

1. Composition, Exposure and Regulations

1.1 Composition

1.1.1 *Secondhand smoke*

During smoking of cigarettes, cigars, pipes and other tobacco products, in addition to the mainstream smoke drawn and inhaled by smokers, a stream of smoke is released between puffs into the air from the burning cone. Once released, this stream (also known as the sidestream smoke) is mixed with exhaled mainstream smoke as well as the air in an indoor environment to form the secondhand smoke to which both smokers and nonsmokers are exposed. A small additional contribution to the smoke issues from the tip of the cigarette and through the cigarette paper during puffing and through the paper and from the mouth end of the cigarette between puffs (IARC, 1986; NRC, 1986; US EPA, 1992). Thus, secondhand tobacco smoke is composed of aged exhaled mainstream smoke and diluted sidestream smoke.

Secondhand tobacco smoke contains a variable proportion of exhaled mainstream smoke ranging from 1 to 43% (Baker & Proctor, 1990). Because of its rapid dilution and dispersion into the indoor environment, secondhand tobacco smoke acquires different physicochemical properties to those of mainstream smoke and sidestream smoke and the concentrations of the individual constituents are decreased. The principal physical change is a decrease in the proportion of smoke constituents found in the particulate phase as opposed to the vapour phase of the smoke. The median particle size of secondhand tobacco smoke is subsequently smaller than that of the particles of mainstream smoke. The principal chemical change is in the composition (i.e. in the relative quantities of the individual constituents present); this is caused by differences in the ways in which individual constituents respond to ventilation and to contact with indoor surfaces. There is some indication that chemical transformation of reactive species also occurs.

The effects of exposure to secondhand tobacco smoke, or involuntary (passive) smoking, cannot be estimated from any individual constituents. Secondhand tobacco smoke is actually a complex mixture, containing many compounds for which concentrations can vary with time and environmental conditions. Cigarette smoking is the main source for involuntary exposure because it is by far the most prevalent form of tobacco smoking although specific patterns may differ between countries. Emissions of sidestream smoke in indoor environments with low ventilation rates can result in concentrations of

toxic and carcinogenic agents above those generally encountered in ambient air in urban areas (IARC, 1986; Jenkins *et al.*, 2000).

Studies on the complex composition of secondhand tobacco smoke in 'real world' conditions have been limited partly because of the presence of additional sources of secondhand smoke constituents. Therefore compositional and physical studies of secondhand tobacco smoke have often been performed in environmental chambers (also known as a 'controlled experimental atmosphere'). The disadvantage of the controlled experimental atmosphere is that it does not reflect real life situations. The studies of the chemical composition of secondhand tobacco smoke, either in a controlled experimental atmosphere or in the field, have been limited. This is mainly because there are still no standardized criteria for the development of experimental atmospheres that represent secondhand tobacco smoke (Jenkins *et al.*, 2000).

Respirable suspended particles (only those particles that are small enough to reach the lower airways of the human lung) can exist in many forms in indoor air: those resulting from secondhand tobacco smoke are present in the form of liquid or waxy droplets. They are smaller than the particles in mainstream smoke. The mass median diameter of mainstream smoke particles averages 0.35–0.40 μm . Gravimetric determination indicates that the respirable suspended particles of secondhand tobacco smoke in typically encountered environments may comprise one-third of the respirable suspended particles in indoor air. However, in some environments, this fraction may be as much as two-thirds (Jenkins *et al.*, 2000).

Respirable suspended particle concentrations of 4091 $\mu\text{g}/\text{m}^3$ were measured in an experimental room in which 120 cigarettes were smoked during 9 hours in 1 day for the evaluation of exposure to benzene and other toxic compounds (Adlkofer *et al.*, 1990).

Worldwide in indoor environments where people smoke, the mean levels of respirable suspended particles ranged from 24 to 1947 $\mu\text{g}/\text{m}^3$. Background levels of respirable suspended particles depend on many factors including local vehicular traffic patterns, quality of ventilation systems and the presence of other sources (e.g. cooking and wood-burning stoves). Comparisons between smoking and nonsmoking locations revealed up to threefold higher concentrations of respirable suspended particles in smoking areas. The US EPA-proposed maximal level for fine particles in outdoor ambient air (65 $\mu\text{g}/\text{m}^3$ particulate matter, that is 2.5 μm or smaller in size, for 24 h) is frequently exceeded in indoor situations where people are smoking (Jenkins *et al.*, 2000).

Besides respirable suspended particles and nicotine (see Section 1.2), carbon monoxide (CO) has been the most extensively studied constituent of secondhand tobacco smoke. The contemporary commercial cigarettes in the USA deliver approximately 15 mg CO in mainstream smoke and an additional 50 mg in sidestream smoke (Jenkins *et al.*, 2000). In an indoor environment, CO concentrations are rapidly diluted. The measured mean concentrations reported for CO in offices, other workplaces, functions and public gatherings, transportation, restaurants and cafeterias, bars and taverns where people smoke ranged from 0.2 to 33 ppm. The American Society of Heating, Refrigerating and

Air-Conditioning Engineers (ASHRAE) standard for CO concentration in indoor air is 9 ppm (Jenkins *et al.*, 2000).

The mean levels of nitric oxide (NO) in indoor areas were reported from not detected to 500 ppb and those of nitrogen dioxide (NO₂) from not detected to 76 ppb (Jenkins *et al.*, 2000).

There are a number of studies that have addressed the composition of secondhand tobacco smoke beyond the 'common' constituents such as nicotine, CO and respirable suspended particles. A few of these are shown in Table 1.1 (Eatough *et al.*, 1989; Löfroth *et al.* 1989; Higgins *et al.*, 1990; Löfroth, 1993; Martin *et al.*, 1997). The focus of these studies was primarily on vapour-phase constituents. Vapour phase represents the bulk of the mass of secondhand tobacco smoke whereas the respirable suspended particle-related constituents are present at very low concentrations that are very difficult to quantify. For example, if the levels of respirable suspended particles are in the range of 20 to 1000 µg/m³, constituents of the particulate phase present at concentrations of 1–100 ppm in the particles themselves will be present at airborne concentrations from 20 pg/m³ to 100 ng/m³. These are very low concentrations for detection by any sampling and analysis method (Jenkins *et al.*, 2000).

As can be seen from the data in Table 1.1, the field studies show considerable variation in the measured levels of constituents of secondhand tobacco smoke. Similar concentrations of benzene and isoprene to those shown in Table 1.1 were reported in a smoke-filled bar (from 26 to 36 µg/m³ and 80–106 µg/m³, respectively), although the nicotine levels were much lower (22 µg/m³). The concentration of 1,3-butadiene measured in the smoke-filled bar was from 2.7 to 4.5 µg/m³ (Brunnemann *et al.*, 1990). In a field study of 25 homes of smokers, Heavner *et al.* (1995) estimated that the median fraction of benzene contributed by secondhand tobacco smoke was 13% (ranging from 0 to 63%).

In a study of six homes of smokers, secondhand tobacco smoke was found to make a substantial contribution to the concentrations of 1,3-butadiene (Kim *et al.*, 2001).

The levels of carbonyl compounds measured in an experimental room under extremely high concentrations of secondhand tobacco smoke (Adlkofer *et al.*, 1990) were: formaldehyde, 49 µg/m³; acetaldehyde, 1390 µg/m³ and propionaldehyde, 120 µg/m³. The concentrations of other constituents of secondhand tobacco smoke were: nicotine, 71 µg/m³; benzene, 206 µg/m³; benzo[*a*]pyrene, 26.7 ng/m³; pyrene, 25 ng/m³ and chrysene, 70.5 ng/m³.

Benzo[*a*]pyrene was also detected in natural environments containing secondhand tobacco smoke, with concentrations ranging from not detected to 3.6 ng/m³ (or up to 3.35 ng/m³ when the background concentration was subtracted) (Jenkins *et al.*, 2000). Trace levels of some other polycyclic aromatic hydrocarbons (PAHs; such as naphthalene, chrysene, anthracene, phenanthrene and benzofluoranthenes) were also reported. The vapour-phase 2- to 3-ring PAHs predominate quantitatively over the higher-ring system PAHs.

Table 1.1. Concentrations (in $\mu\text{g}/\text{m}^3$) of selected constituents of secondhand tobacco smoke in some experimental and real-life situations^a

Constituent	18-m ³ chamber: mean for 50 best-selling US cigarettes (Martin <i>et al.</i> , 1997)	Living quarters (Löfroth, 1993)	Tavern (Löfroth <i>et al.</i> , 1989)	Discothèque (Eatough <i>et al.</i> , 1989)	Home (Higgins <i>et al.</i> , 1990)
Respirable suspended particles	1440	240–480	420	801 ^b	–
Nicotine	90.8	8–87	71	120	51.8
CO (ppm)	5.09	–	4.8	22.1	–
Benzene	30	–	27	–	17.6
Formaldehyde	143	–	104	–	–
1,3-Butadiene	40	–	19	–	–
Acetaldehyde	268	–	204	–	–
Isoprene	657	50–200	150	–	83.3
Styrene	10	–	–	–	7.3
Catechol	1.24	–	–	–	–
3-Ethenyl pyridine	37.1	–	–	18.2	–
Ethylbenzene	8.5	–	–	–	8.0
Pyridine	23.8	–	–	17.6	6.5
Toluene	54.5	–	–	–	51.2
Limonene	29.1	–	–	–	22.0

Modified from Jenkins *et al.* (2000)

–, not reported

^a These are not typical average concentrations, but represent the higher end of the exposure scale.

^b Fine particles (< 2 μm size)

The levels of *N*-nitrosodimethylamine (NDMA) measured in the field (e.g. in work-rooms, conference rooms, restaurants and bars where people smoked) ranged from less than 10 ng/m³ to 240 ng/m³ (Jenkins *et al.*, 2000). In unventilated offices in which 11–18 cigarettes were smoked during a 2-h period, up to 8.6 ng/m³ *N*-nitrosodiethylamine (NDEA) and up to 13 ng/m³ *N*-nitrosopyrrolidine (NPYR) were measured.

The *N'*-nitrosonornicotine (NNN) concentrations measured in a poorly ventilated office where heavy smoking of cigarettes, cigars and pipes took place ranged from not detected to 6 ng/m³ and those of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) from not detected to 13.5 ng/m³ (Klus *et al.*, 1992). The upper levels reported by Klus *et al.* (1992) and by Adlkofer *et al.* (1990) for the 'heavily smoked rooms' (11 cigarettes smoked in 2 h in a 84 m² office) were somewhat lower than those measured by Brunne-mann *et al.* (1992): NNN concentrations ranged from not detected to 22.8 ng/m³ and NNK concentrations between 1.4 and 29.3 ng/m³, measured inside bars, restaurants, trains, and a car, an office and a smoker's home (Brunne-mann *et al.*, 1992).

The effects of cigar smoking on indoor levels of CO, respirable suspended particles and particle-bound PAH particles were investigated in an office where several brands of cigar were machine smoked, in a residence where two cigars were smoked by a person and at cigar social events where up to 18 cigars were smoked at a time. The average concentrations of CO at cigar social events were comparable with, or larger than, those measured on a main road during rush-hour traffic. A mass balance model developed for predicting secondhand tobacco smoke was used in this study to obtain CO, respirable suspended particle and PAH emission. These factors show that cigars can be a stronger source of CO than cigarettes. In contrast, cigars may have lower emissions of respirable suspended particles and PAHs per gram of tobacco consumed than cigarettes, but the greater size and longer smoking time of a single cigar results in greater total respirable suspended particle and PAH emission than from a single cigarette (Klepeis *et al.*, 1999). Nelson *et al.* (1997) tested six brands of cigar and the yields of respirable suspended particles averaged 52 mg/cigar. Yields of CO, nitric oxide (NO) and nitrogen dioxide (NO₂) averaged 32, 10.5 and 2.1 mg/cigar, respectively, and that of volatile organic compounds (VOC) (analysed with gas chromatograph-flame ionization detection (FID)) was estimated to be 340 mg/cigar (propane equivalent). Ratios of secondhand tobacco smoke respirable suspended particles to the surrogate standards for the particulate markers ultra-violet particulate matter (UVPM) and fluorescent particulate matter (FPM) were 6.5 and 27.8, respectively. Another particulate marker, solanesol, made up 2.1% of the particles from cigars. For the two gas-phase markers, the ratio of 3-ethenyl pyridine to other gas-phase species was more consistent than ratios involving nicotine.

The comparative analysis of the composition of secondhand tobacco smoke from Eclipse (a cigarette that primarily heats tobacco rather than burning it) and from four commercial cigarettes with a wide range of Federal Trade Commission (FTC) yields is shown in Table 1.2 (Bombick *et al.*, 1998). Eclipse contributed similar amounts of CO to secondhand tobacco smoke to those contributed by burning-tobacco cigarettes but contributed 86–90% less respirable suspended particles. Commercial cigarettes, however,

Table 1.2. Mean concentrations (in $\mu\text{g}/\text{m}^3$) of selected components of secondhand smoke of four commercial cigarette brands and Eclipse^a measured in a chamber with a controlled experimental atmosphere

Constituent	Full-flavour brand	Full-flavour light	100-mm brand	Ultra-light	Eclipse
Respirable suspended particles	1458	1345	1706	1184	181
Nicotine	54	63	58	51	4.3
CO (ppm)	6.5	6.2	7.9	6.6	5.2
3-Ethenylpyridine	25	28	28	34	0.56
Acetaldehyde	313	301	384	312	46
Phenol	17.4	16.7	20.0	16.8	4
NO _x (ppb)	241	233	268	250	24
Total hydrocarbons ^b	2.6	2.6	3.0	2.8	0.47

From Bombick *et al.* (1998)

^a A cigarette that primarily heats (rather than burns) tobacco

^b Analysed with gas chromatograph–flame ionizing detector (ppm)

contributed a similar amount of constituents to secondhand tobacco smoke, regardless of their ranking on the FTC scale.

