ENVIRONMENTAL SCIENCE AND TECHNOLOGY

EXPOSURE ANALYSIS

Written by experts, ***Exposure Analysis*** is the first complete resource in the emerging scientific discipline of exposure analysis. A comprehensive source on the environmental pollutants that affect human health, the book discusses human exposure through pathways including air, food, water, dermal absorption, and, for children, non-food ingestion.

The book summarizes existing definitions of exposure, dose, and related concepts and provides the mathematical framework at the heart of these conceptual definitions. Using secondhand smoke as an example, the book illustrates how exposure analysis studies can change human behavior and improve public health. An extensive section on air pollutants considers volatile organic compounds (VOCs), carbon monoxide (CO), fine and ultrafine particles, and the latest personal air quality monitors for measuring individual exposure. Another detailed section examines exposures to pesticides, metals such as lead, and dioxin that may occur through multiple routes such as air, food, and dust ingestion. The book explores important aspects of dermal exposure such as the absorption of volatile organic compounds while showering or bathing and exposure through multiple carrier media. The authors describe quantitative methods that have been validated for predicting the concentrations in enclosed everyday locations, such as automobiles and rooms of the home. They also discuss existing laws and examine the relationship between exposure and national policies.

Defining the new field of exposure analysis, this book provides the basic tools needed to identify sources, understand causes, measure exposures, and develop strategies for improving public health.

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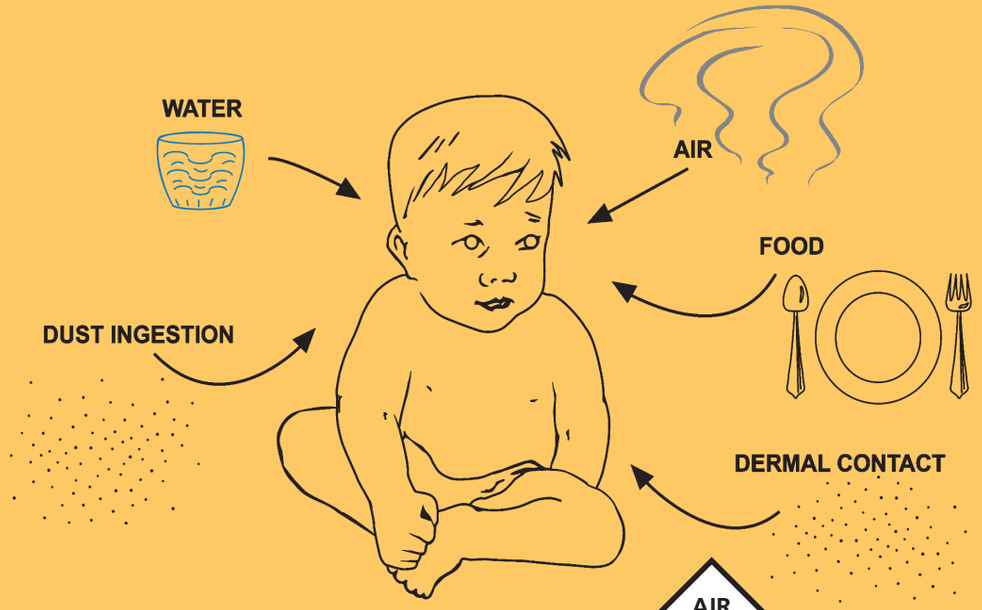

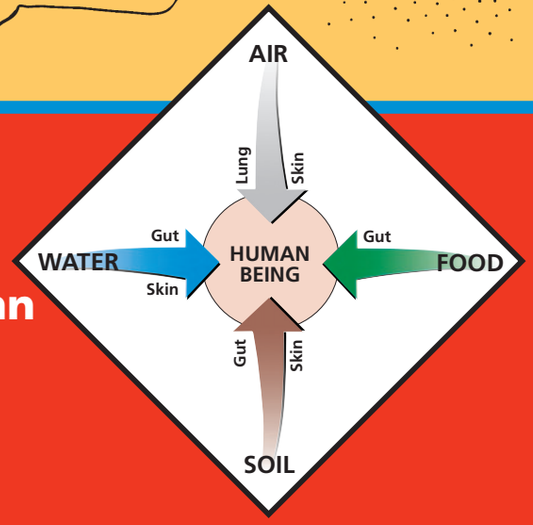

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9 Exposure to Secondhand Smoke

James L. Repace
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CONTENTS

9.1 SYNOPSIS

Secondhand smoke (SHS) has been estimated to cause as much as 2.7% of all deaths in the United States annually. Its adverse health effects have been estimated to cost more than $25 billion annually in California alone. SHS is a source of at least 172 toxic substances in indoor air. A major pollutant

emitted by SHS is respirable suspended particles (RSP). RSP concentrations in indoor microenvironments where smoking occurs greatly exceed the levels encountered in smoke-free environments, outdoors, and in vehicles on busy highways. SHS appears to contribute the overwhelming majority of carcinogenic particle-bound polycyclic aromatic hydrocarbons in the air of most buildings where smoking occurs. SHS-carbon monoxide levels measured in pubs are in the range that produces acute cardiovascular effects. This chapter discusses the toxic constituents of SHS, and the prevalence of nonsmokers' exposure, as well as factors determining exposure and dose. The microenvironments of greatest importance are those where the population spends the most time: at home and in the workplace. In-vehicle exposure is also of concern due to the high concentrations observed. Use of a personal exposure monitor to estimate relative contributions of smoking, cooking, and diesel exhaust to a person's RSP exposure is illustrated. How such personal exposures combine into a population distribution is illuminated, and the major U.S. field study of SHS dose in the population is deconstructed. Field studies and controlled measurements of SHS concentrations in homes and workplaces are reviewed. SHS emission and removal rates are discussed in the context of the time-averaged mass-balance model for estimating concentrations in naturally and mechanically ventilated buildings, and examples for homes and bars are given. SHS-RSP can be related to SHS-nicotine, which when inhaled is metabolized into the SHS biomarker, cotinine. National surveys have shown that nearly half of nonsmokers who report no SHS exposure have detectable levels of cotinine in their body fluids. Despite growing trends toward indoor public and workplace smoking bans, SHS exposure continues for half of all children at home in the United States, and for most bar and casino workers.

9.2 INTRODUCTION

Cigarette smoking proliferated after World War I, but not until the 1964 U.S. Surgeon General's Report on Smoking (Surgeon General 1964) was the public made aware that smoking could kill. At that time there was little understanding that indoor air pollution from the tobacco smoke exhaled by smokers and emitted from the burning ends of cigarettes, pipes, and cigars — i.e., secondhand smoke (SHS), also known as environmental tobacco smoke (ETS) (Figure 9.1) — was for nonsmokers actually the most significant source of human air pollution exposure. Moreover, as smoking was then wrongly but widely regarded as a "personal choice," public health advice was largely limited to smoking cessation. Regulation of tobacco products in the United States became difficult as the tobacco industry quietly got Congress to exempt its products from every conceivable federal law under which their emissions could possibly be regulated (Repace 1981). Yet, exposure to tobacco smoke at the levels encountered in smoking damages nearly every organ in the human body and caused an estimated 440,000 excess deaths per year in 2004 (Surgeon General 2004). Thus, tobacco smoke is a seriously toxic substance, for which no safe level of exposure has been identified.

Research into indoor air pollution from SHS proliferated in the 1970s, resulting in a cascade of lengthy, authoritative, peer-reviewed reports by national and international environmental, occupational, and public health authorities. The Surgeon General (1986), the National Academy of Sciences (National Research Council 1986), the International Agency for Research on Cancer (IARC 1987, 2004), the National Institute for Occupational Safety and Health (NIOSH 1991), the U.S. Environmental Protection Agency (USEPA 1992), the Occupational Safety & Health Administration (OSHA 1994), the National Cancer Institute (NCI 1993, 1998, 1999), the California EPA (CalEPA 1997, 2005), and the National Toxicology Program (NTP 2000) variously concluded that nonsmokers' exposure to SHS causes fatal heart disease; lung, breast, and nasal sinus cancer; asthma induction and aggravation; middle ear infection; sudden infant death syndrome; and respiratory impairment; as well as irritation of the mucous membranes of the eyes, nose, and throat. SHS is now widely accepted as the third leading preventable health hazard after active smoking and alcohol (Surgeon General 2004); nevertheless it continues to be a widespread indoor pollutant in many homes, workplaces, and public access buildings in the United States and abroad.

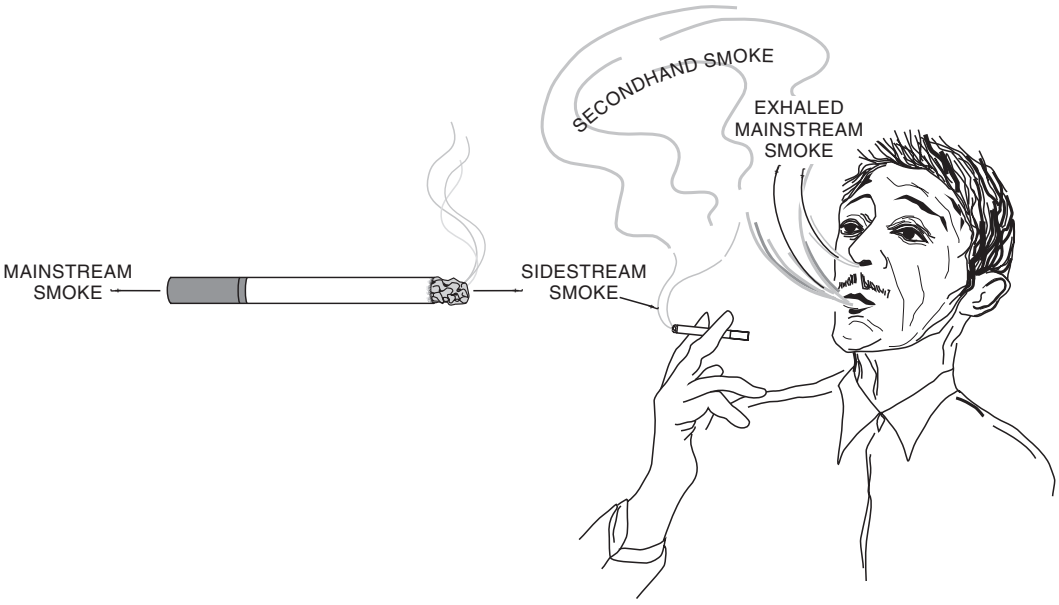

FIGURE 9.1 Mainstream smoke is inhaled by the smoker during puffing, and to a small extent diffuses through the cigarette paper. Between puffs, the smoker emits exhaled mainstream smoke, and the burning end of the cigarette emits sidestream smoke. The combination of sidestream (~90%) and exhaled mainstream smoke (~10%) is called secondhand smoke. An older term for secondhand smoke is environmental tobacco smoke.

