$.[52]

Table III—Known and probable human carcinogens in tobacco smoke, as determined by the American Conference of Governmental Industrial Hygienists Inc. (ACGIH)[1] and the International Agency for Research on Cancer (IARC)[49]

Carcinogen	Status, according to ACGIH or IARC or both, of carcinogen		Threshold limit value (mg/m^3) assigned by ACGIH
	Known	Probable	
Acrylonitrile	ACGIH		4.5
Vinyl chloride	IARC, ACGIH		10
2-naphthylamine	IARC, ACGIH		0
4-aminobiphenyl	IARC, ACGIH		0
Arsenic	IARC		0.2
Benzene	IARC	ACGIH	30
Soots, tars and oils	IARC		Not assigned
Whole tobacco smoke	IARC		Not assigned
Benzo(*a*)pyrene		IARC, ACGIH	Not assigned
Formaldehyde		IARC, ACGIH	1.5
Hydrazine		IARC, ACGIH	0.1
N-nitrosodimethylamine		ACGIH	Not assigned; avoidance of exposure recommended
Nickel		IARC	1.0
Cadmium		IARC	0.05

Summary

Several authoritative agencies have reviewed the scientific evidence concerning involuntary exposure to tobacco smoke and have concluded that it is a health hazard to be avoided if possible. These agencies include the US Surgeon General's Office,[2,7] the US National Research Council,[48] the Ontario Council of Health's Task Force on Smoking[53] and the Ontario Medical Association (*The Citizen*, Ottawa, Mar. 31, 1983: page 18).

Repace and Lowrey[52] have proposed a threshold limit value for tobacco smoke particulates that is much lower than the existing observed levels in office accommodations. Our review of recommended limits of exposure in the workplace to the known and probable carcinogens in ambient tobacco smoke revealed that there are no published limits of exposure to tobacco smoke per se. However, for several of the components in tobacco smoke the

recommended exposure limit is either zero or not assigned, suggesting that there may not be a safe level for involuntary exposure to tobacco smoke.