1.1.2 Exhaled mainstream smoke

Baker and Proctor (1990) estimated that exhaled mainstream smoke contributes 3–11% of CO, 15–43% of particles and 1–9% of nicotine to secondhand tobacco smoke. Non-inhaling smokers can contribute larger amounts. There is little information on how much exhaled mainstream smoke contributes to the overall composition of secondhand tobacco smoke except that the contribution to the particulate phase is more significant than that to the vapour phase (see monograph on tobacco smoke/Section 4 — breath compounds).

1.1.3 Sidestream smoke

The composition of cigarette sidestream smoke is similar to that of mainstream smoke. However, the relative quantities of many of the individual constituents of sidestream smoke are different from those found in mainstream smoke. Also, the absolute quantities of most of the constituents released in sidestream smoke differ from those delivered in mainstream smoke (Jenkins *et al.*, 2000).

Like mainstream cigarette smoke, sidestream smoke contains many compounds that are emitted as gases and particles. The distinction between particle and vapour-phase constituents is appropriate for those constituents that are non-volatile (e.g. high-mole-

cular-weight organic compounds and most metals) and those that are clearly gases (e.g. CO). Constituents with appreciable vapour pressure (i.e. most of the constituents of tobacco smoke) can be found in both the particulate phase and the vapour phase of cigarette smoke. The term 'semivolatiles' has been used to describe such constituents. The degree to which these compounds are distributed between the particle and vapour phases is determined by their volatility (and stability) and the characteristics of their environment. These constituents are distributed preferentially in the particulate phase in highly concentrated smokes, such as those inhaled by smokers, and preferentially in the particle and vapour phases in highly diluted smokes, such as those encountered by involuntary smokers. The phase distribution and the ultimate fate of any given constituent released into the ambient environment is likely to differ depending upon ambient conditions and upon the chemical or physical properties of that constituent.

The manner in which cigarettes are smoked greatly influences their mainstream delivery and sidestream emissions. For the different machine-smoking protocols referred to in this monograph, see Table 1.9 in the monograph on tobacco smoke. Particulate matter that is released in mainstream smoke during active smoking enters the respiratory tract largely intact, whereas the particulate matter in sidestream smoke is available for inhalation only after dilution in ambient air and after the physical and chemical changes that occurred during that dilution. However, conventional analysis of sidestream smoke provides only information on the quantities of individual smoke constituents released into the air. Moreover, the methods for analysis of sidestream smoke are not as well defined as those for mainstream smoke (Jenkins *et al.*, 2000).

As they leave the cigarette, sidestream smoke particles are initially slightly smaller than mainstream smoke particles (geometric mean diameter, 0.1 μm versus 0.18 μm ; Guerin *et al.*, 1987). The natural dissipation rates of sidestream smoke particles dispersed in an experimental chamber were studied from the standpoint of a static atmosphere and were expressed as half-lives of residence in the air. The half-lives for particles with diameters less than 0.3 μm , 0.3–0.5 μm and 0.5–1 μm were found to be 25.5, 12.8 and 4.9 h, respectively. Total particulate matter decreased by half over 6.2 h (Vu Duc & Huynh, 1987). However in real-life situations the ageing of sidestream smoke over several minutes may lead to an increase in particle size of secondhand tobacco smoke due to the coagulation of particles and the removal of smaller particles that attach to surfaces in the environment. Sidestream smoke is produced at generally lower temperatures and with a very different oxygen flux to that of mainstream smoke.

The ratio of sidestream smoke to mainstream smoke is customarily used to express the distribution of individual constituents between the two smoke matrices. The distribution of specific components is dependent on their mechanism of formation. Higher ratios of sidestream smoke to mainstream smoke between cigarettes or smoking conditions generally reflect a lower mainstream smoke delivery with no significant change in sidestream smoke delivery. Under similar smoking conditions, filter-tipped cigarettes will have lower mainstream smoke yields than their untipped analogues. Sidestream smoke yields will not vary greatly, because they reflect the weight of tobacco burned during

smouldering. In general, more tobacco is burned during smouldering than during puffing (Guerin *et al.*, 1987).

Adams *et al.* (1987) reported that sidestream smoke contains more alkaline and neutral compounds than mainstream smoke (the pH of the sidestream smoke of cigarettes with a wide range of FTC yields averaged 7.5 [7.2–7.7], whereas the pH of mainstream smoke averaged 6.1 [6.0–6.3]). The differences are due to temperature during burning and mechanisms of chemical transfer (release) from unburned tobacco.

Many constituents of sidestream smoke belong to chemical classes known to be genotoxic and carcinogenic. These include the IARC group 1 carcinogens benzene, cadmium, 2-aminonaphthalene, nickel, chromium, arsenic and 4-aminobiphenyl; the IARC group 2A carcinogens formaldehyde, 1,3-butadiene and benzo[*a*]pyrene; and the IARC group 2B carcinogens acetaldehyde, isoprene, catechol, acrylonitrile, styrene, NNK, NNN and lead among others.

Adams *et al.* (1987) determined the levels of selected toxic and carcinogenic agents in the mainstream and sidestream smoke of four different types of US commercial cigarette brands — untipped and filter-tipped — with a wide range of FTC yields. In this study, smoke was generated by a machine using the standard FTC method. The concentrations of all agents except NNN were higher in the sidestream than in the mainstream smoke of both untipped and filter-tipped brands. The tar yields in sidestream smoke ranged from 14 to 24 mg per cigarette (similar to the range reported by Ramsey *et al.*, 1990) and were, on average, 5.3 times higher than those in mainstream smoke. The highest sidestream smoke/mainstream smoke ratio for tar was calculated for ultra low-yield cigarettes (ratio, 15.7) and the lowest for untipped cigarettes (ratio, 1.12). The mainstream smoke yields are strongly affected by variables that only slightly affect sidestream smoke yields (Guerin *et al.*, 1987). The ratios of sidestream smoke to mainstream smoke for nicotine, CO and NNK for the ultra low-yield brand were 21.1, 14.9 and 22.3, respectively. The highest emissions of nicotine, NNK and NNN were measured in the sidestream smoke of untipped cigarettes (4.62 mg, 1444 ng and 857 ng, respectively). In mainstream smoke, these values were 2.04 mg, 425 ng and 1007 ng, respectively. The levels of volatile *N*-nitrosamines in sidestream smoke greatly exceeded those measured in mainstream smoke (e.g. 735 ng versus 31.1 ng NDMA per untipped cigarette and 685 ng versus 4.1 ng NDMA for ventilated filter-tipped cigarettes; average ratio, 95). The average ratio of sidestream smoke to mainstream smoke for the carcinogen NPYR was 10. The authors concluded that the availability of cigarettes with greatly reduced amounts of carcinogens in mainstream smoke had little bearing on the emissions of carcinogens in sidestream smoke.

Chortyk and Schlotzhauer (1989) compared the emissions of various smoke components for 19 low-yield brands of filter-tipped cigarettes with those measured for a reference high-yield untipped brand. It was found that low-yield cigarettes produced large quantities of tar in sidestream smoke, about equal to that of the high-yield cigarette. On an equal weight basis, the low-tar cigarettes emitted more of these hazardous compounds into sidestream and secondhand smoke than did the high-tar cigarette.

The yields of various constituents of sidestream smoke of 15 Canadian cigarette brands measured using the FTC machine-smoking protocol with the exception that cigarettes were smoked to a butt length of 30 mm, ranged as follows: tar, from 15.8 to 29.3 mg per cigarette in non-ventilated brands and from 24.2 mg to 36.0 mg in ventilated brands; nicotine, from 2.7 mg to 4.6 mg in non-ventilated brands and 3.0 mg to 6.1 mg in ventilated brands, and CO, from 40.5 mg to 67.3 mg in non-ventilated brands and 46.5 mg to 63.1 mg in ventilated brands. Yields in sidestream smoke were much higher than those in mainstream smoke for all brands tested. The average ratios for sidestream smoke to mainstream smoke were 3.5, 6.6 and 6.8 for tar, nicotine and CO, respectively. The highest yields from sidestream smoke were obtained from the brands with the lowest mainstream smoke yields (Rickert *et al.*, 1984). The concentrations of carbon monoxide in the sidestream smoke in the Canadian cigarettes were higher than those reported for four different types of American blend cigarettes smoked according to the FTC protocol (Adams *et al.*, 1987). Differences in the tobacco blend may be one explanation for this discrepancy and Canadian cigarettes are made predominantly from flue-cured tobacco.

The average yields of total particulate matter and nicotine in sidestream smoke generated by the machine-smoking of two cigarette brands that are popular among smokers in India (one filter-tipped and one untipped) were 16.51 and 0.9 mg per cigarette, respectively (Pakhale & Maru, 1998). In the sidestream smoke from bidis, these concentrations were 5.5 mg total particulate matter and 0.25 mg nicotine. The sidestream smoke released from chuttas contained 19.8 mg total particulate matter and 2.07 mg nicotine per unit. In all Indian products, the emissions of total particulate matter were much higher in mainstream smoke than in sidestream smoke, which is demonstrated clearly by the ratio of sidestream smoke to mainstream smoke which ranged from 0.13 to 0.49 (see also the monograph on tobacco smoke, Section 1.2.7). This is a modified, more intensive smoking standard (two puffs per minute instead of one) used because of the poor burning properties of the tobacco in Indian products.

In the 1999 Massachusetts Benchmark Study (Borgerding *et al.*, 2000), a subset of 12 brands was analysed for the chemical composition of the sidestream smoke that was generated by machine-smoking using the 'more intense' Massachusetts method. The data obtained are summarized in Table 1.3. The concentrations of the constituents of the sidestream smoke determined in this study differed significantly from those obtained using the standard FTC method that had been reported by Adams *et al.* (1987): the yields of CO, ammonia and benzo[*a*]pyrene were higher, those of tar and catechol were of the same order of magnitude and those of NNN and NNK were significantly lower. NNN and NNK levels were even lower than those measured in the mainstream smoke generated by the same intense machine-smoking method. The values obtained by the Massachusetts Study for some gaseous compounds such as 1,3-butadiene, acrolein, isoprene, benzene and toluene were also far below those obtained by machine-smoking using the FTC method (Brunnemann *et al.*, 1990; Table 1.4). For the 12 commercial cigarette brands tested by the Massachusetts puffing parameters, the highest median sidestream smoke/mainstream smoke ratios in the Massachusetts study were obtained for ammonia (ratio, 147),

Table 1.3. Average values of 44 smoke constituents in the sidestream smoke of 12 commercial cigarette brands assayed in the 1999 Massachusetts Benchmark Study using Massachusetts smoking parameters

Constituent	Unit	Range	SS/MS ratio ^a
Ammonia	mg/cig.	4.0–6.6	147
1-Aminonaphthalene	ng/cig.	165.8–273.9	7.10
2-Aminonaphthalene	ng/cig.	113.5–171.6	8.83
3-Aminobiphenyl	ng/cig.	28.0–42.2	10.83
4-Aminobiphenyl	ng/cig.	20.8–31.8	5.41
Benzo[<i>a</i>]pyrene	ng/cig.	51.8–94.5	3.22
Formaldehyde	µg/cig.	540.4–967.5	14.78
Acetaldehyde	µg/cig.	1683.7–2586.8	1.31
Acetone	µg/cig.	811.3–1204.8	1.52
Acrolein	µg/cig.	342.1–522.7	2.53
Propionaldehyde	µg/cig.	151.8–267.6	1.06
Crotonaldehyde	µg/cig.	62.2–121.8	1.95
Methyl ethyl ketone	µg/cig.	184.5–332.6	1.49
Butyraldehyde	µg/cig.	138.0–244.9	2.68
Hydrogen cyanide	mg/cig.	0.19–0.35	0.77
Mercury	ng/cig.	5.2–13.7	1.09
Nickel	ng/cig.	ND–NQ	
Chromium	ng/cig.	ND–ND	
Cadmium	ng/cig.	122–265	1.47
Arsenic	ng/cig.	3.5–26.5	1.51
Selenium	ng/cig.	ND–ND	
Lead	ng/cig.	2.7–6.6	0.09
Nitric oxide	mg/cig.	1.0–1.6	2.79
Carbon monoxide	mg/cig.	31.5–54.1	1.87
‘Tar’	mg/cig.	10.5–34.4	0.91
Nicotine	mg/cig.	1.9–5.3	2.31
Pyridine	µg/cig.	195.7–320.7	16.08
Quinoline	µg/cig.	9.0–20.5	12.09
Phenol	µg/cig.	121.3–323.8	9.01
Catechol	µg/cig.	64.5–107.0	0.85
Hydroquinone	µg/cig.	49.8–134.1	0.94
Resorcinol	µg/cig.	ND–5.1	
<i>meta</i> -Cresol + <i>para</i> -Cresol ^b	µg/cig.	40.9–113.2	4.36
<i>ortho</i> -Cresol	µg/cig.	12.4–45.9	4.15 ^c
NNN	ng/cig.	69.8–115.2	0.43
NNK	ng/cig.	50.7–95.7	0.40
NAT	ng/cig.	38.4–73.4	0.26
NAB	ng/cig.	11.9–17.8	0.55
1,3-Butadiene	µg/cig.	81.3–134.7	1.30

Table 1.3 (contd)