9.3 POLLUTANTS FROM SHS

The tobacco smoke aerosol is a mixture of more than 4,000 chemical by-products of tobacco combustion, 500 of which are in the gas phase (Hoffmann and Hoffmann 1998). Of these SHS by-products, 172 are known toxic substances, many of which are regulated — except in the non-industrial indoor air environment, where most exposure takes place. SHS includes 3 criteria air pollutants and 33 hazardous air pollutants regulated under the Clean Air Act, 47 pollutants that are classified as hazardous wastes whose disposal in solid or liquid form is regulated by the Resource Conservation and Recovery Act, 67 known human or animal carcinogens, and 3 industrial chemicals regulated under the Occupational Health and Safety Act (Table 9.1). Nevertheless, although widely regarded as a major nuisance and worse by nonsmokers due to its irritating properties and health hazards, until the 1990s there were few successful attempts to regulate SHS exposure.

In the mid-1990s the U.S. Centers for Disease Control discovered that most nonsmokers of all ages had tobacco combustion products in their blood (Pirkle et al. 1996). By the late 1990s SHS had been linked to a wide variety of fatal and nonfatal diseases (Table 9.2), with credible estimates of the toll from passive smoking reaching as high as 60,000 U.S. nonsmoker deaths per year from all known or suspected causes (Wells 1999; CalEPA 1997). By comparison, for the period 1990–1994, the estimated total annual average death toll from active smoking was 431,000 persons, or 19.5% of all U.S. deaths (Centers for Disease Control 1997) and 8.8% of all the world's deaths annually (Brandt and Richmond 2004). Thus, in the United States, SHS pollution may be responsible for as many as 2.7% of all U.S. deaths annually.

TABLE 9.1
172 Toxic Substances in Tobacco Smoke, Including 33 Hazardous Air Pollutants (HAPs), 47 Chemicals Restricted as Hazardous Waste (HW), 67 Known Human or Animal Carcinogens, and 3 EPA Criteria Pollutants (CP)

Compound(s)[a] Known Human Carcinogen (*H*) Probable Human Carcinogen (*HP*) Animal Carcinogen (*A*)	Toxic = T, from references 1, 2, 3, 4, 5, 6, or 9; hazardous wastes = HW, reference 7; hazardous air pollutants = HAP reference 8; OSHA = Regulated Workplace Carcinogen, from reference 10
1. 1,1-Dimethylhydrazine *HP*	T[4,9] HW, HAP
2. 1,3 Butadiene	T[1,9] HAP
3. 1-Methylindole	T[5]
4. 2,6-Dimethylaniline *A*	T[9]
5. 2-Naphthylamine *A, H*	T[4,9] HW, OSHA
6. 2-Nitropropane *A*	T[4,9] HW
7. 2-Toluidine *A*	T[4,9] HW, HAP
8. 3-Vinylpyridine	T[4]
9. 4-(Methylnitrosamino)-1-(3-pyridil)-1-butanone (NNK) *A*	T[4,9]
10. 4,4-Dichlorostilbene	T[5]
11. 4-Aminobiphenyl *A, H*	T[4,9] HW, HAP, OSHA
12. 5-Methylchrysene *A*	T[4,9]
13. 7H-Dibenzo(c,g)carbazole *A*	T[4] HW
14. 9-Methylcarbazole	T[5]
15. AaC* *A*	T[9]
16. Acetaldehyde *A*	T[4,9] HW, HAP
17. Acetamide *A*	T[9]
18. Acetone	T[4]
19. Acetonitrile	T[1]
20. Acrolein	T[4] HW, HAP
21. Acrylonitrile	T[4,9] HW, HAP
22. Acrylymide *A*	T[9]
23. Alkylcatechols	T[5]
24. Ammonia	T[1]
25. Anabasine	T[3]
26. Aniline	T[1] HAP
27. Anthracenes (5)	T[2]
28. Antimony	T[2,5] HAP
29. Arsenic *H*	T[4,9] HW, HAP
30. Aza-arenes *A*	T[9]
31. Benz(a)anthracene *A*	T[4,9] HW
32. Benzene *A,H*	T[4,9] HW, HAP
33. Benzo(a)pyrene *A,H*	T[4,9] HW
34. Benzo(b)fluoranthene *A*	T[4,9] HW
35. Benzo(b)furan *A*	T[9]
36. Benzo(j)fluoranthene *A*	T[4,9]
37. Benzo(k)fluoranthene *A, HP*	T[4,9] HW
38. Benzofurans (4) *A*	T[2]
39. Beryllium *H*	T[9]
40. Butyrolactone	T[6]
41. Cadmium *H*	T[4,9] HW, HAP
42. Caffeic acid *A*	T[9]
43. Carbon monoxide	T[4]
44. Carbonyl sulfide	T[4]

TABLE 9.1 (CONTINUED)
172 Toxic Substances in Tobacco Smoke, Including 33 Hazardous Air Pollutants (HAPs), 47 Chemicals Restricted as Hazardous Waste (HW), 67 Known Human or Animal Carcinogens, and 3 EPA Criteria Pollutants (CP)

Compound(s)[a]
Known Human Carcinogen (*H*)
Probable Human Carcinogen (*HP*)
Animal Carcinogen (*A*)
	Toxic = T, from references 1, 2, 3, 4, 5, 6, or 9;
	hazardous wastes = HW, reference 7;
	hazardous air pollutants = HAP, reference 8;
	OSHA = Regulated Workplace Carcinogen,
	from reference 10

45. Catechol	T[4,9] HAP
46. Chromium VI *H*	T[4,9] HW, HAP
47. Chrysene	T[4] HW
48. Cobalt	T[9]
49. Cresols (all 3 isomers)	T[5] HW, HAP
50. Crotonaldehyde	T[4] HW
51. Cyanogen	T[2] HW, HAP
52. DDD	T[5,2] HW
53. DDE *A*	T[9]
54. DDT *A*	T[5,2,9] HW
55. Dibenz(a,h)acridine *A*	T[4,9] HW
56. Dibenz(a,j)acridine *A*	T[4,9] HW
57. Dibenz(a,h)anthracene *A*	T[4,9] HW
58. Dibenzo(a,e)pyrene *A*	T[9]
59. Dibenzo(a,i)pyrene *A*	T[4]
60. Dibenzo(a,l)pyrene *A*	T[4,9] HW
61. Dibenzo(c,g)carbazole *A*	T[9]
62. Dimethylamine	T[2,6]
63. Di(2-ethylhexyl)phthalate *A*	T[9]
64. Endosulfan	T[5] HW
65. Endrin	T[5,2] HW
66. Ethylbenzene	T[1,12]
67. Ethyl Carbamate *A*	T[4,9] HAP
68. Ethylene Oxide *A,H*	T[9]
69. Fluoranthenes (5)	T[2]
70. Fluorenes (7)	T[2] HAP
71. Formaldehyde	T[1,9] HW
72. Furan	T[2,9]
73. Glu-P-1* *A*	T[9]
74. Glu-P-2* *A*	T[9]
75. Hydrazine	T[4] HW, HAP
76. Hydrogen cyanide	T[4] HW
77. Hydrogen sulfide	T[1]
78. Hydroquinone	T[5,2] HAP
79. Indeno(1,2,3-c,d)pyrene *A*	T[4,9]
80. Indole	T[2]
81. IQ* *A, HP*	T[9]
82. Isoprene	T[2,9]
83. Lead ^{210} *A, H*	T[5,9] HW, HAP
84. Limonene	T[2]
85. Maleic hydrazide	T[5] HW
86. Manganese	T[5,2] HAP
87. Mercury	T[5,2] HW, HAP
88. Methanol	T[1] HAP

TABLE 9.1 (CONTINUED)
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	hazardous wastes = HW, reference 7;
	hazardous air pollutants = HAP, reference 8;
	OSHA = Regulated Workplace Carcinogen,
	from reference 10

89. Methyl formate	T[1]
90. Methyl chloride	T[3]
91. Methylamine	T[1]
92. Methyleugenol	T[9]
93. Naphthalene	T[1] HAP
94. Nickel *A,H*	T[4,9] HAP
95. Nicotine	T[4] HW
96. Nitric oxide	T[4]
97. Nitrobenzene *A, HP*	T[9]
98. Nitrogen dioxide	T[4]
99. Nitromethane *A*	T[9]
100. *N*-Nitrosodi-*n*-propylamine *A*	T[9]
101. *N*′-Nitrosoanabasine *A*	T[4]
102. *N*-Nitrosodiethanolamine *A*	T[4,9] HW
103. *N*-Nitrosodiethylamine *A*	T[4,9] HW
104. *N*-Nitrosodimethylamine *A*	T[4,9] HAP, HW, OSHA
105. *N*-Nitrosodi-*n*-butylamine *A*	T[9]
106. *N*-Nitrosoethylmethylamine *A*	T[4,9]
107. *N*-Nitrosomorpholine *A*	T[4] HAP
108. *N*′-Nitrosonornicotine *A*	T[4,9] HW
109. *N*-Nitrosopiperidine *A*	T[9]
110. *N*-nitrosopyrrolidine *A*	T[4,9]
111. NAT* *A*	T[9]
112. NNN* *A*	T[4]
113. Nornicotine	T[3]
114. Octane	T[1,12]
115. *o*-Toluidine	T[4] HW
116. Palmitic acid	T[2]
117. Parathion	T[5] HW
118. Phenol	T[2] HW, HAP
119. Phenols (volatile)	T[4] HW
120. PhlP *A*	T[9]
121. Picolines (3)	T[3]
122. Polonium^{210} *A, H*	T[4] HAP
123. Propionic acid	T[1]
124. Propylene oxide *A*	T[9]
125. Pyrenes (6)	T[2]
126. Pyridine	T[1] HW
127. Quinolines (7) *A*	T[2,9]
128. Resorcinol	T[5] HW
129. Styrene	T[1,9,12] HAP
130. Toluene	T[1] HAP
131. Trp-P-1* *A*	T[9]
132. Trp-P-2* *A*	T[9]

TABLE 9.1 (CONTINUED)
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133. Urethane	$T^{5,2}$
134. Vinyl chloride *H*	$T^{4,9}$ HW, HAP
135. Xylenes (3)	$T^{1,12}$
136. $PM_{2.5}$	CP
137. Nitrogen dioxide	CP
138. Carbon monoxide	CP

[a] From Tables 5, 6, 7, 8, or 9 in Reference 4 or 5.

* Abbreviations: (all PAHs): AaC, 2-amino-9H-pyrido[2,3-b]indole; IQ, 2-amino-3-methylimidazo[4,5,b]quinoline; Glu-P-1, 2-amino-6-methyl[1,2,-a:3′,2″-d]imidazole; Glu-P-2, 2-aminodipyrido[1,2-a:3′2″-d]imidazole; Phlp, 2-amino-1-methyl-6-phenylimidazo[4,5-b]pyridine; Trp-P-1, 3-amino-1,4,-dimethyl-5Hpyrio[4,3-b]indole; Trp-2, 3 amino-1-methyl-5H-pyrido[4,3,-b]indole. NAT, *N*′-nitrosoanatabine; NNN, *N*′-nitrosonornicotine.

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Note: The substances in Table 9.1 are all listed as constituents of tobacco smoke. Although few of them have actually been reported as measured in secondhand smoke, all of them have been measured in mainstream, and to a lesser extent, sidestream, smoke. Secondhand smoke consists of fresh and aged exhaled mainstream and sidestream smoke, and mainstream smoke is formed in the same burning cone as sidestream. Generally, sidestream and secondhand smoke contain greater total quantities of given chemicals (e.g., more NO_2 and more NNK), and are more toxic than mainstream smoke, which is formed at a higher temperature, and is also filtered by the tobacco rod and the cigarette filter.