References

1. American Conference of Governmental Industrial Hygienists Inc: *Documentation of the Threshold Limit Values*, 4th ed, Cincinnati, Ohio, 1980
2. US Department of Health and Human Services: *The Health Consequences of Smoking: Cancer. A Report of the Surgeon General* (DHHS [PHS] 82-50179), Rockville, Md, 1982
3. Repace JL, Lowrey AH: Modelling exposure of non-smokers to ambient tobacco smoke. Presented at the 76th annual meeting of the Air Pollution Control Association, Atlanta, Ga, June 20–25, 1983
4. Collishaw NE: Is cigarette consumption declining in Canada? Presented at the Fifth World Conference on Smoking and Health, Winnipeg, 1983
5. Collishaw NE, Mulligan L: Recent trends in tobacco consumption in Canada and other countries. *Chronic Dis Can* 1984; 4: 52–54
6. McCusker K, Hiller FC, Wilson JD et al: Aerodynamic sizing of tobacco smoke particulate from commercial cigarettes. *Arch Environ Health* 1983; 38: 215–218
7. US Department of Health and Human Services: *The Health Consequences of Smoking: Chronic Obstructive Lung Disease. A Report of the Surgeon General* (DHHS [PHS] 84-50205), Rockville, Md, 1984
8. US Department of Health, Education, and Welfare: *Smoking and Health: a Report of the Surgeon General* (DHEW publ no [PHS] 79-50066), Rockville, Md, 1979
9. Cohen D, Arai SF, Brain JD: Smoking impairs long-term dust clearance from the lung. *Science* 1979; 204: 514–517
10. Hoffmann D, Haley NJ, Brunnemann KD et al: Cigarette sidestream smoke: formation, analysis and model studies on the uptake by non-smokers. Presented at the US–Japan Meeting on New Etiology of Lung Cancer, Honolulu, Mar 21–23, 1983
11. Ayer HE, Yeager DW: Irritants in cigarette smoke plumes. *Am J Public Health* 1982; 72: 1283–1285
12. Klus H, Kuhn H: Distribution of various tobacco smoke components among mainstream and sidestream smoke: a survey. *Beitr Tabakforsch Int* 1982; 11: 229–265
13. US Department of Health and Human Services: *The Health Consequences of Smoking: Cardiovascular Disease. A Report of the Surgeon General* (DHHS [PHS] 84-50204), Rockville, Md, 1983
14. Aronow WS: Effect of passive smoking on angina pectoris. *N Engl J Med* 1978; 299: 21–24
15. Wright G, Randell P, Shephard RJ: Carbon monoxide and driving skills. *Arch Environ Health* 1973; 27: 349–354
16. Speer F: Tobacco and the non-smoker: a study of subjective symptoms. *Arch Environ Health* 1968; 16: 443–446
17. Hugod C, Hawkins LH, Astrup P: Exposure of passive smokers to tobacco smoke constituents. *Int Arch Occup Environ Health* 1978; 42: 21–29
18. Weber A, Fischer T, Grandjean E: Passive smoking in experimental and field conditions. *Environ Res* 1979; 20: 205–216
19. Shor RE, Williams DC: Reported physiological and psychological symptoms of tobacco smoke pollution in non-smoking and smoking college students. *J Psychol* 1979; 101: 203–218
20. Shephard RJ, Ponsford E, LaBarre R et al: Effect of cigarette smoke on the eyes and airway. *Int Arch Occup Environ Health* 1979; 43: 135–144
21. Weber A, Fischer T, Grandjean E: Passive smoking: irritating effects of the total smoke and the gas phase. *Int Arch Occup Environ Health* 1979; 43: 183–193
22. Weber A, Fischer T: Passive smoking at work. *Int Arch Occup Environ Health* 1980; 44: 209–221
23. Bocanegra TS, Espinoza LR: Raynaud's phenomenon in passive smokers [C]. *N Engl J Med* 1980; 303: 1419
24. Prevalence in Canada of health conditions aggravated by tobacco smoke. *Chronic Dis Can* 1983; 4: 9–11
25. Zussman BM: Tobacco sensitivity in the allergic patient. *Ann Allergy* 1970; 28: 371–377
26. Dahms TE, Bolin JF, Slavin RG: Passive smoking: effects on bronchial asthma. *Chest* 1981; 80: 530–534
27. White JR, Froeb HF: Small-airways dysfunction in non-smokers chronically exposed to tobacco smoke. *N Engl J Med* 1980; 302: 720–723
28. Repace JL, Lowrey AH: Indoor air pollution, tobacco smoke and public health. *Science* 1980; 208: 464–472
29. Kauffmann F, Tessier J-F, Oriol P: Adult passive smoking in the home environment: a risk factor for chronic airflow limitation. *Am J Epidemiol* 1983; 117: 269–280
30. Tager IB, Weiss ST, Munoy S et al: Longitudinal study of the effects of maternal smoking on pulmonary function in children. *N Engl J Med* 1983; 309: 699–703
31. Hirayama T: Non-smoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan. *Br Med J* 1981; 282: 183–185
32. Trichopoulos D, Kalandidi A, Sparros L et al: Lung cancer and passive smoking. *Int J Cancer* 1981; 27: 1–4
33. Correa P, Pickle LW, Fontham E et al: Passive smoking and lung cancer. *Lancet* 1983; 2: 595–597
34. Trichopoulos D, Kalandidi A, Sparros L: Lung cancer and passive smoking: conclusion of Greek study. Ibid: 677–678
35. Garfinkel L: Time trends in lung cancer mortality among non-smokers and a note on passive smoking. *J Natl Cancer Inst* 1981; 66: 1061–1066
36. Friedman GD, Petitti DB, Bawol RD: Prevalence and correlates of passive smoking. *Am J Public Health* 1983; 73: 401–405
37. Repace JL: Consistency of research data on passive smoking and lung cancer [C]. *Lancet* 1984; 1: 506
38. Hirayama T: Passive smoking and lung cancer: consistency of association. *Lancet* 1983; 2: 1425–1426
39. Selikoff IJ, Hammond EC, Churg J: Asbestos exposure, smoking and neoplasia. *JAMA* 1968; 204: 104–110
40. Russell MAH, Cole PV, Brown E: Absorption by non-smokers of carbon monoxide from air polluted by tobacco smoke. *Lancet* 1973; 1: 576–579
41. Russell MAH, Feyerabend C: Blood and urinary nicotine in non-smokers. *Lancet* 1975; 1: 179–181
42. Feyerabend C, Higenbottam T, Russell MAH: Nicotine concentrations in urine and saliva of smokers and non-smokers. *Br Med J* 1982; 284: 1002–1004
43. Olshansky SJ: Is smoker/non-smoker segregation effective in reducing passive inhalation among non-smokers? *Am J Public Health* 1982; 72: 737–739
44. Foliart D, Benowitz NL, Becker CE: Passive absorption of nicotine in airline flight attendants [C]. *N Engl J Med* 1983; 308: 1105
45. Bos RP, Theuws JLG, Henderson PT: Excretion of mutagens in human urine after passive smoking. *Cancer Lett* 1982; 19: 85–90
46. Hardee GE, Stewart T, Capomacchia AC: Tobacco smoke xenobiotic compound appearance in mothers' milk after involuntary smoke exposures: I. Nicotine and cotinine. *Toxicol Lett* 1983; 15: 109–112
47. American Conference of Governmental Industrial Hygienists Inc: *Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1983–84*, Cincinnati, Ohio, 1983
48. National Research Council: *Indoor Pollutants*, Natl Acad Pr, Washington, 1981
49. *Chemicals, Industrial Processes and Industries Associated with Cancer in Humans* (IARC monogr, vols 1–29, suppl 4), International Agency for Research on Cancer, Lyon, 1982: 14–16
50. Brunnemann KD, Hoffmann D: Chemical studies on tobacco smoke — 59. Analysis of volatile nitrosamines in tobacco smoke and polluted indoor environments. In *Environmental Aspects of N-Nitroso Compounds* (IARC monogr, vol 19), International Agency for Research on Cancer, Lyon, 1978: 343–355
51. Environment Canada: *National Air Pollution Surveillance, 1981*, Ottawa, 1982: 64–65
52. Repace JL, Lowrey AH: A proposed indoor air quality standard for ambient tobacco smoke. Presented at the Indoor Air Quality and Climate Conference, Stockholm, Aug 20–24, 1984
53. McMichael WC: *Tobacco Smoke and Energy Conservation*, Building Systems Design, Oct/Nov 1973