Constituent	Unit	Range	SS/MS ratio ^a
Isoprene	µg/cig.	743.2–1162.8	1.33
Acrylonitrile	µg/cig.	24.1–43.9	1.27
Benzene	µg/cig.	70.7–134.3	1.07
Toluene	µg/cig.	134.9–238.6	1.27
Styrene	µg/cig.	23.2–46.1	2.60

From Borgerding *et al.* (2000)

SS, sidestream smoke; MS, mainstream smoke; NNN, *N*'-nitrosonornicotine; NNK, 4-(*N*-nitrosomethylamino)-1-(3-pyridyl)-1-butanone; NAT, *N*'-nitrosoanatabine; NAB, *N*'-nitrosoanabasine; ND, not detected; limit of detection for chromium, 8 ng/cigarette; for selenium, 5 ng/cigarette; for resorcinol, 0.6 µg/cigarette; for nickel, 6.8 ng/cigarette; NQ, not quantifiable; limit of quantification for nickel, 10 ng/cigarette

^a Median value for the sidestream/mainstream smoke ratios for the 12 commercial cigarette brands

^b Reported together

Table 1.4. Concentrations of selected gas-phase compounds in sidestream smoke of commercial cigarettes

Compound	Federal Trade Commission method (Adams <i>et al.</i> , 1987; Brunnemann <i>et al.</i> , 1990)	1999 Massachusetts Benchmark Study (Borgerding <i>et al.</i> , 2000)
NNN (ng/cig.)	185–857	70–115
NNK (ng/cig.)	386–1444	51–96
1,3-Butadiene (µg/cig.)	205–250 ^a	81–135
Acrolein (µg/cig.)	723–1000	342–523
Isoprene (µg/cig.)	4380–6450	743–1163
Benzene (µg/cig.)	345–529	71–134
Toluene (µg/cig.)	758–1060	135–239

NNN, *N*'-nitrosonornicotine; NNK, 4-(*N*-nitrosomethylamino)-1-(3-pyridyl)-1-butanone)

^a 400 µg 1,3-butadiene measured in the sidestream smoke collected after emission into an environmental chamber (Löfroth, 1989)

Table 1.5. Yields of IARC carcinogens in regular-sized Canadian cigarettes. Comparison of International Organization for Standardization (ISO)^a and Health Canada (HC)^b machine-smoking parameters^c

Compound	ISO smoking parameters						
	Regular (full flavour)	Light	Extra light	Ultra light	ISO/ISO regular/light	ISO/ISO regular/extra light	ISO/ISO regular/ultra light
<i>IARC Group 1 carcinogens</i>							
Benzene (µg/cig.)	222.0	250.0	260.0	296.0*	0.9	0.9	0.8*
Cadmium (ng/cig.)	438.0	484.0	502.0*	627.0*	0.9	0.9*	0.7*
2-Aminonaphthalene (ng/cig.)	157.0	147.0	175.0	186.0	1.1	0.9	0.8
Nickel (ng/cig.)	34.3	45.1	74.4*	73.0*	0.8	0.5*	0.5*
Chromium (ng/cig.)	61.0	62.0	121*	82.9*	1.0	0.5*	0.7*
Arsenic (ng/cig.)	ND	NQ	ND	ND			
4-Aminobiphenyl (ng/cig.)	22.1	19.5	21.0	21.2	1.1	1.1	1.0
<i>IARC Group 2A carcinogens</i>							
Formaldehyde (µg/cig.)	378.0	326.0	414.0	431.0	1.2	0.9	0.9
1,3-Butadiene (µg/cig.)	196.0	185.0	264.0	299.0	1.1	0.7	0.7
Benzo[a]pyrene (ng/cig.)	48.8	98.3	92.2	113.0	0.5	0.5	0.4
<i>IARC Group 2B carcinogens</i>							
Acetaldehyde (µg/cig.)	1416.0	1454.0	1449.0	1492.0	1.0	1.0	0.9
Isoprene (µg/cig.)	1043.0	1164.0	1060.0	1172.0	0.9	1.0	0.9
Catechol (µg/cig.)	130.0	117.0	149.0	148.0	1.1	0.9	0.9
Acrylonitrile (µg/cig.)	78.6	85.6	74.1	81.8	0.9	1.1	1.0
Styrene (µg/cig.)	74.0	84.7	87.5	108.0*	0.9	0.8	0.7*
NNK (ng/cig.)	95.2	153.4	38.3	34.7	0.6	2.5	2.7
NNN (ng/cig.)	23.3	53.9	43.7	45.2	0.4	0.5	0.5
Lead (ng/cig.)	54.8	39.4	22.3	18.5	1.4	2.5	3.0

Source: Government of British Columbia (2003)

NNN, *N'*-nitrosornicotine; NNK, 4-(*N*-nitrosomethylamino)-1-(3-pyridyl)-1-butanone; ND, not detectable; NQ, not quantifiable

^aISO smoking parameters: 35 mL puff in 2 sec, interval 60 sec, ventilation holes not blocked

^bHC: Health Canada smoking parameters: 56 mL puff in 2 sec, interval 26 sec, ventilation holes fully blocked

^cReporting period: year 1999

* Changed according to personal communication with B. Beech, Health Canada

3-aminobiphenyl (ratio, 10.8), formaldehyde (ratio, 14.8), pyridine (ratio, 16.1) and quinoline (ratio, 12.1).

Often, conflicting results concerning the phase distribution of individual constituents and poor agreement between laboratories for quantitation of sidestream emissions are attributed to different methods used for smoke generation and collection.

Table 1.5 shows the yields of IARC carcinogens in sidestream smoke generated under standard International Organization for Standardization (ISO) and the more intense Health Canada methods, of four popular regular-size Canadian cigarette brands.

On the basis of their mainstream smoke tar yields as measured by the ISO/FTC machine-smoking method, the four cigarette brands may be classified as 'full flavour', 'light', 'extra light' and 'ultra light'. British Columbia has established the Tobacco Testing and Disclosure Regulation and became the first jurisdiction in the world to require Canadian tobacco manufacturers to disclose on a brand-by-brand basis the contents of cigarettes and tobacco and the levels of potentially toxic chemicals in tobacco smoke.

Table 1.5. (contd)

HC smoking parameters										
Regular (full flavour)	Light	Extra light	Ultra light	HC/HC regular/ light	HC/HC regular/ extra light	HC/HC regular/ ultra light	HC/ISO Regular	HC/ISO Light	HC/ISO Extra light	HC/ISO Ultra light
98.1	140.0	141.0	158.0	0.7	0.7	0.6	0.4	0.6	0.5	0.5*
256.0	276.0	282.0	355.0	0.9	0.9	0.7	0.6	0.6	0.5*	0.5*
113.0	71.1	112.0	102.0	1.6	1.0	1.1	0.7	0.5	0.6	0.5
17.6	49.3	35.5	34.8	0.4	0.5	0.5	0.5	1.1	0.5*	0.5*
47.1	57.2	54.6	69.4	0.8	0.9	0.7	0.8	0.9	0.5*	0.8*
ND	ND	ND	ND							
16.3	12.5	17.2	15.1	1.3	0.9	1.1	0.7	0.6	0.8	0.7
311.0	208.0	256.0	327.0	1.5	1.2	1.0	0.8	0.6	0.6	0.8
120.0	109.0	168.0	175.0	1.1	0.7	0.7	0.6	0.6	0.6	0.6
31.9	39.5	41.2	44.0	0.8	0.8	0.7	0.7	0.4	0.4	0.4
1174.0	969.0	1079.0	1277.0	1.2	1.1	0.9	0.8	0.7	0.7	0.9
525.0	818.0	763.0	858.0	0.6	0.7	0.6	0.5	0.7	0.7	0.7
104.0	82.0	96.1	109.0	1.3	1.1	1.0	0.8	0.7	0.6	0.7
41.1	50.1	47.6	51.9	0.8	0.9	0.8	0.5	0.6	0.6	0.6
38.7	61.6	50.8	55.6	0.6	0.8	0.7	0.5	0.7	0.6	0.5*
69.8	116.5	65.6	89.9	0.6	1.1	0.8	0.7	0.8	1.7	2.6
19.3	37.8	24.3	30.1	0.5	0.8	0.6	0.8	0.7	0.6	0.7
40.0	30.1	27.0	24.3	1.3	1.5	1.6	0.7	0.8	1.2	1.3

Among the 44 smoke components reported by the manufacturers on a yearly basis, there are seven IARC group 1 carcinogens (benzene, cadmium, 2-aminonaphthalene, nickel, chromium, arsenic and 4-aminobiphenyl), three IARC group 2A carcinogens (formaldehyde, 1,3-butadiene and benzo[*a*]pyrene) and eight IARC group 2B carcinogens (acetaldehyde, isoprene, catechol, acrylonitrile, styrene, NNK, NNN and lead).

Of the seven IARC group 1 carcinogens, arsenic yields in sidestream smoke were below the detection limits of both the ISO and Health Canada smoking methods. In general, yields of the six other IARC group 1 carcinogens in sidestream smoke were higher when measured by the ISO than by the Health Canada smoking method. The ISO and Health Canada methods gave similar yields for nickel and chromium in the 'light' cigarette and for chromium in the 'ultra light' cigarette.

For most IARC group 2A and 2B carcinogens, the yields in sidestream smoke measured by the Health Canada method were 40–80% of corresponding yields measured by the ISO method. Exceptionally, for NNK and lead the yields measured by the Health Canada method were higher than the yields measured by the ISO method, but only for the 'extra light' and 'ultra light' brands. The yields of NNK measured by the Health Canada method were up to 2.6-fold higher than the yields measured by the ISO method (Government of British Columbia, 2003).

Table 1.5 also allows comparisons of sidestream smoke yields between the brands. There is no significant difference between the total sidestream smoke yields of IARC group 1 carcinogens of 'full flavour' and 'light', extra light and ultra light cigarettes when measured by either the ISO or Health Canada methods.

The data in Tables 1.3 and 1.5 suggest that during more intense smoking (as employed by the Massachusetts and Health Canada methods: i.e. larger puffs, shorter interval between puffs and partial or complete blockage of ventilation holes), smaller quantities of tobacco are burned during the smouldering of the cigarette, thus affecting the emissions of toxins in sidestream smoke. Therefore, the real-life contribution of sidestream smoke to the overall concentrations of selected components of secondhand tobacco smoke may have been overestimated in the past because most smokers draw smoke from their cigarettes with an intensity more similar to that of the Massachusetts or Health Canada methods than the FTC machine-smoking method (Djordjevic *et al.*, 2000). This concept needs to be investigated more thoroughly, especially in view of the finding that an increase in puff volume from 17.5 mL to 50 mL and in filter ventilation from 0 to 83% failed to reduce the levels of tar, CO and nicotine in the sidestream smoke, whereas the yields in mainstream smoke and subsequently the ratios of sidestream smoke to mainstream smoke changed significantly (Guerin *et al.*, 1987).

In addition to the constituents listed in Table 1.3, some further constituents have been quantified in sidestream smoke since the publication of the 1986 *IARC Monograph*. These are NDMA (up to 735 ng per cigarette), *N*-nitrosopiperidine (NPIP, 19.8 ng) and NPYR (up to 234 ng) (Adams *et al.*, 1987); and volatile hydrocarbons, e.g. ethene up to 1200 µg, propene up to 1300 µg, butenes up to 900 µg and pentenes up to 2100 µg. The sidestream smoke emissions of various unsaturated gaseous hydrocarbons were 3–30 times those reported for the mainstream smoke emissions. These compounds constitute a potential health risk as they are metabolized *in vivo* to reactive genotoxic epoxides (Löfroth *et al.*, 1987; Löfroth, 1989). High-molecular-weight *n*-alkanes (C₂₇ [66–86.5 µg per cigarette], C₂₉ [28–39 µg per cigarette] C₃₁ [148–197 µg per cigarette], C₃₃ [43.5–62 µg per cigarette]) were also quantified in the sidestream smoke of commercial cigarettes (Ramsey *et al.*, 1990).

The co-mutagenic beta-carbolines, norharman and harman, were quantified in the sidestream smoke condensates of some Japanese cigarette brands. The concentrations per cigarette were 4.1–9.0 µg for norharman and 2.1–3.0 µg for harman (Totsuka *et al.*, 1999).

1.2 Exposure

Exposure to secondhand smoke can take place in any of the environments where people spend time. A useful conceptual framework for considering exposure to secondhand smoke is offered by the microenvironmental model that describes personal exposure to secondhand smoke as the weighted sum of the concentrations of secondhand smoke in the microenvironments where time is spent and the weights supplied by the time spent in each (Jaakkola & Jaakkola, 1997). A microenvironment is a space, e.g. a room in a

dwelling or an office area, with a relatively uniform concentration of secondhand smoke during the time that is spent in that particular microenvironment. For research purposes and for considering health risks, personal exposure is the most relevant measure for evaluating and projecting risk (Samet & Yang, 2001).

Within the framework of the microenvironmental model, there are several useful indicators of exposure to secondhand smoke, ranging from surrogate indicators to direct measurements of exposure and of biomarkers that reflect dose (Table 1.6). One useful surrogate, and the only indicator available for many countries, is the prevalence rate of smoking among men and women. It provides at least a measure of likelihood of exposure. For the countries of Asia, for example, where smoking rates among men are very high and those among women are low, the prevalence data for men imply that most women are exposed to tobacco smoke at home (Samet & Yang, 2001).