TABLE 9.2
Health Effects Associated with Exposure to Secondhand Smoke

I. Effects Causally Associated with SHS Exposure

Developmental Effects

Fetal growth: low birth weight and decrease in birth weight
Sudden Infant Death Syndrome (SIDS)

Respiratory Effects

Acute lower respiratory tract infections in children (e.g., bronchitis and pneumonia)
Asthma induction and exacerbation in children and adults
Chronic respiratory symptoms in children
Eye and nasal irritation in adults
Middle ear infections in children

Carcinogenic Effects

Lung cancer
Nasal sinus cancer
Breast cancer

Cardiovascular Effects

Heart disease mortality
Acute and chronic coronary heart disease morbidity
Altered vascular properties

II. Effects with Suggestive Evidence of a Causal Association with ETS Exposure

Reproductive and Developmental Effects

Spontaneous abortion, intrauterine growth retardation
Adverse impact on cognition and behavior
Allergic sensitization
Decreased pulmonary function growth
Adverse effects on fertility or fecundability
Menstrual cycle disorders

Cardiovascular and Hematological Effects

Elevated stroke risk in adults

Respiratory Effects

Exacerbation of cystic fibrosis
Chronic respiratory symptoms in adults

Carcinogenic Effects

Cervical cancer
Brain cancer and lymphomas in children
Nasopharyngeal cancer
All cancers — adult and child

Source: Data from CalEPA (1997); CalEPA (2005).

9.4 SMOKING PREVALENCE AND TRENDS

Smoking prevalence varies by historical time period, by age group, and by group characteristics. During the period prior to 1990, two-thirds of U.S. children grew up in households with one or more smokers, and it was nearly impossible to find a job in a workplace where smoking was prohibited (Repace 1985). U.S. smoking trends show that cigarette smoking prevalence peaked in 1965 at 42.4%, with a notable race and gender disparity, and has steadily declined since (Figure 9.2). In 2001, 46.2 million adults (22.8%) in the U.S. were current cigarette smokers — 25.2% of men and 20.7% of women. Current U.S. smoking prevalence varies markedly by state, from a low of 13% in Utah to a high of 31% in Kentucky, and has become increasingly concentrated in lower income and less educated segments of the populace. Smoking prevalence also varies by age group; in the past decade, prevalence rates have been declining in all age groups except among persons 18 to 24, among whom prevalence has increased, from 23% in 1991 to 27% in 2000. Smoking is also higher among veterans than in the general population, with the prevalence of smoking among Vietnam War veterans at a very high 47%. Smoking prevalence also ranges from 50% to over 80% among persons with psychiatric or substance-abuse disorders, with one study estimating that such persons may account for 44% of all cigarettes smoked in the U.S. (Schroeder 2004); very high smoking prevalence is also common among the prison population. The declining prevalence of smoking and the proliferation of smoke-free workplace laws (in six states at this writing) has produced a reduction in population exposure to SHS, as evidenced by a 70% decline in blood (serum) cotinine, a biomarker for SHS exposure, from a median of 0.20 ng/ml in 1988–1991 to 0.059 ng/ml in 2003 (Pirkle et al. 1996; Centers for Disease Control 2003).

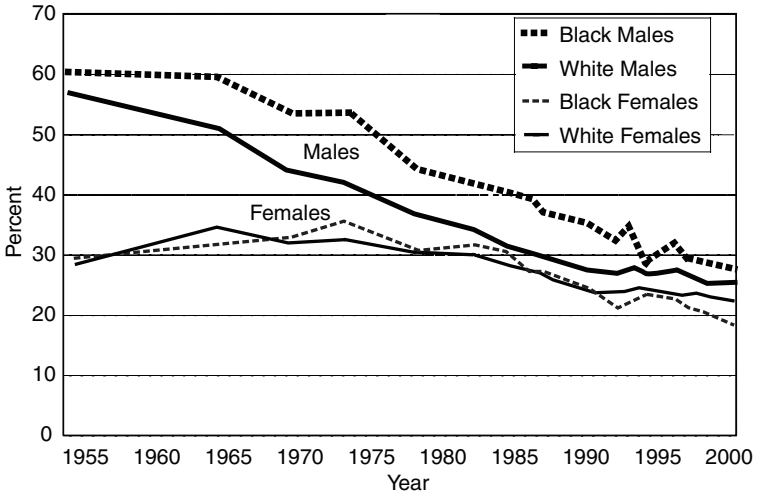

FIGURE 9.2 Trends in the prevalence of cigarette smoking among adults by race and gender in the U.S. 1955–2001. (From Shopland D., personal communication, 2003; National Cancer Institute. [1955 Data are based on the Census Bureau's Current Population Survey, data from 1965–2000 are based on estimates from the National Health Interview Survey, 1965–2001.])

9.5 WORKPLACE EXPOSURE

Workplace smoking policies are important determinants of exposure. While over three-quarters of white-collar workers are covered by smoke-free policies, including 90% of teachers, just 43% of the U.S.'s 6.6 million food preparation and service occupations workers benefit from this level of protection, and of these, only 13% of bartenders can avoid breathing smoke on the job (Shopland et al. 2004). Furthermore, under intense lobbying by tobacco-allied interests, 20 state legislatures have passed intentionally weak laws preempting local efforts to enact strict clean indoor air laws, which particularly limit smoking restrictions in the hospitality industry (Shopland et al. 2004, Schroeder 2004). Only six U.S. states ban smoking in all workplaces; of the remaining 44, a patchwork of local laws provides a mixture of bans, ventilation, air cleaning, designated smoking areas, and no restrictions. Moreover, even in California, one of the states with the most complete workplace restrictions, as of 2000, half of smokers continued to smoke inside the home.

9.6 DETERMINING EXPOSURE

9.6.1 Biomarkers for SHS: Body Fluid Cotinine

Microenvironmental air pollution contributes to total exposure as follows. The total inhaled SHS exposure of a person over a lifetime is the time-weighted sum over all of the microenvironments visited during each day of life, taken over all days lived, of the product of three factors: the concentrations of pollutants a person encounters in each microenvironment; the person's respiration rate during the exposure; and the duration of that person's exposure in each microenvironment. All individuals in the population have their own exposure profiles (Repace, Ott, and Wallace 1980). For secondhand smoke, an inhaled exposure of the atmospheric SHS marker, nicotine, would be absorbed through the lung and converted by the liver into a dose of its metabolites, of which cotinine is one, by the pharmacokinetics of the individual (Repace and Lowrey 1993; Repace et al. 1998; Benowitz 1999). What does this dose distribution look like? Figure 9.3 shows the distribution of serum cotinine, which reflects the total body burden of SHS, for a representative sample of all non-tobacco-using individuals in the U.S. population from 1989–1991. Figure 9.3 then reflects the U.S. population's cross-sectional exposure profiles, incorporating the concentrations of SHS nicotine to which each member of the population was exposed, duration of exposure, and the respiration rates of those nonsmokers during exposure, as modulated by individuals' metabolisms which combine to transform inhaled SHS exposure into actual dose of cotinine (Repace and Lowrey 1993; Repace et al. 1998; Benowitz 1999). In 1990, an estimated 25.5% of the adult population were current cigarette smokers (MMWR 1992), and workplace smoking restrictions were just beginning to be enforced in offices, but not in restaurants or bars.

9.6.2 Misclassification Problems

How much exposure to SHS do people receive? An obvious, but not necessarily accurate, way to find out is to ask a nonsmoker "Does your spouse smoke?" Assessing the level of exposure by questionnaire is highly subjective, since it depends upon an individual's sensitivity to SHS. The health risks of SHS have often been assessed in the epidemiological literature by comparing disease incidence in nonsmokers who live with smokers and who therefore are presumed to be SHS exposed, to that of nonsmokers who live with nonsmokers, and who are therefore presumed to be unexposed to SHS. There has been scant appreciation of how poor an exposure assessment such self-reports often yield. The problem of people not comprehending their true smoke exposure is illustrated by the Third National Health and Nutrition Examination Survey (NHANES III), the first nationwide survey in which both questionnaires and measurements of serum cotinine were combined. A major finding of the 1989–1991 NHANES III survey was that exposure of the U.S. nonsmoking population to SHS was pandemic, with 87.3% manifesting detectable levels of cotinine in the blood, despite

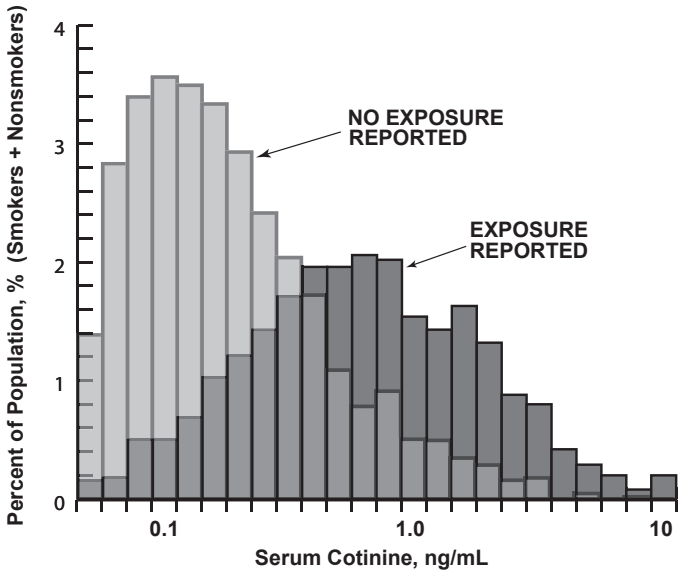

FIGURE 9.3 Distribution of non-tobacco users' blood (serum) cotinine (n = 6,056), reflecting SHS dose in a national probability sample of the U.S. population during 1989–1991 adapted from Pirkle et al. (1996). 88% of this nonsmoking population sample has detectable cotinine levels, showing widespread public exposure, but only 40% actually report SHS exposure on the NHANES III questionnaire. The intersection (medium gray) of the distribution of those reporting SHS exposure at work or at home (dark gray) with that of those reporting no exposure in those locations (light gray) indicates that many persons who report having no SHS exposure actually have exposures greater than those who do report having SHS exposure. (Note that the values on the horizontal axis represent the antilogs of the cotinine distribution.)

only 40% actually reporting SHS exposure. The geometric mean of those reporting exposure was much higher than those who did not (0.925 ng/ml vs. 0.132 ng/ml) (Pirkle et al. 1996). However, the frequency distribution of cotinine for the group reporting "no exposure" (histogram on the left side of Figure 9.3) overlapped with the frequency distribution for the group reporting exposure to SHS (histogram on right side of Figure 9.3). In this overlap zone, some nonsmokers who reported "no exposure to SHS" had higher cotinine levels than some nonsmokers who reported "exposure to SHS." Persons who reported no exposure but had high cotinine levels apparently were exposed without knowing it.