The components of secondhand smoke include a number of irritating and odiferous gaseous components, such as aldehydes. Nonsmokers typically identify the odour of secondhand smoke as annoying, and the odour detection thresholds determined for secondhand smoke is at concentrations that are three or more orders of magnitude lower

Table 1.6. Indicators of exposure to secondhand tobacco smoke

Measure	Indicator
Surrogate measures	Prevalence of smoking in men and women
Indirect measures	Report of secondhand tobacco smoke exposure in the home and in the workplace
	Smoking in the household
	Number of smokers
	Smoking by parent(s)
	Number of cigarettes smoked
	Smoking in the workplace
	Presence of secondhand tobacco smoke
	Number of smokers
Direct measures	Concentration of secondhand tobacco smoke components
	Nicotine
	Respirable particles
	Other markers
	Biomarker concentrations
	Cotinine
	Carboxyhaemoglobin

From Samet & Yang (2001)

than the secondhand smoke concentrations measured in field settings and correspond to a fresh air dilution volume $> 19\,000\text{ m}^3$ per cigarette (Junker *et al.*, 2001).

The indirect measures listed in Table 1.6 are generally obtained by questionnaires. These measures include self-reported exposure and descriptions of the source of secondhand smoke (e.g. smoking), in relevant microenvironments, most often the home and workplace. Self-reported exposure to secondhand smoke is a useful indicator of being exposed, although questionnaire-based reports of intensity of exposure are of uncertain validity. Questionnaires have been used to ascertain the prevalence of passive smoking; some of these have included questions directly related to the WHO definition of passive smoking: i.e. exposure for at least 15 minutes per day on more than 1 day per week (Samet & Yang, 2001).

Questionnaires have been used widely for research purposes to characterize smoking (the source of secondhand smoke) in the home and work environments. A simple mass-balance model gives the concentration of secondhand smoke as reflecting the rate of its generation, i.e., the number of smokers and of cigarettes smoked, the volume of the space into which the smoke is released, and the rate of smoke removal by either air exchange or air cleaning (Ott, 1999). Information on smoking can be collected readily by adults within the household (the source term), although reports of numbers of cigarettes smoked in the home are probably less valid than exposure predicted using the mass balance model. For workplace environments, smoking can be reported by co-workers, although the complexity of some workplace environments may preclude the determination of the numbers of smokers in the work area or the numbers of cigarettes smoked. The other determinants of secondhand smoke concentration, namely, room volume and air exchange are not readily determined by questionnaire and are assessed only for specific research purposes (Samet & Yang, 2001).

The direct measures of exposure to secondhand smoke include measurement of the concentrations of components of secondhand smoke in the air and of the levels of secondhand smoke biomarker in biological specimens. Using the microenvironmental model, researchers can estimate exposure to secondhand smoke by measuring the concentration of secondhand smoke in the home, workplace, or other environments and then combining the data on concentrations with information on the time spent in the microenvironments where exposure took place. For example, to estimate exposure to secondhand smoke in the home, the concentration of a marker in the air, e.g. nicotine, would be measured and the time spent in the home would be assessed, possibly using a time-activity diary in which information on all locations where time is spent is collected (Samet & Yang, 2001).

Because cigarette smoke is a complex mixture, exposure assessment depends on the choice of a suitable marker compound that is found in both mainstream smoke and secondhand tobacco smoke. No compound has a consistent ratio with all other components. Therefore, the choice of marker can affect the estimate of exposure.

The selection of a particular secondhand smoke component for monitoring is largely based on technological feasibility. Air can be sampled either actively, using a pump that passes air through a filter or a sorbent, or passively, using a badge that operates on the

principle of diffusion. A number of secondhand smoke components have been proposed as potential indicators; these include small particles in the respirable size range and the gases, nicotine, which is present in the vapour phase in secondhand smoke, and carbon monoxide. Other proposed indicators include more specific measures of particles and other gaseous components (Guerin *et al.*, 1992; Jenkins & Counts, 1999). The most widely studied components have been respirable particles, which are sampled actively with a pump and filter, and nicotine, which can be collected using either active or passive sampling methods. The respirable particles in indoor air have sources other than active smoking and are nonspecific indicators of secondhand smoke; nicotine in air, by contrast, is highly specific because smoking is its only source (Jenkins *et al.*, 2000). Nicotine concentration can be measured readily using a passive filter badge, which is sufficiently small to be worn by a child or an adult or to be placed in a room (Hammond, 1999).

Biomarkers of exposure are compounds that can be measured in biological materials such as blood, urine or saliva. Cotinine, a metabolite of nicotine, is a highly specific indicator of exposure to secondhand smoke in nonsmokers (Benowitz, 1999). Some foods contain small amounts of nicotine, but for most persons cotinine level offers a highly specific and sensitive indicator of exposure to secondhand smoke (Benowitz, 1999). In nonsmokers, the half-life of cotinine is about 20 h; it therefore provides a measure of exposure to secondhand smoke over several days. It is an integrative measure that reflects exposure to secondhand smoke in all environments where time has been spent. Cotinine can be readily measured in blood, urine and even saliva with either radioimmunoassay or chromatography. New methods for analysis extend the sensitivity to extremely low levels (Benowitz, 1996; Benowitz, 1999). Alternatives to nicotine as a tobacco-specific marker substance are few. One such compound is 3-ethenylpyridine (also called 3-vinyl pyridine); it is a pyrolysis product of nicotine degradation during smoking present almost exclusively in the vapour phase of tobacco smoke. It has been employed to a small extent for measuring the concentrations of secondhand tobacco smoke in air (Heavner *et al.*, 1995; Hodgson *et al.*, 1996; Scherer *et al.*, 2000; Vainiotalo *et al.*, 2001), and a correlation between nicotine and 3-ethenylpyridine has been reported in some studies (Jenkins *et al.*, 1996; Moschandreas & Vuilleumier, 1999; Hyvärinen *et al.*, 2000). 3-Ethenylpyridine, solanesol and ultraviolet-absorbing particulate matter as markers of secondhand smoke have been suggested as being potentially better correlated with other constituents of secondhand smoke than nicotine and respirable particles (Hodgson *et al.*, 1996; Jenkins *et al.*, 1996). There are however many fewer data available on measurements using other tobacco-specific marker compounds than those based on air nicotine.

1.2.1 *Measurements of nicotine and particulate matter in indoor air*

The report of the US Environmental Protection Agency (US EPA, 1992) summarizes over 25 separate studies that reported concentrations of nicotine in air measured in more than 100 different indoor microenvironments. Hammond (1999) also reported an extensive survey of the concentrations of nicotine in air. Based on the large numbers of

measurements made in various indoor environments in the USA between 1957 and 1991, the average concentrations of nicotine in air showed about 100-fold variation, i.e. from 0.3–30 $\mu\text{g}/\text{m}^3$ (US EPA, 1992). The average concentrations in homes with one or more smokers typically ranged from 2 to 10 $\mu\text{g}/\text{m}^3$, with the highest averages being up to 14 $\mu\text{g}/\text{m}^3$. Data from the mid 1970s until 1991 indicate that the nicotine concentrations in offices were similar to those measured in homes, with a large overlap in the range of air concentrations for the two types of environment. The maximum levels of nicotine, however, were considerably higher in offices than in domestic environments (US EPA, 1992; California EPA, 1997). In studies using controlled and field conditions, the concentrations of nicotine in air were found to increase as a function of the number of smokers present and the number of cigarettes consumed (US EPA, 1992).

Jenkins *et al.* (1996) studied exposure to secondhand tobacco smoke in 16 cities in the USA by sampling personal breathing zone air from about 100 nonsmokers in each city. The demographics of the study subjects were comparable with the population of the USA in general, although more women than men participated in the study. The mean 24-h time-weighted average concentration of nicotine was 3.27 $\mu\text{g}/\text{m}^3$ for those exposed to secondhand tobacco smoke both at work and away from work, 1.41 $\mu\text{g}/\text{m}^3$ for those only exposed away from work and 0.69 $\mu\text{g}/\text{m}^3$ for those who were exposed only at work. The mean 24-h time-weighted average concentration of nicotine in air measured by personal monitoring, for those who were not exposed to secondhand tobacco smoke was 0.05 $\mu\text{g}/\text{m}^3$ (Jenkins *et al.*, 1996).

Personal exposure to particulate matter associated with secondhand tobacco smoke was determined using the set of specific markers such as respirable suspended particles, fluorescent particulate matter and solanesol-particulate matter. The ranges of mean concentrations of these particles for workers exposed to secondhand smoke in 11 countries were: respirable suspended particles, from 24 to 112 $\mu\text{g}/\text{m}^3$; fluorescent particulate matter, from 5.7 to 57 $\mu\text{g}/\text{m}^3$; and solanesol-particulate matter, from 3.6 to 64 $\mu\text{g}/\text{m}^3$ (Jenkins *et al.*, 2000). By measuring the levels of solanesol-particulate matter and nicotine, the exposure to secondhand tobacco smoke of office workers living and working with smokers was determined to be higher in winter than in summer (median 24-h time-weighted average concentrations, 25 μg versus 2.4 μg solanesol-particulate matter and 1.3 μg versus 0.26 μg nicotine, respectively) (Phillips & Bentley, 2001).

1.2.2 *Population-based measurements of exposure*

Most population-based estimates of exposure to secondhand tobacco smoke have been obtained from self-reports. When measuring exposure to secondhand smoke in indoor areas, nicotine or respirable suspended particles can be measured in air sampled using personal monitors. In a few studies, biomarkers such as cotinine have been measured in physiological fluids.

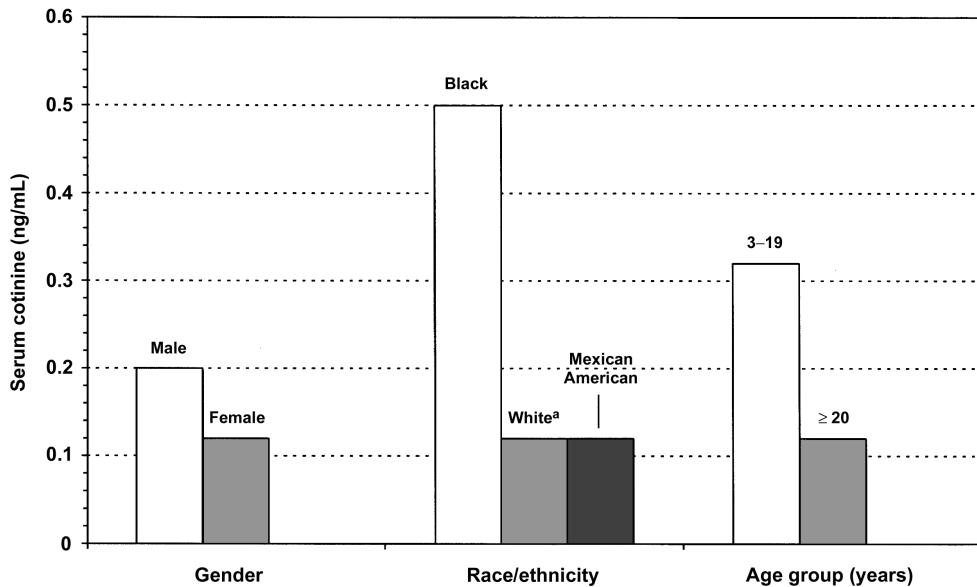
(a) *Adults*

Some studies suggest that exposure to secondhand tobacco smoke is related to occupation and socioeconomic status, and that higher exposure is more common among adults employed in blue-collar jobs, service occupations and poorly paid jobs and among the less well educated (Gerlach *et al.*, 1997; Curtin *et al.*, 1998; Whitlock *et al.*, 1998). Exposure to secondhand tobacco smoke may also be higher among racial and ethnic minority groups in areas of the USA, although it is unclear if this is due to different socioeconomic status (Gerlach *et al.*, 1997).

Relatively few data are available on the prevalence of nonsmokers' exposure to secondhand tobacco smoke on a population basis, using biomarkers. Survey data from a study in the USA in 1988–91 showed that 37% of adult non-tobacco users lived in a home with at least one smoker or reported exposure to secondhand tobacco smoke at work, whereas serum cotinine levels indicated more widespread exposure to nicotine. Of all the non-tobacco users surveyed including children, 88% had detectable serum cotinine levels, indicating widespread exposure to secondhand tobacco smoke in residents of the USA (Pirkle *et al.*, 1996). Data were recently published on the serum cotinine levels measured in 2263 nonsmokers in 12 locations across the USA (Figure 1.1) (CDC, 2001). As reported previously (Pirkle *et al.*, 1996), exposure to secondhand tobacco smoke tended to be higher among men than among women. Among racial/ethnic groups, blacks had the highest cotinine levels. People younger than 20 years of age had higher cotinine levels than those aged 20 years and older.

Table 1.7 summarizes the data obtained from a number of recent population-based studies that used questionnaires to characterize exposure. Some of these studies were national in scope, e.g. the national samples in Australia, China and the USA, whereas others were from single states or specific localities. Several of the studies incorporated cotinine as a biomarker. Unfortunately, few data are available from developing countries (Samet & Yang, 2001). In a case-control study of lung cancer and exposure to secondhand smoke in 12 centres from seven European countries, 1542 control subjects up to 74 years of age were interviewed between 1988 and 1994 about their exposure to secondhand smoke. Exposure of adults to secondhand smoke from a spouse who smoked was reported by 45% of the subjects (including 2% who were exposed to smoke from cigars or pipes only); an additional 8% were not exposed to spousal smoke, but reported exposure to secondhand smoke produced by cohabitants other than spouses. Exposure to secondhand smoke in the workplace was reported by 71% of men and 46% of women. Combined exposure to secondhand smoke both from the spouse and at the workplace was reported by 78%. Exposure to secondhand smoke in vehicles was reported by 20% and exposure in public indoor settings such as restaurants was reported by 29% (Boffetta *et al.*, 1998). Recent data from Finland illustrate trends in self-reported exposure to secondhand tobacco smoke at work and at home over a 15-year period. In 1985, 25% of employed nonsmoking men and 15% of employed nonsmoking women were exposed to

Figure 1.1. 75th percentile of serum cotinine concentrations for the US nonsmoking population aged 3 years and older, National Health and Nutrition Examination Survey, 1999



From CDC (2001)

^a Includes other racial/ethnic groups

secondhand tobacco smoke at work. In contrast, the figures for exposure to secondhand smoke in 2000 were 8% of men and 4% of women (Jousilahti & Helakorpi, 2002).

In a 1993 review of existing studies, Siegel (1993) noted wide variation in the concentrations of secondhand tobacco smoke by location when measured by weighted mean levels of nicotine in the ambient air of offices (4.1 $\mu\text{g}/\text{m}^3$), restaurants (6.5 $\mu\text{g}/\text{m}^3$), bars (19.7 $\mu\text{g}/\text{m}^3$) and dwellings with at least one smoker (4.3 $\mu\text{g}/\text{m}^3$).

The recently published study in the USA of the median serum cotinine concentration measured in nonsmokers aged 3 years and older found a 75% decrease over the period 1991–99 (CDC, 2001), suggesting positive effects of policies for cleaner indoor air.

(b) Children

Because the home is a predominant location for smoking, children are exposed to tobacco smoke as they go about their daily lives, i.e. while eating, playing and even sleeping. The exposure at home may be added to exposure at school and in vehicles. Consequently, in many countries, children cannot avoid inhaling tobacco smoke (Samet & Yang, 2001).

Table 1.7. Prevalence of exposure to secondhand tobacco smoke — population-based studies

Reference	Study design and population	Results
Europe		
Somerville <i>et al.</i> (1988)	Cross-sectional study; 4337 children aged 5–11 years in England and 766 in Scotland, from the 1982 National Health Interview Survey on Child Health in the United Kingdom	Prevalence = 42% in England and 60% in Scotland
Dijkstra <i>et al.</i> (1990)	Cohort study; nonsmoking children aged 6–12 years over a 2-year period, in the Netherlands	Prevalence = 66%
Jaakkola <i>et al.</i> (1994)	Population-based cross-sectional study; random sample of 1003 children aged 1–6 years in Espoo, Finland	25.2% reported exposure to secondhand tobacco smoke at home; 74.8% of children did not, assessed by parent-completed questionnaire.
Brenner <i>et al.</i> (1997)	Cross-sectional study; survey of 974 predominantly blue-collar employees of a south German metal company	> 60% of nonsmoking blue collar workers reported being exposed to secondhand smoke at work; 52% of nonsmoking white collar workers exposed if smoking allowed in immediate work area, and 18% if smoking not allowed
Boffetta <i>et al.</i> (1998)	Case-control study of lung cancer and exposure to secondhand tobacco smoke in 12 centres from seven European countries, 1542 control subjects up to 74 years of age were interviewed between 1988 and 1994 about exposure to secondhand tobacco smoke	Prevalence during childhood was 66%. Prevalence in adulthood (spousal smoke) was 45% (including 2% with exposure to cigar or pipe smoke only). In addition, 8% who were not exposed to spousal smoke were exposed to secondhand tobacco smoke produced by cohabitants other than spouse. Prevalence in the workplace was 71% among men and 46% among women. Combined prevalence from the spouse and at the workplace was 78%. Prevalence of exposure to secondhand tobacco smoke in vehicles was 20% and in public indoor settings such as restaurants, it was 29%.

Table 1.7 (contd)

Reference	Study design and population	Results
Lund <i>et al.</i> (1998)	Children born in 1992; descriptive study of exposure to secondhand tobacco smoke at home in 3547 households in Denmark, Finland, Iceland, Norway and Sweden in 1995–96	Prevalence of weekly exposure was 47% in Denmark, 7% in Finland, 46% in Iceland, 32% in Norway and 15% in Sweden.
Jousilahti & Helakorpi (2002)	A cohort of 58 721 men and women aged 15 to 64 years was followed-up by annual questionnaires on exposure to secondhand smoke at work and at home between 1985 and 2000 in Finland.	In the middle of the 1980s, about a quarter of employed nonsmoking men and 15% of nonsmoking women were exposed for at least 1 hour daily to environmental tobacco smoke at work. In 2000, the proportions were 7.9% and 4.4%, respectively. Exposure to environmental tobacco smoke at home also decreased slightly. In 2000, 14.3% of the nonsmoking men and 13% of the nonsmoking women aged 15 to 64 years were exposed to secondhand smoke either at work or at home.
America		
Coultas <i>et al.</i> (1987)	Cross-sectional study; 2029 Hispanic children and adults in New Mexico (1360 nonsmokers and ex-smokers also had salivary cotinine measured)	Prevalence = 39% ≥ 18 years; 48%, 13–17 years; 45%, 6–12 years, and 54% < 5 years. Mean salivary cotinine concentrations = 0 to 6 ng/mL; 35% of members of nonsmoking households had detectable levels of cotinine.
Greenberg <i>et al.</i> (1989)	Questionnaire-based cross-sectional study; mothers of 433 infants from a representative population of healthy neonates from 1986–87 in North Carolina	55% lived in a household with at least one smoker; 42% of infants had been exposed during the week preceding data collection; cotinine was detected in 60% of urine samples (median = 121 ng/mg creatinine).
Chilmonczyk <i>et al.</i> (1990)	Cross-sectional study; 518 infants aged 6–8 weeks receiving well-child care in the offices of private physicians in greater Portland, Maine	91% of infants living in households where only the mother smoked (43 households) had urinary cotinine levels ≥ 10 µg/L; 8% of infants living in households where no smoking was reported (305 households) had urinary cotinine levels ≥ 10 µg/L.

Table 1.7 (contd)

Reference	Study design and population	Results
Overpeck & Moss (1991)	Cross-sectional study; sample of 5356 children ≤ 5 years of age from the National Health Interview Survey in 1988	Approximately 50% of all US children ≤ 5 years of age exposed to prenatal maternal smoking and/or secondhand smoke from household members after birth; 28% were exposed both prenatally and postnatally, 21% only after birth and 1.2% only prenatally.
Borland <i>et al.</i> (1992)	Cross-sectional study; sample of 7301 nonsmokers from the larger study of Burns & Pierce (1992)	31.3% of nonsmoking workers reported exposure at work ≥ 1 time in the preceding 2 weeks; 35.8% males vs 22.9% females; 41.9% < 25 years vs 26.4% for older workers; 43.1% with < 12 years of education vs 18.6% with ≥ 16 years of college education
Burns & Pierce (1992)	Cross-sectional study; head of household in 32 135 homes in California, contacted by stratified random-digit dialling from June 1990 to July 1991	32.2% of children aged 5–11 years and 36.5% of adolescents aged 12–17 years were exposed at home.
Jenkins <i>et al.</i> (1992)	Cross-sectional study; telephone interviews with 1579 English-speaking adults and 183 adolescents (12–17 years of age) from October 1987 to September 1988 in California	46% of nonsmokers were exposed during the day: 43% of adult nonsmokers and 64% of adolescent nonsmokers. Exposure most frequently occurred at home, in restaurants or in cars. The average duration of exposure was longest in workplaces.
Jenkins <i>et al.</i> (1992); Lum (1994)	Cross-sectional study; same population as described above and additional 1200 children aged ≤ 11 years (< 8 years old with a parent or guardian) from April 1989 to February 1990 in California	Prevalence of exposure among smokers and nonsmokers = 61% for adults and 70% for adolescents during the day; 35% to 45% of children, infants, and preschoolers were reported to be exposed to secondhand smoke; average duration = 3.5 h.
Pierce <i>et al.</i> (1994)	Cross-sectional study; using the California Adult Tobacco Surveys in 1990, 1992 and 1993 with 8224 to 30 716 adults 18 years and older and 1789 to 5531 teenagers 12–17 years of age interviewed	15.1% smoked prior to pregnancy and of these, 37.5% quit after the pregnancy (between 1988 and 1992, 9.4% of women smoked during pregnancy).

Table 1.7 (contd)

Reference	Study design and population	Results
Pletsch (1994)	Cross-sectional study; 4256 Hispanic women aged 12–49 years who participated in the Hispanic Health and Nutrition Examination Survey (HHANES) from 1982 to 1984	Age-specific household exposure for nonsmokers was 31%–62% for Mexican-Americans, 22%–59% for Puerto Ricans and 40%–53% for Cuban-Americans; 59% of Puerto Rican and 62% of Cuban-American adolescents had high levels of exposure.
Thompson <i>et al.</i> (1995)	Cross-sectional study; 20 801 US employees from 114 work sites	52.4% of respondents reported being exposed to secondhand tobacco smoke at work
Kurtz <i>et al.</i> (1996)	Questionnaire-based cross-sectional survey; sample of 675 African-American students enrolled in grades 5–12 in an urban public school district in Detroit, Michigan	Smoking rates were higher among students with parents who smoked; 48% reported paternal smoking; 46% reported maternal smoking.
Mannino <i>et al.</i> (1996)	17 448 children aged 1–10 years from 1991 US National Health Interview Survey	41% of children with lower socioeconomic status experienced daily exposure at home; 21% of children with higher socioeconomic status experienced daily exposure at home.
Pirkle <i>et al.</i> (1996)	Cross-sectional study; 9744 adults aged 17 years or older from the NHANES III Study, 1988–91	Prevalence for males was 43.5% and for females, 32.9%; 87.9% had detectable serum cotinine levels.
Stamatakis <i>et al.</i> (2002)	Cross-sectional study of ethnically diverse non-smoking women, aged 40 years and older, across the United States ($n = 2326$)	Exposure to secondhand tobacco smoke at home was associated with being American Indian/Alaska Native (aOR, 1.5; 95% CI, 1.0–2.6). Compared with college graduates, exposure to secondhand tobacco smoke at work was higher among women with some high school education (aOR, 2.8; 95% CI, 1.5–5.3) and high school graduates (aOR, 3.1; 95% CI, 1.9–5.1) and substantially higher for women who worked where smoking was allowed in some (aOR, 15.1; 95% CI, 10.2–22.4) or all (aOR, 44.8; 95% CI, 19.6–102.4) work areas.

Table 1.7 (contd)

Reference	Study design and population	Results
Asia		
Lam <i>et al.</i> (1998)	Questionnaire-based cross-sectional study; sample of 6304 students aged 12–15 years, from 172 classes of 61 schools in Hong Kong	53.1% were living in a household with at least one smoker; 35.2% had only one smoker; 9.5% had two and 2.5% had three or more smokers in the household; 38% of fathers and 3.5% of mothers smoked.
Yang <i>et al.</i> (1999)	Cross-sectional study; 120 298 records (63 793 males and 56 020 females) of persons aged 15–69 years from the 1996 National Prevalence Survey of Smoking in China	Of the nonsmoking respondents, 53.5% reported passive exposure to smoke. Over 60% of female nonsmokers between ages 25 and 50 years were passively exposed to tobacco smoke; 71% of participants reported exposure to smoke at home, 32% in public places and 25% in their workplace.
Africa		
Steyn <i>et al.</i> (1997)	Questionnaire-based cross-sectional study; 394 pregnant women attending antenatal services in Johannesburg, Cape Town, Port Elizabeth and Durban in urban South Africa, 1992	Most women who smoked stopped or reduced tobacco use during their pregnancy; 70% lived with at least one smoker in the house.
Australia and New Zealand		
Sherrill <i>et al.</i> (1992)	Cohort study; 634 children aged 9–15 years; New Zealand	Overall prevalence = 40%
Lister & Jorm (1998)	Cross-sectional study; data from the Australian Bureau of Statistics 1989–90 National Health Survey of parents and their children ($n = 4281$) aged 0–4 years; Australia	45% of children lived in households with ≥ 1 current smoker; 29% had a mother who smoked.

Modified from Samet & Yang (2001)

aOR, adjusted odds ratio for sociodemographic characteristics (race, age, education, location and having children in the home), health risk behaviours, and the type of smoking policy in the workplace

Data on the exposure of children to secondhand tobacco smoke are limited. In perhaps the most comprehensive cross-sectional study to date, researchers examined exposure to secondhand tobacco smoke in 17 448 children aged 1–10 years in the USA. Exposure varied considerably according to socioeconomic status: 41% of children of lower socioeconomic status experienced daily exposure to secondhand tobacco smoke in their home, whereas only 21% of children of higher socioeconomic status were exposed daily. Exposure to secondhand tobacco smoke did not vary by race, family size, gender or season (Mannino *et al.*, 1996). In a multicentre study conducted in 1988–94 in seven European countries, exposure to secondhand smoke in childhood was reported by 66% of respondents (Boffetta *et al.*, 1998). Parent-reported exposure to secondhand tobacco smoke among children varied widely across the countries of Denmark, Finland, Iceland, Norway and Sweden (Lund *et al.*, 1998). For example, Finnish parents were more likely than all other Nordic parents to protect their children from secondhand tobacco smoke. Exposure was highest in Denmark and Iceland, where children were exposed in almost half of all households and in nine of ten households with daily smokers. The lack of common metrics for measuring exposure to secondhand tobacco smoke in children is a significant challenge when comparing data between countries.

1.3 Regulations

1.3.1 Policy options

There are a range of options available for the regulation of secondhand tobacco smoke. Of these options, the least effective is designating smoking areas that have no separate ventilation. This option provides only minimal protection to nonsmokers; studies have shown that substantial exposure to secondhand tobacco smoke occurs in workplaces where there are smoking areas without separate ventilation (Repace, 1994). A more effective option is the use of separately ventilated smoking lounges; this protects nonsmokers but is costly and may elevate lung cancer risk in smokers (Siegel *et al.*, 1995). Separately ventilated smoking lounges also endanger workers (e.g. waiting staff) who must enter these areas as part of their job. Finally, the most effective alternative is a totally smoke-free workplace (Brownson *et al.*, 2002).