In a health effects study, an epidemiologist with only the questionnaire results available might erroneously assume that the persons who said they were not exposed had zero exposure. The overlap between the two groups, based on measured cotinine in their blood, reveals a serious statistical problem called "misclassification." For many people in this population, the questionnaire alone gives erroneous information about who was exposed and who was not. Because differences in observable health effects may be small, misclassification reduces the apparent magnitude of the health effect induced by SHS, as well as its statistical significance. On the other hand, misclassification can also work to increase apparent risk, as when smokers do not respond accurately about their smoking status; tobacco industry consultants (e.g., Lee and Forey 1996) have argued that this explains the observed risk elevation in epidemiological studies of passive smoking; however, the USEPA (1992) and others (Wells et al. 1998) persuasively rejected these arguments, concluding that this effect is a small fraction of the observed risk. This problem could be addressed (e.g., Repace, Hughes, and Benowitz in press) by directly measuring each person's cotinine or by asking respondents to wear a personal monitor to directly measure their exposure (see Chapter 6). Unfortunately, such measurements are rarely made in epidemiological studies.

Pirkle et al. (1996) reported that for children aged 2 months to 11 years, 43% lived in a home with at least one smoker. Both home and workplace environments contributed significantly to SHS exposure. Since the survey was based on a large national statistical sample with measured blood levels (n = 10,642 persons), the authors' conclusions are significant: "The high proportion of the population with detectable serum cotinine levels indicates widespread exposure to environmental tobacco smoke in the U.S. population." Since that time, U.S. nonsmokers' exposures have declined with the proliferation of smoking restrictions (Centers for Disease Control 2003).

9.7 SECONDHAND SMOKE AS MICROENVIRONMENTAL AIR POLLUTION

9.7.1 SHS Concentrations

High U.S. smoking prevalence, peaking at 57% of men and 34% of women in 1955, coupled with scant restrictions on indoor smoking until the late 1980s, led to widespread nonsmokers' exposures during this era (Schroeder 2004). In the late 1970s, two-thirds of homes, most offices, most factories and other workplaces, as well as trains, buses, ships, aircraft, theaters, college classrooms, doctors' offices, hospitals, waiting rooms, restaurants, bars, public buildings, stores, supermarkets, and small businesses, as well as social events, including parties, weddings, and dances, were smoke filled (Repace and Lowrey 1980, 1982; Repace 1985). A 1978 investigation of the range and nature of the nonsmoking public's exposure to SHS concluded that nonsmokers were exposed to significant air pollution burdens from smoking (Repace and Lowrey 1980), for two basic reasons. First, people on average spent 90% of their time indoors (Repace and Lowrey 1980), and, second, respirable suspended particulates (RSP) in microenvironments where smoking occurred greatly exceeded the concentrations of RSP encountered in smoke-free environments. Figure 9.4 shows ~20-minute-average Piezobalance measurements of RSP levels in 17 smoking (alphabetically labeled data) and 13 nonsmoking microenvironments (unlabeled) as a function of the density of burning cigarettes (D_s). The measurements shown in Figure 9.4 are of $PM_{3.5}$, also called respirable suspended particles (RSPs), an older generic term widely used in much of the SHS measurement literature, which includes the closely related fine particles (FP), or $PM_{2.5}$, a regulated (outdoor) air pollutant since 1997. Both fresh and aged SHS consist of FP. Both RSP and FP consist of fine solid or liquid particles that remain airborne for extended periods, are able to penetrate deep into the lung when breathed, and have slow pulmonary clearance times. (At the time of the study in Figure 9.4, the only regulated outdoor particulate air pollutant was total suspended particulate, TSP, which includes the nonrespirable particulate fraction between PM_{10} and PM_{50}. TSP was superseded by PM_{10}, which remains regulated, since it is inhaled by mouth breathers, but $PM_{2.5}$ appears to be the more serious health threat.) Indoor microenvironments, such as in homes, offices, restaurants, bars, theaters, lodge halls, bowling alleys, bingo games, and other commonly frequented venues, including emergency rooms at hospitals, all measured in Figure 9.4, were found polluted with RSP to a much greater extent than indoor nonsmoking environments such as homes, churches, and libraries or outdoors on city streets, or even in vehicles on busy commuter highways. The essential findings of this field study were that the nonsmoking microenvironment RSP levels ranged from about 20–60 $\mu g/m^3$, while the smoking microenvironment RSP levels ranged from about 90–700 $\mu g/m^3$, and that the RSP concentration differences between the smoking and nonsmoking microenvironments were due to tobacco smoke. As shown by the linear regression equation in Figure 9.4 (r^2 = 0.50), 50% of the variance in RSP levels is explained by the smoker density. Such levels of indoor air pollution, although ubiquitous in that era, were unpleasant for many nonsmokers; to place these measurements into a nonsmoker's perspective, Junker et al. (2001) reported an odor acceptability threshold of 1 $\mu g/m^3$ for RSP from SHS, and an irritation threshold of 4.4 $\mu g/m^3$. Viewed from the perspective of early 21st century air quality health advisories for fine-particle pollution ($PM_{2.5}$), if the $PM_{3.5}$ air quality levels of RSP in these typical 20th century smoking microenvironments had

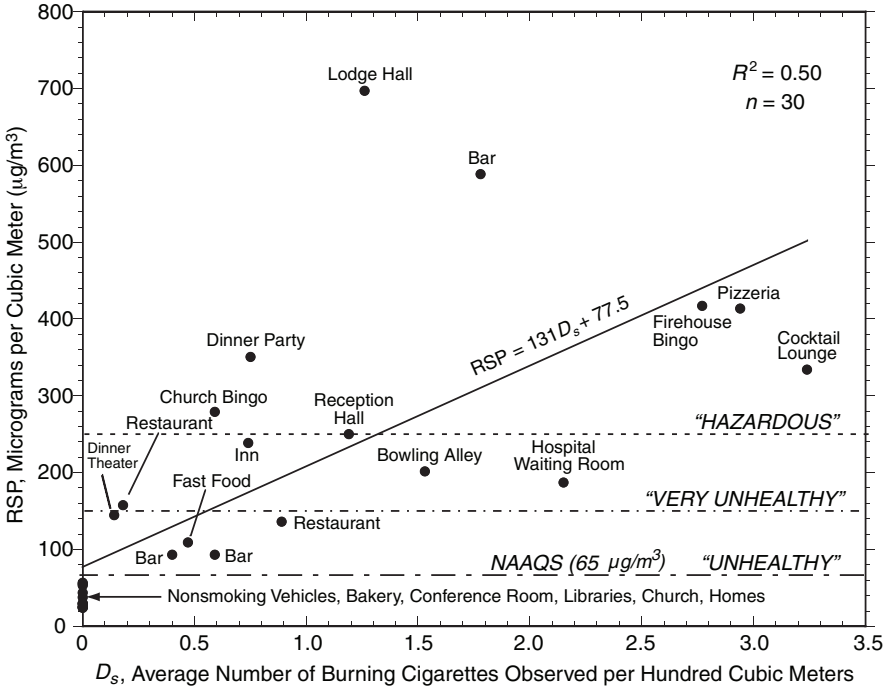

FIGURE 9.4 Respirable particle mass concentration (RSP) vs. smoker density for 17 smoking microenvironments and 13 nonsmoking microenvironments in the Washington, DC metro area in the Spring of 1978 (data adapted from Repace and Lowrey 1980; 1982). The locations with active smoking included restaurants, bars, nightclubs, bingo games, a bowling alley, and a hospital waiting room. The smoke-free locations (D_s = 0) included 5 homes, 2 libraries, a church, a bagel bakery, and a conference room in an office building. Most visits lasted for about 20 minutes. Environmental Protection Agency health-based short-term air quality advisories for $PM_{2.5}$ in the outdoor air are shown for comparison as "Unhealthy," "Very Unhealthy," and "Hazardous." (Data from Repace and Lowrey 1980, 1982.)

been interpreted using the current outdoor air criteria, they would have been judged variously as *Unhealthy (Code Red)*, *Very Unhealthy,* or *Hazardous*, as shown by the horizontal dashed lines in Figure 9.4.

9.7.2 Smoking Rates and Emissions

The concentrations of SHS to which the population are exposed will depend upon the smoking prevalence and the number of cigarettes, pipes, and cigars smoked by each smoker per unit time in each of the microenvironments that the nonsmoking population frequents. Because the vast majority of smokers smoke cigarettes, the treatment given here will focus on cigarettes. To illuminate the smoking rates that contributed to the nonsmokers' exposures shown in Figure 9.4 and doses shown in Figure 9.3, consider the following. In 1994, among U.S. adults aged ≥18 years, there were 48.0 million adult current smokers, of whom 25.3 million were men, and 22.7 million were women. These persons smoked 485 billion cigarettes, and thus smoked 10,104 cigarettes per smoker per year, or ~28 cigarettes per smoker per day (MMWR 1996; USDA 1995; Maxwell 1995). If one assumes a 16-hour smoking day, this is equivalent to about 1.8 cigarettes per smoker-hour.

RSP is the major pollutant emitted by burning tobacco products, and cigars emit three times as much RSP as cigarettes (Repace, Ott, and Klepeis 1990; Klepeis, Ott, and Repace 1999). Figure 9.5 shows a histogram of the SHS RSP emissions of the top 50 brands of cigarettes (Nelson 1994; Martin et al. 1997). These data are useful for showing the range in RSP emissions of cigarettes,

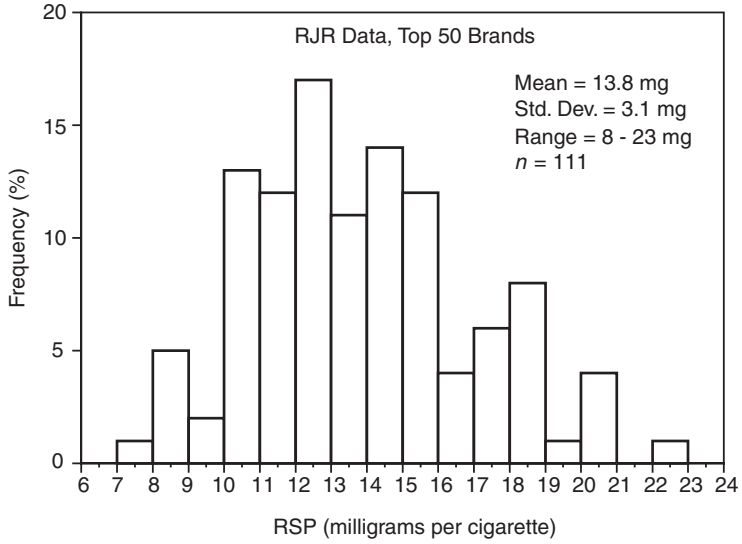

FIGURE 9.5 Histogram of SHS RSP emission factors for the top 50 brands of cigarettes, representing 65.3% of the U.S. cigarette market (plus a University of Kentucky research cigarette K1R4F), when two of each brand were smoked for 11 minutes each by human smokers in 1994. Data were digitized from a plot presented by RJ Reynolds Tobacco Company in testimony before the Occupational Safety and Health Administration (OSHA) hearing on OSHA's proposed Indoor Air Rule in Washington, DC in January 1995 (Nelson 1994). Other details from this study were published by Martin et al. (1997), which reported an overall sales-weighted RSP emission average of 13.67 mg/cigarette (Standard Error, 0.4106 mg/cigarette), $n = 100$.

explaining one source of the variation in RSP levels in field studies of the impact of smoking on indoor air. Note, however, that the actual variation in emissions may be smaller because only a few prominent brands dominate sales. In 2003, the top five cigarette brands alone accounted for two thirds of total U.S. cigarette consumption. Marlboro is the most popular brand, with sales greater than the five leading competitors combined. The market share for Marlboro was 37.5%, followed by Newport (8.0%), Doral (6.3%), Camel (6.1%), Winston (4.6%), and Basic (4.4%) (Maxwell 1995). The range in RSP emissions of cigarettes is determined primarily by the differences in mass when smoked identically.

9.7.3 A Person's Daily Exposure to Particulate Air Pollution

Epidemiological studies (studies of the causation of disease) have linked increases in daily average outdoor inhalable (PM_{10}) exposures to increased morbidity and mortality, effects that are stronger for finer particles ($PM_{2.5}$) (Holgate et al. 1999). There is recent evidence that even shorter-term $PM_{2.5}$ exposures can have cardiopulmonary health effects (Pope and Dockery 1999). For this reason, particulate air pollution is regulated under the Clean Air Act, and $PM_{2.5}$ is subject to an annual average health-based National Ambient Air Quality Standard (NAAQS) of 15 $\mu g/m^3$ in the outdoor air, and a 24-hour NAAQS of 65 $\mu g/m^3$. RSP from SHS is at least as toxic as outdoor RSP, and likely far more toxic, as illustrated by Table 9.1.

If a person carries a personal air pollution monitor for 24 hours as he or she visits various microenvironments, a picture emerges of that person's total daily exposure. Figure 9.6 (Repace, Ott, and Wallace 1980) shows one of the first experiments designed to investigate this issue:

> On October 16, 1979, I awoke to a new day in my home, commuted 25 miles from my residence in suburban Maryland to the Environmental Protection Agency (EPA) headquarters in Washington, DC, where I had a private smoke-free office. During my waking hours, I carried along a portable, battery-powered TSI Model 3500 Piezobalance (see Chapter 6) to measure RSP (each datum represents at least

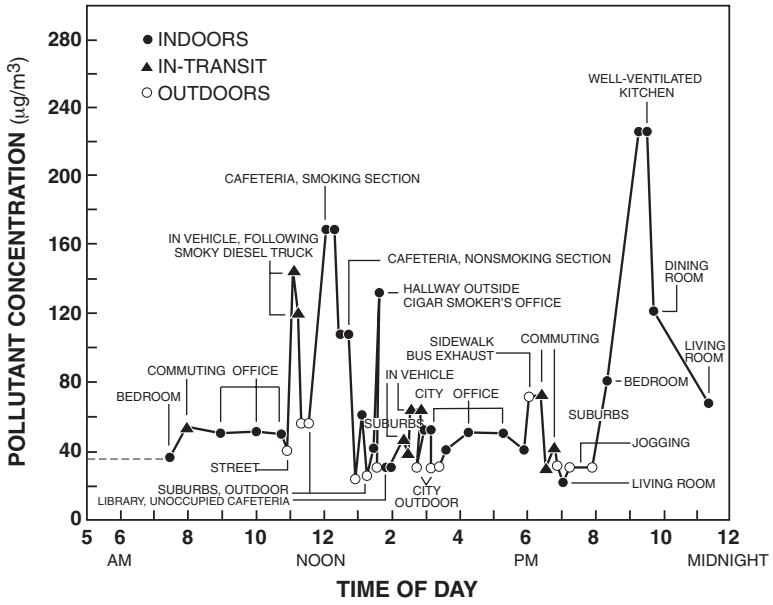

FIGURE 9.6 A nonsmoker's 24-hour exposure to RSP on October 16, 1979, illustrating a person's air pollution exposure in the metropolitan Washington, DC area, on one day of his life. A series of these daily patterns comprise an individual's lifetime exposure.

ten 2-minute $PM_{3.5}$ averages). I next traveled by car to the Goddard Space Flight Center in suburban Maryland, partly stuck in traffic behind a smoky diesel truck. I dined in the Goddard cafeteria, followed by a short tour of various buildings. The RSP level in the smoking section of the cafeteria was 55% greater than in the nonsmoking section, and 8.5 times greater than levels outdoors. On my return trip to Washington, RSP levels were much lower in the absence of diesel exhaust. Another period of a few hours in my smoke-free office was followed by a second encounter with diesel bus exhaust as I walked along the city sidewalk. My commute back to my suburban residence was followed by jogging outdoors, then dinner. Despite a powerful kitchen ceiling exhaust fan, roasting chicken in the electric oven caused high particulate levels in the kitchen, which penetrated into the dining and living rooms.

The data in Figure 9.6 show that I spent 84% of my time indoors, 9% in transit, and 7% outdoors. My total 24-hour integrated exposure for the day was 1428 microgram-hours per cubic meter (μg-h/m^3), equivalent to a 24-hour average exposure concentration of 59.5 μg/m^3. Contributions to my total RSP exposure break down into 82% from indoor microenvironments, 10% from in-transit microenvironments, and 8% from outdoors. (Note that even though high levels of RSP due to diesel exhaust were encountered in transit, the contribution to total exposure was still little more than that expected from the fraction of time spent traveling.) On this day, indoor RSP levels are generally higher than outdoors; diesel exhaust produces higher RSP levels than gasoline-powered vehicles; both cooking and smoking produced the highest RSP levels of the day. More recent field studies of personal exposure to particulate matter, such as the USEPA particle total exposure assessment methodology (PTEAM) studies (Özkaynak et al. 1996) have confirmed that smoking, cooking, and other indoor activities are major sources of human exposure to particulate matter.

9.8 ATMOSPHERIC TRACERS FOR SHS: NICOTINE AND RSP

What is the best atmospheric marker for SHS, a mixture of over 4,000 chemicals? Nicotine, a semi-volatile organic compound unique to tobacco smoke, is the most commonly used atmospheric tracer for SHS. Hammond (1999) has reviewed a number of field measurements of nicotine concentrations

in homes, offices, restaurants, etc. These "opportunistic" (that is, not necessarily representative) samples using both personal or area monitors, with relatively long averaging times from an 8-hour work shift to a day or a week or more, show that workplaces with smoke-free policies generally have measured nicotine levels below 1 μg/m^3. On the other hand, mean nicotine concentrations in workplaces that allow smoking generally range from 2–6 μg/m^3 in offices, 3–8 μg/m^3 in restaurants, and from 1–6 μg/m^3 in blue-collar workplaces, compared to 1–3 μg/m^3 in the homes of smokers (Hammond 1999). However, workplace concentrations have been highly variable, with some studies reporting concentrations more than 10–100 times the average home levels. For example, bars and discos have had measured median nicotine concentrations from SHS ranging from 19 μg/m^3 to 122 μg/m^3 (Nebot et al. 2005).

Field-deployable exposure monitors for nicotine use either active pump-and-filter methods, with minimum averaging times of about 15 minutes, or passive techniques with minimum averaging times of about a day. Both methods require chemical analysis. Repace et al. (1998) developed a model for the prediction of time-averaged nicotine concentrations based upon smoker densities and design ventilation rates, making it possible to generalize measured nicotine data if smoker densities are also measured.

Although RSP is not unique to SHS, measurement of RSP concentrations from SHS is important because the fine-particulate fraction of RSP, $PM_{2.5}$, is a regulated outdoor air pollutant with health-based national air quality standards, and daily air quality advisories are widely reported in the media in qualitative terms, using an Air Quality Index (AQI). Available real-time RSP monitors have averaging times of minutes or seconds, and do not require laboratory analysis, (see Chapter 6). Therefore, these monitors are useful for real-time studies of the growth and decay of individual cigarette smoke concentrations, as well as for short-term field surveys of SHS, as will be described later in this chapter. Cigarettes (and pipes, and cigars as well) have major emission components in the $PM_{2.5}$ size and range (Klepeis et al. 2003; National Research Council 1986). A number of studies have examined the SHS-RSP emissions of tobacco products. Martin et al. (1997) reported that the average gravimetric SHS-RSP yield was 13.7 mg/cigarette (standard error [SE] 0.4 mg/cigarette, n = 100) for the 50 top-selling U.S. cigarette brand styles in 1991. Martin et al. (1997) measured "RSP" both using a Piezobalance and a 1-μm pore gravimetric filter, but did not otherwise characterize the particle size distribution. The yield estimated by Piezobalance data was slightly lower, at 11.55 mg/cigarette (SE 0.36 mg/cigarette, n = 100). The SHS was generated by human smokers in a test chamber, who smoked each cigarette for 11 minutes. This value is in good agreement with the data presented in Figure 9.5, and with the ~14 mg/cigarette value ($PM_{2.5}$) obtained by regression analysis from the USEPA's Riverside PTEAM study (Wallace 1996). Dr. W.R. Ott (personal communication 2003) observed smoking times for 33 people in a Las Vegas casino reporting a mean of 9.25 minutes (SD = 2.3 min). Assuming a smoking time of 9.25 minutes and a cigarette emission factor of 1.43 mg/min, as reported by Klepeis, Ott, and Switzer (1996) for smoking lounges in San Francisco and San Jose, CA, airports, this yields an estimated RSP ($PM_{3.5}$) yield of 13.2 mg/cigarette. Repace and Lowrey (1980) observed a smoking rate of 9.8 minutes per cigarette in seven smokers, suggesting little change in cigarette smoking duration over a period of 2 decades. Industry studies report a range of about 8 to 23 mg/cigarette for RSP emissions from cigarettes (Figure 9.5), so that controlled experiments performed with individual cigarettes may differ. It is reasonable to assume a default emission rate of approximately14 mg/cigarette and a smoking duration of ~10 min/cigarette.

For SHS nicotine, Martin et al. (1997) reported that the average measured SHS nicotine yield for the 50 top-selling U.S. cigarette brand styles in 1991 was 1,585 μg/cigarette (standard error, SE 42.21 μg/cigarette, n = 100), yielding an RSP-to-nicotine emission ratio of 8.6 for SHS from these cigarettes. Daisey (1999) in an excellent discussion of atmospheric tracers for SHS, observes that nicotine can be used to estimate RSP exposures provided that smoking occurs regularly in the microenvironment, that the system is in a steady state, and that the sampling time is longer than the characteristic times for removal processes. Under these conditions, the ratio of RSP to nicotine

from SHS is approximately 10:1 (Nagda et al. 1989, Leaderer and Hammond 1991, Daisey 1999). This 10:1 ratio permits body fluid cotinine (e.g., Figure 9.3) which is derived from atmospheric nicotine, to be related to that portion of the RSP air pollution exposures of the population that are due to SHS using pharmacokinetic models (Repace and Lowrey 1993; Repace, Hughes, and Benowitz in press; Repace, Al-Delaimy, and Bernert 2006).

9.9 TIME-AVERAGED MODELS FOR SHS CONCENTRATIONS

Models for SHS concentrations are important for the prediction of human exposures in indoor air quality, epidemiological, or forensic investigations, to generalize field measurements (Repace 1987), to evaluate putative SHS control measures such as ventilation or air cleaning, as well as to debunk SHS junk science (Repace 2004a; Ott 1999; Repace and Lowrey 1995). As described in Chapter 18, mathematical models have been developed for predicting indoor air concentrations for a variety of sources, including cigarettes. These models are derived from the mass-conservation law of physics. The only other required assumption is that SHS concentrations be reasonably spatially uniform at any instant of time, as when air motion and convection cause the smoke in a room to mix rapidly with the air. The spatial variation of the concentration in a room may not be uniform while a point source is emitting, because concentrations usually are higher very close to the source (McBride et al. 1999), but concentrations in a room, or even a home, often become spatially homogeneous soon after the source stops emitting (Klepeis, Nelson, Ott et al. 2003; see Chapter 18 of this book).