1.3.2 Prevalence of regulations

In 1985, only about 38% of workers in the USA were employed by firms that had policies restricting smoking (Farrelly *et al.*, 1999). Since that time, smoking restrictions have become increasingly common. According to the 1999 National Worksite Health Promotion Survey, 79% of workplaces with 50 or more employees had formal smoking policies that prohibited smoking or allowed it only in separately ventilated areas (US Department of Health and Human Services, 2000). Data showed that, from 1995–96, 64% of indoor workers in the USA were covered by a total ban on smoking in the workplace.

The proportion of workers in the USA who work in a smoke-free workplace varies considerably by state — from 84% in Maryland and Utah to 40% in Nevada (Burns *et al.*, 2000). There are few systematic data available on the enforcement of existing policies to restrict smoking in the workplace, although existing studies suggest that compliance is likely to be high (Stillman *et al.*, 1990; Wakefield *et al.*, 1996). National data from the USA also suggest that despite some protective laws, workers in blue-collar and service occupations remain much more likely to be exposed to secondhand tobacco smoke in the workplace than other categories of workers (Gerlach *et al.*, 1997). In the USA, hospitals are the only sector that has voluntarily implemented a nationwide smoking ban. This ban was announced in November 1991 and full implementation was required by December 31, 1993. Two years after implementation, the policy was found to have been successful with 96% of hospitals complying with the smoking ban standard (Longo *et al.*, 1995; Brownson *et al.*, 2002).

A recent overview of the legislation restricting smoking at work in different countries has been provided by the American Cancer Society (Corrao *et al.*, 2000) (Table 1.8). Despite using a wide range of already published sources together with Internet searches, no information could be found for many countries. Thus, absence of an entry in this Table does not imply certainty that no legislation exists in a particular country. Conversely, the existence of a law banning or restricting smoking implies nothing about its enforcement. In general, it appears that voluntary restrictions under control of the employer are more common in developing countries than in developed countries (Brownson *et al.*, 2002). A country that relies on voluntary action to ban or restrict smoking may have quite high rates of worker protection. For example, although Australia banned smoking in all federal government workplaces in 1988, it has been left to individual employers to determine their own policies. Yet, in 1999, 71% of indoor workers in the state of Victoria reported a total ban on smoking at their workplace, 21% reported some restrictions on smoking and 8% reported unrestricted smoking (Letcher & Borland, 2000). As in other countries, employees in small Australian workplaces are less likely to report protection, as are workers in particular types of employment (Wakefield *et al.*, 1996; McMaugh & Rissell, 2000). In a survey of indoor workers, 38% of those employed in a restaurant or hotel, 15% of warehouse/store workers and 17% of those working in a workshop or factory reported unrestricted smoking where they worked, compared with only 3% of workers in open-plan offices (Letcher & Borland, 2000). In the United Kingdom, workplace restrictions are also voluntary. In 1997, 40% of the workforce was estimated to be working in a totally smoke-free environment (Freeth, 1998). A survey of 1500 workplaces in Scotland found that 79% of them had designated nonsmoking areas, but only 22% had banned smoking completely (ASH, 2001). Despite the limitations of the data presented in Table 1.9, it is apparent that most countries have some laws that restrict smoking. However, it is very likely that there is considerable need for improvement in protection of workers from secondhand tobacco smoke in almost all countries (Brownson *et al.*, 2002).

A few researchers have begun to examine the prevalence of smoking restrictions in the home because such restrictions are likely to have beneficial effects on the health of

Table 1.8. Variations in workplace smoking policies in selected countries

Country	Type of policy ^a				Comments
	B	R	V	X	
Africa					
Benin	x				Certain workplaces only
Botswana	x				Areas accessible to public, common areas
Mali	x				Public service offices
Nigeria	x				Offices
South Africa	x				Designated smoking areas
Uganda			x		
Tanzania			x		
Zambia	x				
Americas					
Argentina		x			
Barbados			x		
Belize			x		Some private workplaces
Brazil			x		
Canada	x				
Chile	x				Areas accessible to public
Costa Rica	x				
Cuba			x		
Dominican Republic	x				Offices
Ecuador	x				Working areas
El Salvador		x			
Grenada			x		
Guatemala	x				Areas accessible to the public
Honduras	x				
Mexico	x				Working areas
Panama	x				Areas accessible to public
Peru	x				
Trinidad and Tobago			x		
United States				x	State and local levels
Venezuela		x			
Eastern Mediterranean					
Cyprus	x				Private and public
Egypt	x				Enclosed public places
Iran	x				Areas accessible to public
Iraq		x			Administrative measures
Kuwait			x		
Lebanon			x		Upon request by nonsmokers
Morocco	x				Public administration and service offices
Sudan	x				Areas accessible to public
Syria	x				

Table 1.8 (contd)

Country	Type of policy ^a				Comments
	B	R	V	X	
Tunisia			x		
Europe					
Austria	x				Unless appropriate ventilation exists
Belarus		x			
Belgium	x		x		Areas accessible to public and other areas
Bosnia & Herzegovina		x			
Bulgaria	x				Unless nonsmokers give written permission for smoking
Croatia	x				
Czech Republic	x				During work hours when nonsmokers are present
Denmark	x				Voluntary restrictions in private workplaces
Estonia	x				Labour environments
Finland	x				Designated smoking areas
France	x				Except individual offices
Germany			x		
Greece		x			
Hungary	x				Areas accessible to public
Iceland	x				Areas accessible to public
Ireland	x				Areas accessible to public
Israel	x				Except in designated areas
Krygyzstan			x		
Latvia		x			
Lithuania	x				Enclosed areas
Netherlands		x			Public and private
Norway	x				With 2 or more employees
Poland	x				
Portugal		x			
Moldova	x				
Romania			x		
Russia	x				
San Marino	x				
Slovakia		x			
Slovenia		x			
Spain		x			
Sweden		x			
Switzerland		x			
Turkey	x				With 5 or more employees
Ukraine	x				
United Kingdom			x		

Table 1.8 (contd)

Country	Type of policy ^a				Comments
	B	R	V	X	
South-East Asia					
Bangladesh			x		
India		x			
Nepal		x			
Sri Lanka			x		Administrative measures
Thailand	x				
Western Pacific					
Australia			x		
Cambodia	x				Partial ban
China	x				Administrative measures
Cook Islands		x			
Fiji			x		
Japan			x		Guideline, set by Ministry of Labour
Kiribati			x		
Lao People's Democratic Republic			x		
Malaysia	x				Areas accessible to the public
Micronesia		x			
Mongolia	x				Designated smoking areas
New Zealand	x				Common work areas and public areas
Niue			x		
Philippines	x				
Republic of Korea		x			
Samoa			x		
Solomon Islands		x			
Tokelau		x			
Tonga			x		

Brownson *et al.* (2002); adapted from Corrao *et al.* (2000); number of additional countries for which no information is available: Africa = 38; Americas = 15; Eastern Mediterranean = 13; Europe = 15; South-East Asia = 5; Western Pacific = 11.

^aB, smoking is prohibited in workplaces according to national legislation and/or regulations; facilities with a designated smoking area are included in this category if nonsmoking areas must always remain uncontaminated by smoke; R, smoking is restricted, but not prohibited, in workplaces according to national legislation and/or regulations; V, employers voluntarily prohibit or restrict smoking in areas under their management; X, different state and county laws apply.

Table 1.9. Summary of selected studies on the effects of workplace smoking bans and restrictions on exposure to secondhand tobacco smoke

Reference/location	Industry/setting	Sample size	Outcome(s) studied/size of effect ^a
Millar (1988)/ Ontario, Canada	Department of Health and Welfare	4200 (12 locations)	Change in mean respirable suspended particulates = $-6 \mu\text{g}/\text{m}^3$ to $-22 \mu\text{g}/\text{m}^3$ (depending on the storey of the building)
Becker <i>et al.</i> (1989)/ Maryland, USA	Children's hospital	951 (9 locations)	Change in average nicotine vapour concentrations = $-12.53 \mu\text{g}/\text{m}^3$ to $+0.08 \mu\text{g}/\text{m}^3$ (depending on the location)
Biener <i>et al.</i> (1989)/ Rhode Island, USA	Hospital	535	Percentage of workers 'bothered' by secondhand smoke in various workplace locations: offices = -20% ; lounges = -20%
Gottlieb <i>et al.</i> (1990)/Texas, USA	Government agency	1158	Percentage of workers 'never bothered' by secondhand smoke = $+38.8\%$
Mullooly <i>et al.</i> (1990)/Oregon, USA	Health maintenance organization	13 736 1985: pre-ban 764 post-ban 1027 1986: pre-ban 1352 post-ban 1219	Presence of smoke in workplace = -21% (1985 sites); -35% (1986 sites)
Stillman <i>et al.</i> (1990)/Maryland, USA	Medical centre	8742 (7 locations)	Change in average 7-day nicotine vapour concentrations = $-7.71 \mu\text{g}/\text{m}^3$ to $-0.72 \mu\text{g}/\text{m}^3$ (depending on the location)
Borland <i>et al.</i> (1992)/ California, USA	Indoor workers in California	7301	Percentage of employees exposed to secondhand smoke at work = -42.1% between no policy and smoke-free policy
Broder <i>et al.</i> (1993)/ Toronto, Canada	Public sector workplaces	179 (3 buildings; 8–12 samples per floor)	Change in the mean measurements (for several secondhand smoke components) Volatile organic compounds = $-0.7 \text{mg}/\text{m}^3$
Patten <i>et al.</i> (1995)/California, USA	Statewide workers	8580 (at baseline survey)	Percentage of employees exposed to secondhand smoke at work = -56.3% difference between work area ban and no ban

Table 1.9 (contd)

Reference/location	Industry/setting	Sample size	Outcome(s) studied/size of effect ^a
Etter <i>et al.</i> (1999)/ Geneva, Switzerland	University	2908	Exposure to secondhand smoke (score 0–100; ‘not at all’ (0) to ‘very much’ (100)) = –4% (follow-up compared to baseline)

Brownson *et al.* (2002); modified from Hopkins *et al.* (2001)

^a Values noted are absolute differences from baseline.

children. In 1997, a population-based, cross-sectional telephone survey was conducted using random-digit-dialling asking 6199 adult Oregonians to provide baseline data on tobacco use in Oregon. Seventy per cent of the households were composed of nonsmokers only, and 85% of those had a full ban on smoking inside the home. Of the households containing one or more smokers, 38% had a full household ban on smoking. Fifty per cent of households with at least a smoker and a child present did not have a full ban on indoor smoking (Pizacani *et al.*, 2003). Face-to-face interviews were conducted with 380 rural, low-income Native American and white parents of children aged 1–6 years in Oklahoma. The prevalence of complete smoking bans was 49% in Native American homes and 43% in white homes. Bans on smoking in cars were less common, with 35% of Native American and 40% of white caregivers reporting complete bans (Kegler & Malcoe, 2002). In Victoria, Australia, the percentage of respondents who reported discouraging visitors from smoking in the home rose from 27% in 1989 to 53% in 1997 (Borland *et al.*, 1999), and not smoking in the presence of children rose from 14% in 1989 to 33% in 1996. Similarly, attitudes toward smoking in the home have changed in Ontario, Canada. The percentage of respondents favouring not smoking in homes where there were children increased from 51% in 1992 to 70% in 1996 (Ashley *et al.*, 1998).

Only minimal regulation applies to constituents of cigarettes and tobacco smoke. This covers only the content of tar, nicotine and carbon monoxide (cf. Section 1.4(e) of the monograph on tobacco smoke).

1.3.3 Effectiveness of regulations

Evaluations of the effects and effectiveness of workplace smoking policies have used a wide variety of study designs and measurements of exposure to secondhand tobacco smoke and tobacco use behaviours. Most of the published studies are simple assessments conducted before and after adoption of a workplace policy, although more recent (and more complex) investigations have employed cross-sectional surveys of workers in workplaces operating different policies. Few studies have evaluated or controlled for potential bias and confounding of the observed differences or changes in exposure to secondhand smoke or in tobacco use behaviours (Brownson *et al.*, 2002).

The effectiveness of workplace smoking policies has been measured by differences or changes in perceived air quality in the workplace following a ban or restriction, and by differences or changes in active measurements of nicotine vapour concentrations, metabolites, or levels of particles. Overall, workplace smoking policies have been highly effective in reducing the exposure of nonsmokers to secondhand tobacco smoke. The 'best evidence subset' comprised ten studies, including cross-sectional surveys, before-and-after comparisons, different settings or locations (offices, public sector workplaces, medical centres, workplaces community-wide) and different outcome measurements (Table 1.9) (Briss *et al.*, 2000; Hopkins *et al.*, 2001). In nine of ten studies, workplace smoking policies had a significant impact on exposure to secondhand tobacco smoke. In assessments conducted between 4 and 18 months after implementation of the policy, the median relative percentage difference in self-reported exposure to secondhand tobacco smoke was -60%, range +4% to -97%. Workplaces with smoking bans tended to show greater reduction in exposure to secondhand tobacco smoke than did workplaces with smoking restrictions (Hopkins *et al.*, 2001; Brownson *et al.*, 2002).