Consider a particle emitted from a point source in a well-mixed room with emission rate $g(t)$ and air exchange rate a with pollutant-free outdoor air and a deposition rate k on the room surfaces (Ott, Langan, and Switzer 1992). The exchange with outdoor air is due to natural or mechanical ventilation and is measured as the number of room air changes per unit time. The deposition rate is also measured in number of (equivalent) air changes per unit time. The total air exchange rate is then $\phi = a + k$. If the initial concentration is $x(t) = 0$ in the room at time $t = 0$, then the following expression based on the mass balance model (from Chapter 5) gives the relationship between the mean concentration over averaging time $T = (t_f - t_0)$, (where t_f is the end of the averaging period) the mean emission rate over time T, and the instantaneous concentration $x(T)$, in typical units:

$$\overline{x(T)} = \frac{\overline{g(T)}}{\phi v} - \frac{\tau}{T} x(T) \tag{9.1}$$

where

$\overline{x(T)}$ = average indoor concentration over time T ($\mu g/m^3$)

$\overline{g(T)}$ = average source emission rate over time T ($\mu g/hr$)

$x(t)$ = instantaneous concentration at time t ($\mu g/m^3$)

v = volume of the well-mixed room (m^3)

ϕ = total loss rate of particles due to both air exchange and deposition (h^{-1})

τ = mean residence time $1/\phi$ (h)

As discussed in Chapter 5, the last term on the right-hand side of Equation 9.1 includes the ratio of the residence time to the averaging time τ/T, which generally becomes small as the averaging time becomes large relative to the residence time.

Example 1. A typical home with the exterior doors and windows closed might have an air exchange rate of $a = 0.5\ h^{-1}$, and a deposition rate $k = 0.3\ h^{-1}$, which corresponds to a mean residence time of $\tau = 1/\phi = 1/(0.8\ h^{-1}) = 1.25$ hours. If the averaging period were $T = 10$ hours, then the ratio would be $\tau/T = 1.25/10 = 0.125$. Thus, in many practical situations, the rightmost

term in Equation 9.1 is negligible, and setting this term equal to zero gives the following approximate relationship for the average concentration as a function of the average source emission rate:

$$\overline{x(T)} \cong \frac{\overline{g(T)}}{\phi v} \tag{9.2}$$

Example 2. Consider a single cigarette smoked for 10 minutes in a room beginning at time $t = 0$, with a constant RSP emission rate of 1.4 mg/min. Assume an air exchange rate of $a = 0.5$ h^{-1}, a deposition rate of $k = 0.39$ h^{-1} (Özkaynak et al. 1996) so that $\phi = 0.89$ h^{-1}, and a room volume of $v = 43$ m^3 (the size of a small bedroom). To calculate the mean concentration in the room over an 8-hour period beginning at $t = 0$, we first calculate the mean emission rate over the 8-hour period, $\overline{g(8)}$ = (10 min) (1.4 mg/min)/(8 hr) = 1.75 mg/hr. Substituting this average emission rate into the numerator of Equation 9.2 and using $\phi = a + k$ gives:

$$\overline{x(8)} \cong \frac{1.75 \text{ mg/hr}}{(0.5 \text{ hr}^{-1} + 0.39 \text{ hr}^{-1})(43 \text{ m}^3)} = 45.7 \times 10^{-3} \text{ mg/m}^3 = 45.7 \text{ µg/m}^3 \tag{9.3}$$

9.9.1 The Active Smoking Count

Some investigators have formally named the number of cigarettes smoked over a specific time period as the "Active Smoking Count (ASC)" (Ott, Switzer, and Robinson 1996). One can consider either the instantaneous ASC $n(t)$ or the expected value, or average, ASC of $\overline{n}_{cig}$. Using this notation, the average emission rate will be $\overline{g(T)} = \overline{n}_{cig}\,\overline{g}_{cig}\,/T$, where $\overline{g}_{cig}$ is the average emission rate per cigarette. How is the ASC measured? As an example of the timed observation of natural smoking activity patterns of 8 smokers, consider Harry's Hofbrau, a sports tavern in Redwood City, California, sampled in 1995 (Figure 9.7; W.R. Ott personal communication 2005). Averaged on a minute-by-minute basis, the mean number of cigarettes (ASC) being smoked at any one time in Figure 9.7 is $\overline{n}_{cig} = 2.213$ active smokers. In the typical field study where the investigator is recording room sizes, counting cigarettes and people in a large crowd, measuring concentrations, and trying to look inconspicuous, it is difficult to time individual smokers. Accordingly, Repace and Lowrey (1980) developed an approximate method for estimation of the ASC: they assumed the estimated 1978 U.S. national average smoking rate of 2 cigarettes per hour, found empirically that it took about 10 minutes to smoke a cigarette, and concluded that a typical smoker might be expected to spend one third of the hour actively smoking. Based on these assumptions, Repace and Lowrey (1980) demonstrated that an investigator could arrive at an empirical estimate of $\overline{n}_{cig}$ inside a bar or tavern with smokers by counting the number of actively burning cigarettes approximately every 10 minutes while walking around the location, and use this value to estimate the number of smokers present. For example, in the real-life tavern smoking pattern of Figure 9.7, if the number of burning cigarettes is counted every 10 minutes beginning at 8:40 P.M., the result is 3, 4, 2, 2, 2, and 2, for an average of 2.5 cigarettes (close to the more accurate minute-by-minute calculation of 2.2 above); if this is multiplied by 3, it yields an estimated 7.5 smokers present. If 6 of the 8 smokers were counted present for the full observation period; and the remaining 2 (who were not) are counted as being present for a half-cycle of smoking, then an average of 7 smokers would be estimated present over the full period. Thus, in crowded field studies, such as in stand-up bars where the ASC cannot be measured with a fine time resolution, the empirical method of Repace and Lowrey (1980) can be used to provide an estimate of the ASC, as well as an estimate of the total number of smokers present. A better, but often infeasible, alternative would be to collect and count the number of cigarette butts smoked during the averaging time T, and estimate the ASC by assuming an average cigarette burning time, e.g., 10 minutes. Ott, Switzer, and Robinson (1996) found in 52 visits to

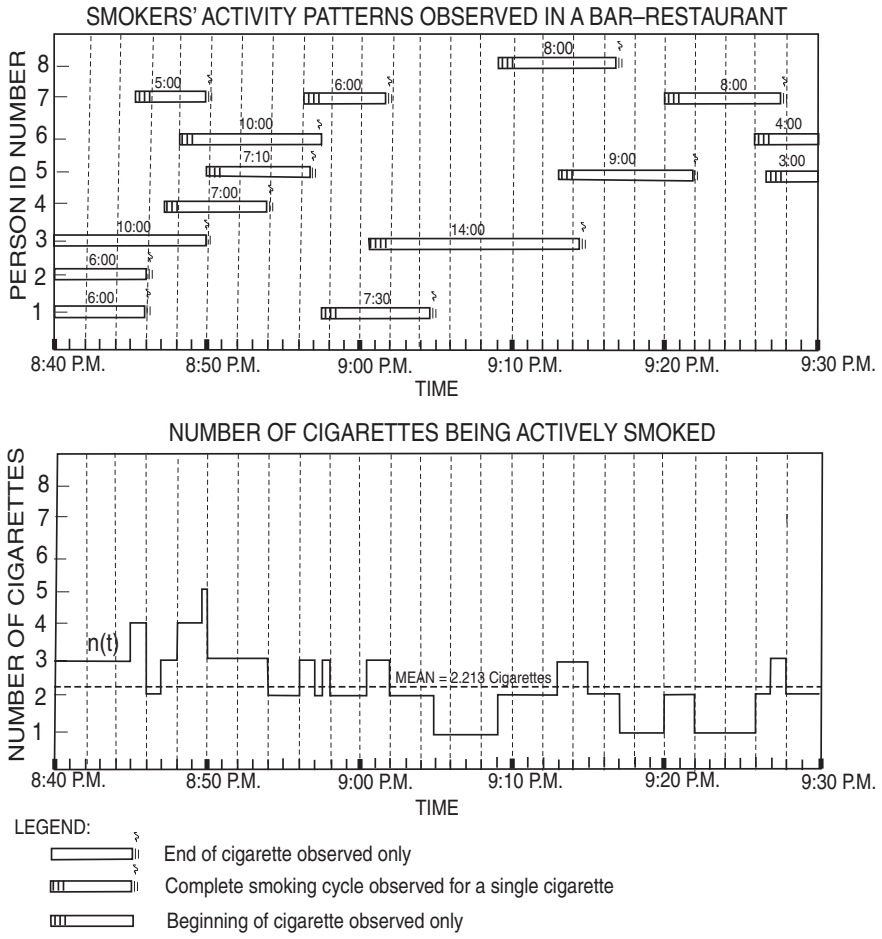

FIGURE 9.7 The smoking time-activity pattern for 8 smokers recorded using a stopwatch and a diary in Harry's Hofbrau in Redwood City, CA, on December 21, 1996 (top). (From W.R. Ott, personal communication, 2005.) The upper figure shows the number of minutes each smoker smoked and the time at which the cigarette was smoked. Smoker #1 departed after smoking two cigarettes, and smoker #8 arrived about 9 P.M. The lower part of the graph shows the actual physical observations in surveys of smoking: $n(t)$, the instantaneous number of cigarettes being smoked. The mean number of cigarettes being smoked over the 50-minute observation period is $n_{ave} = 2.213$ cigarettes.

the Oasis Bar in Redwood City, CA, the measured average RSP concentration from SHS had a correlation coefficient $R^2 = 0.61$ with the active smoking count, similar to the value of $R^2 = 0.50$ derived from the Washington, DC area studies of Repace and Lowrey (1980, 1982) shown in Figure 9.4, and the value of $R^2 = 0.54$ derived by Repace (2004a) for 8 establishments in metropolitan Wilmington, DE.

The ASC concept can also be used to derive a simple model for estimating RSP concentrations based on the number of smokers using certain default assumptions. In many applications, regulators, risk assessors, and investigators making field measurements under real-world uncontrolled conditions are in need of simple models for the prediction of SHS concentrations or the assessment of ventilation rates from measurements of concentrations. The Habitual Smoker Model, based upon Equation 9.2, was developed for these purposes.