Hammond (1999) summarized the existing literature on average indoor nicotine concentrations when various workplace smoking policies were enacted. In workplaces with policies that had banned smoking, nicotine concentrations were generally decreased to less than 1 $\mu\text{g}/\text{m}^3$. The mean concentrations of nicotine in workplaces that allowed smoking ranged from 2 to 6 $\mu\text{g}/\text{m}^3$ in offices, 3 to 8 $\mu\text{g}/\text{m}^3$ in restaurants and from 1 to 6 $\mu\text{g}/\text{m}^3$ in the workplaces of blue-collar workers. By comparison, studies of nicotine concentrations that included at least 10 homes of smokers and that were sampled for 14 h to 1 week found average nicotine concentrations of between 1 and 6 $\mu\text{g}/\text{m}^3$.

References

- Adams, J.D., O'Mara-Adams, K.J. & Hoffmann, D. (1987) Toxic and carcinogenic agents in undiluted mainstream smoke and sidestream smoke of different types of cigarettes. *Carcinogenesis*, **8**, 729-731
- Adlkofer, F., Scherer, G., Conze, C., Angerer, J. & Lehnert, G. (1990) Significance of exposure to benzene and other toxic compounds through environmental tobacco smoke. *J. Cancer Res. clin. Oncol.*, **116**, 591-598
- ASH (Action on Smoking and Health) (2001) Smoking in the workplace. ASH, National Asthma Campaign and Trade Union Congress. WHO-Europe Partnership Project, Copenhagen [<http://www.ash.org.uk/html/workplace/html/workplace.html>]
- Ashley, M.J., Cohen, J., Ferrence, R., Bull, S., Bondy, S., Poland, B. & Pederson, L. (1998) Smoking in the home: Changing attitudes and current practices. *Am. J. public Health*, **88**, 797-800
- Baker, R.R. & Proctor, C.J. (1990) The origins and properties of environmental tobacco smoke. *Environ. int.*, **16**, 231-245
- Becker, D.M., Conner, H.F., Waranch, H.R., Stillman, F., Pennington, L., Lees, P.S. & Oski, F. (1989) The impact of a total ban on smoking in the Johns Hopkins Children's Center. *J. Am. med. Assoc.*, **262**, 799-802

- Benowitz, N.L. (1996) Cotinine as a biomarker of environmental tobacco smoke exposure. *Epidemiol. Rev.*, **18**, 188–204
- Benowitz, N.L. (1999) Biomarkers of environmental tobacco smoke exposure. *Environ. Health Perspect.*, **107** (Suppl. 2), 349–355
- Biener, L., Abrams, D.B., Follick, M.J. & Dean, L. (1989) A comparative evaluation of a restrictive smoking policy in a general hospital. *Am. J. public Health*, **79**, 192–195
- Boffetta, P., Agudo, A., Ahrens, W., Benhamou, E., Benhamou, S., Darby, S.C., Ferro, G., Fortes, C., Gonzalez, C.A., Jöckel, K.H., Krauss, M., Kreienbrock, L., Kreuzer, M., Mendes, A., Merletti, F., Nyberg, F., Pershagen, G., Pohlbeln, H., Riboli, E., Schmid, G., Simonato, L., Trédaniel, J., Whitley, E., Wichmann, H.E., Winck, C., Zambon, P. & Saracci, R. (1998) Multi-center case-control study of exposure to environmental tobacco smoke and lung cancer in Europe. *J. natl Cancer Inst.*, **90**, 1440–1450
- Bombick, B.R., Avalos, J.T., Nelson, P.R., Conrad, F.W. & Doolittle, D.J. (1998) Comparative studies of the mutagenicity of environmental tobacco smoke from cigarettes that burn or primarily heat tobacco. *Environ. Mol. Mutag.*, **31**, 169–175
- Borgerding, M.F., Bodnar, J.A. & Wingate, D.E. (2000) *The 1999 Massachusetts Benchmark Study. Final Report. A Research Study Conducted after Consultation with the Massachusetts Department of Public Health*, Department of Health, Massachusetts
- Borland, R., Pierce, J.P., Burns, D.M., Gilpin, E., Johnson, M. & Bal, D. (1992) Protection from environmental tobacco smoke in California. The case for a smoke-free workplace. *J. Am. med. Assoc.*, **268**, 749–752
- Borland, R., Mullins, R., Trotter, L. & White, V. (1999) Trends in environmental tobacco smoke restrictions in the home in Victoria, Australia. *Tob. Control*, **8**, 266–271
- Brenner, H., Born, J., Novak, P. & Wanek, V. (1997) Smoking behavior and attitude toward smoking regulations and passive smoking in the workplace. A study among 974 employees in the German metal industry. *Prev. Med.*, **26**, 138–143
- Briss, P.A., Zaza, S., Pappaioanou, M., Fielding, J., Wright-De Agüero, L., Truman, B.I., Hopkins, D.P., Mullen, P.D., Thompson, R.S., Woolf, S.H., Carande-Kulis, V.G., Anderson, L., Hinman, A.R., McQueen, D.V., Teutsch, S.M., Harris, J.R. & The Task Force on Community Preventive Services (2000) Developing an evidence-based Guide to Community Preventive Services — Methods. *Am. J. prev. Med.*, **18**, 35–43
- Broder, I., Pilger, C. & Corey, P. (1993) Environment and well-being before and following smoking ban in office buildings. *Can. J. public Health*, **84**, 254–258
- Brownson, R.C., Hopkins, D.P. & Wakefield, M.A. (2002) Effects of smoking restrictions in the workplace. *Ann. Rev. public Health*, **23**, 333–348
- Brunnemann, K.D., Kagan, M.R., Cox, J.E. & Hoffmann, D. (1990) Analysis of 1,3-butadiene and other selected gas-phase components in cigarette mainstream and sidestream smoke by gas chromatography-mass selective detection. *Carcinogenesis*, **11**, 1863–1868
- Brunnemann, K.D., Cox, J.E. & Hoffmann, D. (1992) Analysis of tobacco-specific N-nitrosamines in indoor air. *Carcinogenesis*, **13**, 2415–2418
- Burns, D. & Pierce, J.P. (1992) *Tobacco Use in California 1990–1991*, Sacramento, CA, California Department of Health Services
- Burns, D.M., Shanks, T.G., Major, J.M., Gower, K.B. & Shopland, D.R. (2000) Restrictions on smoking in the workplace. In: *Population Based Smoking Cessation, Proceedings of a Conference on what Works to Influence Cessation in the General Population. Smoking and Tobacco*

- Control Monograph No. 12*, Bethesda, MD, US Department of Health and Human Services, National Institutes of Health, National Cancer Institute (NIH Pub. No. 00-4892), pp. 99–128
- CDC (Centers for Disease Control and Prevention) (2001) *National Report on Human Exposure to Environmental Chemicals*, Atlanta, GA
- California EPA (California Environmental Protection Agency) (1997) *Health Effects of Exposure to Environmental Tobacco Smoke, Final Report*, California Environmental Protection Agency, Office of Environmental Health Hazard Assessment
- Chilmonczyk, B.A., Knight, G.J., Palomaki, G.E., Pulkkinen, A.J., Williams, J. & Haddow, J.E. (1990) Environmental tobacco smoke exposure during infancy. *Am. J. Public Health*, **80**, 1205–1208
- Chortyk, O.T. & Schlotzhauer, W.W. (1989) The contribution of low-tar cigarettes to environmental tobacco smoke. *J. anal. Toxicol.*, **13**, 129–134
- Corrao, M.A., Guindon, G.E., Sharma, N. & Shokoohi, D.F., eds (2000) *Tobacco Control Country Profiles*, Atlanta, GA, American Cancer Society [http://www.cancer.org/eprise/main/docroot/res/content/res_6_4x_other_publications]
- Coultas, D.B., Howard, C.A., Peake, G.T., Skipper, B.J. & Samet, J.M. (1987) Salivary cotinine levels and involuntary tobacco smoke exposure in children and adults in New Mexico. *Am. Rev. Respir. Dis.*, **136**, 305–309
- Curtin, F., Morabia, A. & Bernstein, M. (1998) Lifetime exposure to environmental tobacco smoke among urban women: Differences by socioeconomic class. *Am. J. Epidemiol.*, **148**, 1040–1047
- Dijkstra, L., Houthuijs, D., Brunekreef, B., Akkerman, I. & Boleij, J.S. (1990) Respiratory health effects of the indoor environment in a population of Dutch children. *Am. Rev. Respir. Dis.*, **142**, 1172–1178
- Djordjevic, M.V., Stellman, S.D. & Zang, E. (2000) Doses of nicotine and lung carcinogens delivered to cigarette smokers. *J. natl Cancer Inst.*, **92**, 106–111
- Eatough, D.J., Benner, C.L., Tang, H., Landon, V., Richards, G., Caka, F.M., Crawford, J., Lewis, E.A., Hansen, L.D. & Eatough, N.L. (1989) The chemical composition of environmental tobacco smoke. III. Identification of conservative tracers of environmental tobacco smoke. *Environ. int.*, **15**, 19–28
- Etter, J.-F., Ronchi, A. & Perneger, T.V. (1999) Short-term impact of a university based smoke free campaign. *J. Epidemiol. Community Health*, **53**, 710–715
- Farrelly, M.C., Evans, W.N. & Sfekas, A.E. (1999) The impact of workplace smoking bans: Results from a national survey. *Tob. Control*, **8**, 272–277
- Freeth, S. (1998) *Smoking-related Behaviour and Attitudes, 1997. A Report on Research using the Omnibus Survey produced on behalf of the Department of Health*, London, Office for National Statistics, p. 13
- Gerlach, K.K., Shopland, D.R., Hartman, A.M., Gibson, J.T. & Pechacek, T.F. (1997) Workplace smoking policies in the United States: Results from a national survey of more than 100 000 workers. *Tob. Control*, **6**, 199–206
- Gottlieb, N.H., Eriksen, M.P., Lovato, C.Y., Weinstein, R.P. & Green, L.W. (1990) Impact of a restrictive work site smoking policy on smoking behavior, attitudes, and norms. *J. occup. Med.*, **32**, 16–23

- Government of British Columbia (2003) What is in cigarettes? Mainstream smoke and sidestream smoke chemical constituents by cigarette brand. [<http://www.healthplanning.gov.bc.ca/ttdr/index.html> Accessed 07.03.2003]
- Greenberg, R.A., Bauman, K.E., Glover, L.H., Strecher, V.J., Kleinbaum, D.G., Haley, N.J., Stedman, H.C., Fowler, M.G. & Loda, F.A. (1989) Ecology of passive smoking by young infants. *J. Pediatr.*, **114**, 774–780
- Guerin, M.R., Higgins, C.E. & Jenkins, R.A. (1987) Measuring environmental emissions from tobacco combustion: Sidestream cigarette smoke literature review. *Atmos. Environ.*, **21**, 291–297
- Guerin M.R., Jenkins, R.A. & Tomkins, B.A., eds (1992) *The Chemistry of Environmental Tobacco Smoke: Composition and Measurement*. Center for Indoor Air Research, Chelsea, MI, Lewis Publishers
- Hammond, S.K. (1999) Exposure of U.S. workers to environmental tobacco smoke. *Environ. Health Perspect.*, **107** (Suppl. 2), 329–340
- Heavner, D.L., Morgan, W.T. & Ogden, M.W. (1995) Determination of volatile organic compounds and ETS apportionment in 49 homes. *Environ. int.*, **21**, 3–21
- Higgins, C.E., Thompson, C.V., Ilgner, L.H., Jenkins, R.A. & Guerin, M.R. (1990) *Determination of Vapor Phase Hydrocarbons and Nitrogen-containing Constituents in Environmental Tobacco Smoke. Internal Progress Report*, Analytical Chemistry Division, Oak Ridge National Laboratory, Oak Ridge, TN 37831-6120
- Hodgson, A.T., Daisey, J.M., Mahanama, K.R.R., Brinke, T. & Alevantis, L.E. (1996) Use of volatile tracers to determine the contribution of environmental tobacco smoke to concentrations of volatile organic compounds in smoking environments. *Environ. Int.*, **22**, 295–307
- Hopkins, D.P., Briss, P.A., Ricard, C.J., Husten, C.G., Carande-Kulis, V.G., Fielding, J.E., Alao, M.O., McKenna, J.W., Sharp, D.J., Harris, J.R., Woollery, T.A., Harris, K.W. & The Task Force on Community Preventive Services (2001) Reviews of evidence regarding interventions to reduce tobacco use and exposure to environmental tobacco smoke. *Am. J. prev. Med.*, **20**, 16–66
- Hyvärinen, M.J., Rothberg, M., Kähkönen, E., Mielo, T. & Reijula, K. (2000) Nicotine and 3-ethenylpyridine concentrations as markers for environmental tobacco smoke in restaurants. *Indoor Air*, **10**, 121–125
- IARC (1986) *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans*, Vol. 38, *Tobacco Smoking*, Lyon, IARC Press
- IARC (1999) *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, Vol. 71, *Re-evaluation of Some Organic Chemicals, Hydrazine and Hydrogen Peroxide*, Lyon, IARC Press
- Jaakkola, M.S. & Jaakkola, J.J.K. (1997) Assessment of exposure to environmental tobacco smoke. *Eur. Respir. J.*, **10**, 2384–2397
- Jaakkola, N., Ruotsalainen, R. & Jaakkola, J.J.K. (1994) What are the determinants of children's exposure to environmental tobacco smoke at home? *Scand. J. Soc. Med.*, **22**, 107–112
- Jenkins, R.A. & Counts, R.W. (1999) Occupational exposure to environmental tobacco smoke: Results of two personal exposure studies. *Environ. Health Perspect.*, **107** (Suppl. 2), 341–348
- Jenkins, P.L., Phillips, T.J., Mulberg, E.J. & Hui, S.P. (1992) Activity patterns of Californians: Use of and proximity to indoor pollutant sources. *Atmos. Environ.*, **26A**, 2141–2148

- Jenkins, R.A., Palausky, A., Counts, R.W., Bayne, C.K., Dindal, A.B. & Guerin, M.R. (1996) Exposure to environmental tobacco smoke in sixteen cities in the United States as determined by personal breathing zone air sampling. *J. Expo. anal. environ. Epidemiol.*, **6**, 473–502
- Jenkins, R.A., Guerin, M.R. & Tomkins, B.A. (2000) Properties and measure of environmental tobacco smoke. In: *The Chemistry of Environmental Tobacco Smoke. Composition and Measurement*, 2nd Ed., Boca Raton, FL, Lewis Publishers, CRC Press
- Jousilahti, P. & Helakorpi, S. (2002) Prevalence of exposure to environmental tobacco smoke at work and at home — 15-year trends in Finland. *Scand. J. Work Environ. Health*, **28** (Suppl. 2), 16–20
- Junker, M.H., Danuser, B., Monn, C. & Koller, T. (2001) Acute sensory responses of nonsmokers at very low environmental tobacco smoke concentrations in controlled laboratory settings. *Environ. Health Perspect.*, **109**, 1045–1052
- Kegler, M.C. & Malcoe, L.H. (2002) Smoking restrictions in the home and car among rural Native American and white families with young children. *Prev. Med.*, **35**, 334–342
- Kim, Y.M., Harrad, S. & Harrison, R.M. (2001) Concentrations and sources of VOCs in urban domestic and public microenvironments. *Environ. Sci. Technol.*, **35**, 997–1004
- Klepeis, N.E., Ott, W.R. & Repace, J.L. (1999) The effect of cigar smoking on indoor levels of carbon monoxide and particles. *J. exp. anal. environ. Epidemiol.*, **9**, 622–635
- Klus, H., Begutter, H., Scherer, G., Tricker, A.R. & Adlkofer, F. (1992) Tobacco-specific and volatile *N*-nitrosamines in environmental tobacco smoke of offices. *Indoor Environ.*, **1**, 348–350
- Kurtz, M.E., Kurtz, J.C., Johnson, S.M. & Beverly, E.E. (1996) Exposure to environmental tobacco smoke — Perceptions of African American children and adolescents. *Prev. Med.*, **25**, 286–292
- Lam, T.H., Chung, S.F., Betson, C.L., Wong, C.M. & Hedley, A.J. (1998) Respiratory symptoms due to active and passive smoking in junior secondary school students in Hong Kong. *Int. J. Epidemiol.*, **27**, 41–48
- Letcher, T. & Borland, R. (2000) Smoking bans in Victorian workplaces: 1999 update. In: Trotter, L. & Letcher, T., eds, *Quit Evaluation Studies*, Vol. 10, pp. 49–57, Centre for Behavioural Research on Cancer [<http://www.quit.org.au/quit/qe10/10home.html>], Melbourne, Australia. Victorian Smoking and Health Program, The Anti-Cancer Council of Victoria
- Lister, S.M. & Jorm, L.R. (1998) Parental smoking and respiratory illnesses in Australian children aged 0–4 years: ABS 1989–1990 National Health Survey results. *Australian and New Zealand J. Public Health*, **22**, 781–786
- Löfroth, G. (1989) Environmental tobacco smoke: Overview of chemical composition and genotoxic components. *Mutat. Res.*, **222**, 73–80
- Löfroth, G. (1993) Environmental tobacco smoke. Multicomponent analysis and room to room distribution in homes. *Tob. Control*, **2**, 222–225
- Löfroth, G., Burton, R., Goldstein, G., Forehand, L., Hammond, K., Mumford, J., Seila, R. & Lewtas, J. (1987) Genotoxic emission factors for sidestream cigarette smoke components (Abstract). *Environ. Mutag.*, **9** (Suppl. 8), 61
- Löfroth, G., Burton, R.M., Forehand, L., Hammond, S.K., Seila, R.L., Zweidinger, R.B. & Lewtas, J. (1989) Characterization of environmental tobacco smoke. *Environ. Sci. Technol.*, **23**, 610–614

- Longo, D.R., Brownson, R.C. & Kruse, R.L. (1995) Smoking bans in US hospitals. Results of a national survey. *J. Am. med. Assoc.*, **274**, 488–491
- Lum, S. (1994) Duration and location of ETS exposure for the California population. Memorandum from S. Lum, Indoor Exposure Assessment Section, Research Division, California Air Resources Board, to L. Haroun, Reproductive and Cancer Hazard Assessment Section, Office of Environmental Health Hazard Assessment, 3 February (cited according to Samet and Yang, 2001)
- Lund, K.E., Skrondal, A., Vertio, H. & Helgason, A.R. (1998) Children's residential exposure to environmental tobacco smoke varies greatly between the Nordic countries. *Scand. J. soc. Med.*, **26**, 115–120
- Mannino, D.M., Siegel, M., Husten, C., Rose, D. & Etzel, R. (1996) Environmental tobacco smoke exposure and health effects in children: Results from the 1991 National Health Interview Survey. *Tob. Control*, **5**, 13–18
- Martin, P., Heavner, D.L., Nelson, P.R., Maiolo, K.C., Risner, C.H., Simmons, P.S., Morgan, W.T. & Ogden, M.W. (1997) Environmental tobacco smoke (ETS): A market cigarette study. *Environ. int.*, **23**, 75–90
- McMaugh, K. & Rissell, C. (2000) Smoking restrictions in small businesses of inner West Sydney: A management perspective. *J. occup. Health Saf.-Aust. NZ*, **16**, 37–45
- Millar, W.J. (1988) Evaluation of the impact of smoking restrictions in a government work setting. *Can. J. public Health*, **79**, 379–382
- Moschandreas, D.J. & Vuilleumier, K.L. (1999) ETS levels in hospitality environments satisfying ASHRAE standard 62-1989: 'Ventilation for acceptable indoor air quality'. *Atmos. Env.*, **33**, 4327–4340
- Mullooly, J.P., Schuman, K.L., Stevens, V.J., Glasgow, R.E. & Vogt, T.M. (1990) Smoking behavior and attitudes of employees of a large HMO before and after a work site ban on cigarette smoking. *Public Health Rep.*, **105**, 623–628
- Nelson, P.R., Kelly, S.P. & Conrad, F.W. (1997) Generation of environmental tobacco smoke by cigars (Abstract No. 46). In: *Proceedings of the 51st Tobacco Chemist's Research Conference, Winston-Salem, NC, September 14–17, 1997*
- NRC (National Research Council) (1986) *Environmental Tobacco Smoke. Measuring Exposures and Assessing Health Effects*, Washington DC, National Academy Press
- Ott, W.R. (1999) Mathematical models for predicting indoor air quality from smoking activity. *Environ. Health Perspect.*, **107** (Suppl. 2), 375–381
- Overpeck, M.D. & Moss, A.J. (1991) Children's exposure to environmental cigarette smoke before and after birth. Health of our nation's children, United States, 1988. *Adv. Data*, **202**, 1–11
- Pakhale, S.S. & Maru, G.B. (1998) Distribution of major and minor alkaloids in tobacco, mainstream and sidestream smoke of popular Indian smoking products. *Food Chem. Toxicol.*, **36**, 1131–1138
- Patten, C.A., Pierce, J.P., Cavin, S.W., Berry, C.C. & Kaplan, R.M. (1995) Progress in protecting non-smokers from environmental tobacco smoke in California workplaces. *Tob. Control*, **4**, 139–144
- Phillips, K. & Bentley, M.C. (2001) Seasonal assessment of environmental tobacco smoke and respirable suspended particle exposures for nonsmokers in Bremen using personal monitoring. *Environ. int.*, **27**, 69–85

- Pierce, J.P., Evans, N., Farkas, S.J., Cavin, S.W., Berry, C., Kramer, M., Kealey, S., Rosbrook, B., Choi, W. & Kaplan, R.M. (1994) *Tobacco Use in California: An Evaluation of the Tobacco Control Program, 1989–1993*. La Jolla, CA. Cancer Prevention and Control, University of California, San Diego
- Pirkle, J., Flegal, K., Bernert, J., Brody, D., Etzel, R. & Maurer, K. (1996) Exposure of the US population to environmental tobacco smoke. The Third National Health and Nutrition Examination Survey, 1988 to 1991. *J. Am. med. Assoc.*, **275**, 1233–1240
- Pizacani, B.A., Martin, D.P., Stark, M.J., Koepsell, T.D., Thompson, B. & Diehr, P. (2003) Household smoking bans: Which households have them and do they work? *Prev. Med.*, **36**, 99–107
- Pletsch, P.K. (1994) Environmental tobacco smoke exposure among Hispanic women of reproductive age. *Public Health Nursing*, **11**, 229–235
- Ramsey, R.S., Moneyhun, J.H. & Jenkins, R.A. (1990) Generation, sampling and chromatographic analysis of particulate matter in dilute sidestream tobacco smoke. *Anal. chem. Acta*, **236**, 213–220
- Repace, J.L. (1994) Risk management of passive smoking at work and at home. *Saint Louis University Public Law Review*, **13**, 763–785
- Rickert, W.S., Robinson, J.C. & Collishaw, N. (1984) Yields of tar, nicotine, and carbon monoxide in the sidestream smoke from 15 brands of Canadian cigarettes. *Am. J. public Health*, **74**, 228–231
- Samet, J. & Yang, G. (2001) Passive smoking, women and children. In: Samet, J. & Yoon, S.-Y., eds, *Women and the Tobacco Epidemic, Challenges for the 21st Century* (WHO/NMH/TF1/01.1), Geneva, World Health Organization
- Scherer, G., Frank, S., Riedel, K., Meger-Kossien, I. & Renner, T. (2000) Biomonitoring of exposure to polycyclic aromatic hydrocarbons of nonoccupationally exposed persons. *Cancer Epidemiol. Biomarkers Prev.*, **9**, 373–380
- Sherrill, D.L., Martinez, F.D., Lebowitz, M.D., Holdaway, M.D., Flannery, E.M., Herbison, G.P., Stanton, W.R., Silva, P.A. & Sears, M.R. (1992) Longitudinal effects of passive smoking on pulmonary function in New Zealand children. *Am. Rev. Respir. Dis.*, **145**, 1136–1141
- Siegel, M. (1993) Involuntary smoking in the restaurant workplace. A review of employee exposure and health effects. *J. Am. med. Assoc.*, **270**, 490–493
- Siegel, M., Husten, C., Merritt, R.K., Giovino, G.A. & Eriksen, M.P. (1995) Effects of separately ventilated smoking lounges on the health of smokers: Is this an appropriate public health policy? *Tob. Control*, **4**, 22–29
- Somerville, S.M., Rona, R.J. & Chinn, S. (1988) Passive smoking and respiratory conditions in primary school children. *J. Epidemiol. Community Health*, **42**, 105–110
- Stamatakis, K.A., Brownson, R.C. & Luke, D.A. (2002) Risk factors for exposure to environmental tobacco smoke among ethnically diverse women in the United States. *J. Women's Health Gen. Based Med.*, **11**, 45–51
- Steyn, K., Yach, D., Stander, I. & Fourie, J.M. (1997) Smoking in urban pregnant women in South Africa. *S. Afr. Med. J.*, **87**, 460–463
- Stillman, F.A., Becker, D.M., Swank, R.T., Hantula, D., Moses, H., Glantz, S. & Waranch, H.R. (1990) Ending smoking at the Johns Hopkins Medical Institutions. An evaluation of smoking prevalence and indoor air pollution. *J. Am. med. Assoc.*, **264**, 1565–1569

- Thompson, B., Emmons, K., Abrams, D., Ockene, J.K. & Feng, Z. (1995) ETS exposure in the workplace. Perceptions and reactions by employees in 114 work sites. Working Well Research Group [corrected]. *J. Occup. environ. Med.*, **37**, 1086–1092
- Totsuka, Y., Ushiyama, H., Ishihara, J., Sinha, R., Goto, S., Sugimura, T. & Wakabayashi, K. (1999) Quantification of the co-mutagenic beta-carbolines, norharman and harman, in cigarette smoke condensates and cooked foods. *Cancer Lett.*, **143**, 139–143
- US Department of Health and Human Services (2000) *National Worksite Health Promotion Survey*. Available at: <http://www.cdc.gov/nchs/data/hp2000/safety/sumtable.pdf> (accessed 24 January 2003), Washington DC, Office of Disease Prevention and Health Promotion
- US EPA (US Environmental Protection Agency) (1992) *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders* (EPA/600/6-90/006F), Washington DC, United States Environmental Protection Agency
- Vainiotalo, S., Vaaranrinta, R., Tornaeus, J., Aremo, N., Hase, T. & Peltonen, K. (2001) Passive monitoring method for 3-ethenylpyridine: A marker for environmental tobacco smoke. *Environ. Sci. Technol.*, **35**, 1818–1822
- Vu Duc, T. & Huynh, C.K. (1987) Deposition rates of sidestream tobacco smoke particles in an experimental chamber. *Toxicol. Lett.*, **35**, 59–65
- Wakefield, M., Roberts, L. & Owen, N. (1996) Trends in prevalence and acceptance of workplace smoking bans among indoor workers in South Australia. *Tob. Control*, **5**, 205–208
- Whitlock, G., MacMahon, S., Vander Hoorn, S., Davis, P., Jackson, R. & Norton, R. (1998) Association of environmental tobacco smoke exposure with socioeconomic status in a population of 7725 New Zealanders. *Tob. Control*, **7**, 276–280
- Yang, G., Fan, L., Tan, J., Qi, G., Zhang, Y., Samet, J.M., Taylor, C.E., Becker, K. & Xu, J. (1999) Smoking in China. Findings of the 1996 National Prevalence Survey. *JAMA*, **282**, 1247–1253