9.9.2 The Habitual Smoker Model (Equation 9.2 with Defaults)

In order to be able to relate observations of burning cigarettes, room sizes, and the design ventilation rates of the American Society of Heating, Refrigerating, and Ventilating Engineers (ASHRAE 1973 to 1989 et seq.) to measured concentrations of RSP from SHS, Repace and Lowrey (1980, 1982, 1985) introduced the Habitual Smoker Model (HSM). The HSM is simply derived from Equation 9.2 as follows. Assume an RSP emission rate of 1.4 mg/min-cigarette, a cigarette smoking time of 10 min/cigarette, and an average smoking rate of two cigarettes per smoker per hour. Then, over a 1-hour averaging period, if an average of $\bar{n} = 1$ burning cigarette is observed, an average of 6 cigarettes per hour will have been smoked (1 cig/ten minutes)(6 10-minute periods/hour), and Equation 9.2 is written as: $\bar{x}(T) = \bar{g}(T)/\phi v = \bar{x}(1\,\text{h}) = \bar{g}(1\,\text{h})/\phi v$ = [(6 cig/h) (1.4 mg/min-cig) (10 min/cig)]/[ϕv] = (84 mg/cig-h/ϕv) in units of milligrams per cubic meter per cigarette. If the RSP mass emission is expressed in units of micrograms and the space volume in units of hundred cubic meters, then the average number of burning cigarettes per 1-hour averaging time yields a concentration of $\bar{x}(1\,\text{h}) = [\overline{ng}(1\,\text{h})/\phi(100v)](1000\ \mu g/mg) = 840(\bar{n}/v')/\phi$, in units of micrograms per cubic meters, where v' is defined as the space volume expressed in practical units of hundred cubic meters.

If we define $(\bar{n}/v') = D_s$ as the active smoker density (in units of burning cigarettes per hundred cubic meters) and $\phi = a + k$ as before, then

$$\bar{x} = 840\ D_s/\phi\ [\mu g/m^3] \tag{9.4}$$

If we further assume that *all* smokers have identical smoking patterns (2 cigarettes per hour, defined as "habitual smokers") then the number of habitual smokers N_{hs} is three times the ASC, or expressed in terms of smoker densities, the habitual smoker density $(N_{hs}/v') = D_{hs} = 3D_s$. With this substitution, $\bar{x} = 840(D_{hs}/3\phi)$, or

$$\bar{x} = 280\ D_{hs}/\phi\ [\mu g/m^3] \tag{9.5}$$

where D_{hs} is in units of habitual smokers per 100 m^3, and ϕ is in units of inverse hours (i.e., in air changes per hour from ventilation and its equivalent for deposition).

For modeling purposes for existing smoking-permitted commercial premises such as restaurants, bars, and casinos, design ventilation rates q (usually expressed in units of cubic feet per minute per person [P] or liters per second per person) are recommended by ASHRAE Standard 62, Ventilation for Acceptable Indoor Air Quality (ASHRAE pre-2004 versions), where q is related to a by the equation $q = av/P$, in consistent units. While q and a are readily measurable, deposition rates k are not as well understood, and may vary depending upon room characteristics such as surface area, surface type, ventilation type, and perhaps airflow rates in the room. I have long used Equation 9.4 and Equation 9.5 with the default assumption $\phi = 1.3a$ (Repace and Lowrey 1980, 1982; Repace 2004a). In summary, the Habitual Smoker Model has been used when it has not been possible to make measurements of a, k, and $\bar{g}$, as for example, in risk assessment or in forensic applications where the SHS exposure takes place in the past or future and default assumptions are required. Using this assumption, the chamber data of Leaderer, Cain, Isseroff, and Berglund (1984) were predicted reasonably accurately by Repace and Lowrey (1982). Equation 9.2 should always be used with exact parameter values whenever available.

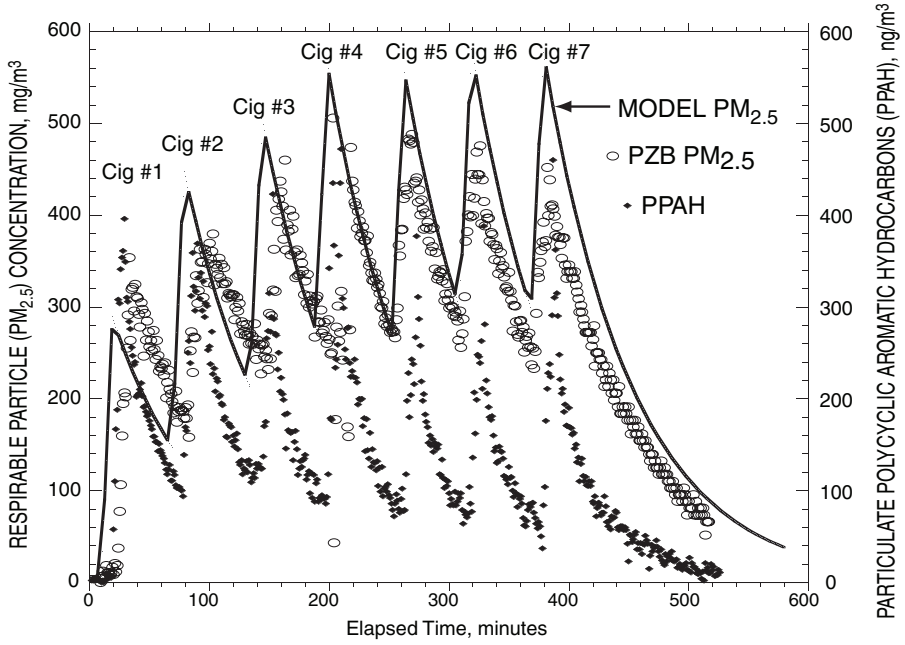

FIGURE 9.8 $PM_{2.5}$ and PPAH sidestream smoke concentrations for a series of seven Marlboro 100 medium cigarettes smoldered for 10 minutes each at the approximate rate of 1 per hour. The experiment was conducted in a closed 41 m^3 bedroom with total decay rates $\phi_{PM2.5} = 0.8\ h^{-1}$, and $\phi_{PPAH} = 1.45\ h^{-1}$, in a single-family home in Redwood City, CA. These measurements were made using a Model 3511 Piezobalance for RSP and an EcoChem PAS2000CE for PPAH. The model (Ott, Langan, and Switzer 1992; see also Chapter 18) assumes a 14 mg/cigarette RSP emission strength and uses the measured decay rate for RSP.

9.10 TIME-VARYING SHS CONCENTRATIONS

The equations given above are time-averaged equations that average over a typically fluctuating concentration of SHS during the smoking period, as distinct from time-dependent models (Ott, Klepeis, and Switzer 2003). This fluctuation is illustrated by the following controlled experiment. Figure 9.8 shows the concentration as a function of time (i.e., the *time series*) for RSP and particle-bound polycyclic aromatic hydrocarbons (PPAH) concentrations for a series of 7 Marlboro cigarettes smoldered (i.e., producing sidestream smoke only) at the approximate rate of 1 cigarette per hour, in a 41 m^3 bedroom of a detached home in Redwood City, CA, with the windows and bedroom door closed. These measurements were made using the Piezobalance for RSP and the EcoChem PPAH monitor described in Chapter 6 of this book. This experiment illustrates how PPAH and RSP concentrations rise and fall as cigarettes burn and are put out; it also illustrates the faster rate at which PPAH are removed from the room air relative to RSP from SHS, shows how this faster rate results in the PPAH oscillations stabilizing about the mean more quickly than RSP does, and the PPAH decaying back to background more rapidly after the last cigarette is smoked. The removal rate for each class of SHS compound may be calculated from the slope of the final decay curve when it is plotted on a semi-logarithmic graph.

What are PPAH? PPAH are part of a broader class of gas- and particulate-PAH, a group of more than 100 different molecules formed during the incomplete combustion of organic material such as tobacco, fossil fuels, or wood, and are usually found in complex mixtures such as soot or tar (see also Chapter 14). PPAH are generally formed in the gas phase with subsequent transition to the particulate form. PAH have at least two benzene rings sharing a common border; 2- and 3-ring PAH generally exist in the vapor phase in the atmosphere, whereas 5- and 6-ring PAH are

TABLE 9.3
Carcinogenic PPAH, IARC Status, Amount in Cigarette Smoke[a]

Particulate Phase PAH (PPAH) with Four or More Rings[b]	IARC Carcinogen in Lab Animals (A) Humans (H)	Amount Measured in Mainstream Smoke (MS) (ng/cig)[c]	Amount Measured in Sidestream Smoke (SS) or SHS (ng/cig)[c]	Reference
Benz(a)anthracene	Sufficient (A)	20–70	412	Hoffmann and Hoffmann (1998); Gundel et al. (1995); IARC (2004)
Benzo(b)fluoranthene	Sufficient (A)	4–22	132	Hoffmann and Hoffmann (1998); Gundel et al. (1995); IARC (2004)
Benzo(j)fluoranthene	Sufficient (A)	6–21	32	Hoffmann and Hoffmann (1998), Gundel et al. (1995); IARC (2004)
Benzo(k)fluoranthene	Sufficient (A)	6–12		Hoffmann and Hoffmann (1998), IARC Monographs (2004)
Benzo(a)pyrene	Sufficient (A,H)	20–40 8.5–11.6	74	Hoffmann and Hoffmann (1998) Gundel et al. (1995); IARC (2004)
Dibenzo(a,e)pyrene	Sufficient	present		IARC (2004)
Dibenzo(a,i)pyrene	Sufficient (A)	1.7–3.2		Hoffmann and Hoffmann (1998)
Dibenz(a,h)anthracene	Sufficient (A)	4		Hoffmann and Hoffmann (1998)
Indeno(1,2,3-cd)pyrene	Sufficient (A)	4–20	51	Hoffmann and Hoffmann (1998), Hecht (2003)
5-methylchrysene	Sufficient (A)	ND–0.6		Hoffmann and Hoffmann (1998)
All PPAH in SS machine-smoked 1R4F Univ. of KY research cigarette	–	–	1,067	Gundel et al. (1995)
All PPAH in SHS plus exhaled MS human-smoked Camel, Merit, Winston, Benson & Hedges cigarettes	–	–	13,500	Rogge, Hildemann, Mazurek et al. (1994)
All PPAH SHS plus exhaled MS human-smoked Marlboro Lite 100s (0.7 g smoked)	–	–	13,260	Repace (abstract, 2004)

[a] Measured by EcoChem PAS 2000CE monitor.
[b] Wynder and Hoffmann (1967).
[c] Nanograms per cigarette; blank cells indicate no data available; IARC = International Agency for Research on Cancer.

predominantly found in the particle phase, and 4-ring compounds exist in both phases. Some PAH are potent locally acting carcinogens in laboratory animals, inducing cancers of the upper respiratory tract when inhaled. Excess cancers in workers are caused by PAH from coke-oven emissions in iron and steel foundries, and PAH are regulated as Hazardous Air Pollutants under Section 112 of the U.S. Clean Air Act. PPAH are particle-bound PAH, which generally consist of 4 or more ring PAH compounds. Table 9.3 shows 10 4+-ring PPAH that have been quantified in MS, SS, and in SHS from human smokers and that are known animal or human carcinogens. Several of the individual PPAH compounds listed in Table 9.3 have been measured in indoor atmospheres at levels ranging from 0.3–2 ng/m^3 (IARC 2004; Hecht 2003; EcoChem 2005).

9.11 APPLICATIONS — SHS IN NATURALLY AND MECHANICALLY VENTILATED BUILDINGS

Time-activity patterns show that the population spends about 88% of its time at home and at work. What are typical SHS concentrations in homes and workplaces? Can time-averaged models help us to understand those levels?

9.11.1 RSP FROM SHS IN HOMES

In the early 1980s, the Harvard 6-City Study (Dockery and Spengler 1981), collected RSP in 55 homes in six U.S. cities; they found that in the average home, the annual mean RSP increased overall by 0.88 $\mu g/m^3$ per cigarette, and in fully air-conditioned homes (which presumably have lower air exchange rates), by 2.11 $\mu g/m^3$ per cigarette. Leaderer et al. (1990) and Leaderer and Hammond (1991), in the New York State Energy Research and Development Authority study in the winter of 1986, measured RSP and nicotine in 96 New York State homes with detectable nicotine concentrations, whose mean air exchange rates a for all homes averaged 0.54 h^{-1}, and whose mean volumes v averaged 353 m^3. A mean daily cigarette usage of 14.2 cigarettes was reported per home, and 24-hour, 7-day averages for SHS-RSP and SHS-nicotine were 29 $\mu g/m^3$ (SD = 25.9 $\mu g/m^3$) and 2.2 $\mu g/m^3$ (SD = 2.43 $\mu g/m^3$), respectively. Wallace (1996) observed that in comparison of the three large-scale RSP in-home studies (Harvard 6-City, New York State, and Riverside PTEAM) that the estimates of smoking contributions to indoor fine particle concentrations in homes with smokers was 25–47 $\mu g/m^3$. Wilson et al. (1996) has collected a large database of air exchange rates in California homes, which show median air exchange rates in the range between 0.5 h^{-1} and 1 h^{-1}, depending upon climate and season. Models incorporating measured values for home volume, air exchange rate, and cigarette source strength predict measured data reasonably well (Repace, Ott, Klepeis, and Wallace 2000).

9.11.2 PREDICTING RSP FROM SHS IN MECHANICALLY VENTILATED BUILDINGS

For mechanically ventilated premises, since 1973, ASHRAE Standard 62, Ventilation for Acceptable Indoor Air Quality, has prescribed ventilation rates based primarily upon controlling carbon dioxide levels from human metabolism. ASHRAE Standard 62-1999 specified that maintaining a ventilation rate of 7.5 L/s per person (15 cfm/P) will yield a steady-state CO_2 concentration of 700 parts per million (ppm) above the outdoor background level. These "design rates" are then based on human occupancy, with default values for occupants per unit occupiable floor area given based on the maximum expected occupancy. Per-occupant design mechanical ventilation rates for commercial buildings based on default occupancy and building type have been prescribed by ASHRAE since 1973. This approach makes it possible to estimate air exchange rates for offices, restaurants, bars, etc. based on the design ventilation rates and default occupancy in the various versions of the standard issued in 1973, 1975, 1981, 1989, 1999, and 2000–2004 when these standards were annually updated. If the smoking prevalence for the historical period obtained from the Centers for Disease Control is assumed, applying the same using a default ceiling height, the smoker density can similarly be estimated. These parameters can then be input into the HSM model to estimate the RSP, PPAH, nicotine, or carbon monoxide concentrations from SHS, or into the active smoking model (ASM) model to estimate particle loss rates ϕ based on measurements of SHS concentration, ceiling height, and smoker density. (Estimating air exchange rates a to compare with the ASHRAE standard requires a further estimate of the deposition rate k.)

9.11.3 SHS IN THE HOSPITALITY INDUSTRY

Now we turn to a topic that has generated more heat than light: control of SHS in the hospitality industry. While occupational and public health authorities have recommended that exposure to SHS

be eliminated, bans on smoking in the hospitality industry have met with determined resistance, particularly from owners of bars and casinos, who have aggressively promoted ventilation alternatives to smoking bans. To appreciate the level of control required, one can ask, What level of SHS should be expected in a bar? The design ventilation standard, ASHRAE Standard 62-2001 recommends a ventilation rate of 15 L/second per occupant at a design maximum occupancy of 100 persons per thousand square feet of occupiable floor space, unchanged from ASHRAE Standard 62-1989, but 40% lower than the 25 L/second–occupant of ASHRAE Standard 62-1981. [Note that ASHRAE Standard 62-2004 only recommends ventilation rates for nonsmoking premises]. By contrast, U.S. smoking prevalence was 33% in 1981; by 2001, the median adult current smoking prevalence was 23.4% (range: 13.3–30.9%) for the 50 U.S. states and DC (MMWR 2003), a decline of 29%. As Equation 9.2 and Equation 9.4 show, unless the cigarette RSP emission rate has changed dramatically, or bars are making widespread use of unusually effective ventilation or filtration, the SHS concentration in the hospitality industry might be about the same in 2002 as it was 20 years earlier. The following study investigates this issue.

9.11.4 The Delaware Air Quality Survey

This study investigated air pollution in the hospitality industry before and after a smoking ban (Repace 2004a). This real-time study of indoor, outdoor, and in-transit air quality measured RSP and PPAH in the Wilmington, DE metropolitan area in 2002–2003. The Wilmington Study illustrates the collection and analysis of field data measured to assess the impact of secondhand smoke on human exposures in the hospitality industry, as well as the use of the mass-balance model to analyze and generalize such data (Repace 2004a). Using concealed real-time data-logging monitoring equipment (the MIE 1200AN nephelometer and the EcoChem PAS 2000CE photoelectric aerosol sampler), coupled with a time-activity pattern diary, I assessed air quality outdoors, in transit, and in eight hospitality venues (a casino, six bars, and a pool hall) on two Friday evenings: first on November 15, 2002, under conditions of unrestricted smoking (Figure 9.9, top) and then on January 24, 2003, 2 months after a statewide smoke-free workplace law (Figure 9.9, bottom). The mean indoor concentration of RSP dropped from 230 μg/m^3 to 24.5 μg/m^3 and indoor PPAH fell from 134 ng/m^3 to 4.6 ng/m^3. After subtracting the measured outdoor levels, the reduction due to the smoking ban was 92% for RSP and 85% for PPAH. This cross-sectional study yielded similar results to that observed in 52 visits to a tavern in the longitudinal study of Ott, Switzer, and Robinson (1996), who found a 90% reduction in measured RSP after a smoking ban. The correlation of RSP and PPAH, adjusted by subtracting outdoor levels, during smoking in this study was $R^2 = 0.55$ (Figure 9.10).

How do the measured RSP concentrations averaged over the six bars compare with the value predicted for a bar using the defaults in the HSM model? Assuming a default ceiling height of 10 ft, a bar at maximum occupancy would have 100 persons per 10,000 ft^3 (283 m^3) of space volume. In 2002, the State of Delaware had a smoking prevalence of 23% (MMWR 2003). Thus, for a bar with a Delaware average smoking prevalence, at maximum occupancy, there would be an expected 23 smokers per 283 m^3, for an expected habitual smoker density D_{hs} = 8 habitual smokers per hundred cubic meters. The design air exchange rate is calculated from the ASHRAE Standard as: a = (100 occupants)(15 L/s-occ)(3600 s/h)(1m^3/1000L)/283 m^3 = 19 h^{-1}. Because ventilation rates are not regulated, operational rates may be much lower than the design values. Applying the HSM yields: SHS = 280 $D_{hs}/(a + k)$ = (280)(8)/(19 + k). Assuming k is small compared to a, we estimate an SHS concentration of 118 μg/m^3 in a bar ventilated according to ASHRAE design criteria. Assuming a non-SHS RSP background of 10 μg/m^3, (about the $PM_{2.5}$ average for all U.S. counties outdoors in 2002) the predicted total RSP concentration in an ASHRAE Standard–ventilated bar at maximum occupancy and Delaware average smoking prevalence would be about 128 μg/m^3. For the six bars measured in the Delaware Study, the measured mean concentrations averaged 109 μg/m^3 (SD = 83 μg/m^3) indoors before the smoking ban and 11 μg/m^3 (SD = 9 μg/m^3) post-ban.

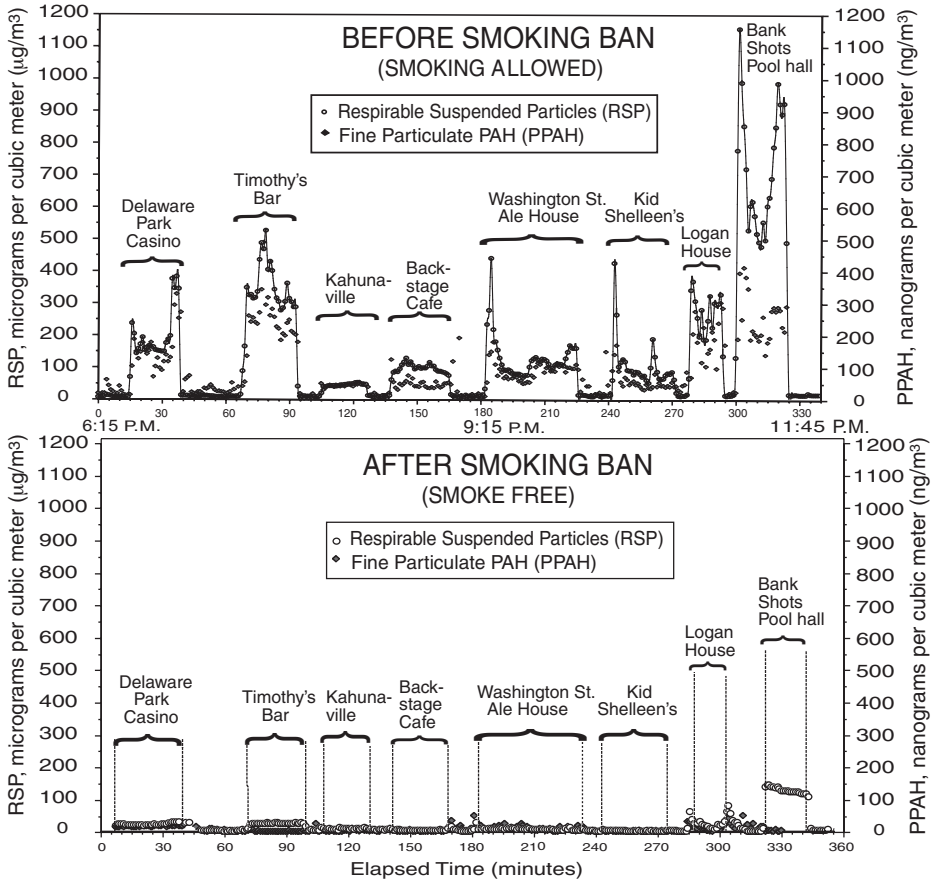

FIGURE 9.9 A real-time study of RSP and PPAH air pollution in a casino, six bars, and a pool hall before and after a smoking ban. An MIE pDR 1200 AN active-mode nephelometer for $PM_{3.5}$ and the EcoChem PAS 2000CE monitor for PPAH, assessed air quality in the 8 hospitality venues indoors, outdoors and in-transit. Top: Before the ban, on November 15, 2002, under conditions of unrestricted smoking. Bottom: This study was repeated on January 24, 2003, 2 months after a statewide smoke-free workplace law. (From Repace 2004a. With permission).

Using my rule-of-thumb default ϕ = 1.3a yields SHS = 98 µg/m³, which when added to the 11 µg/m³ background, yields 109 µg/m³.

9.12 APPLICATIONS — RSP AND CO FROM SHS IN A VEHICLE

Ott, Langan, and Switzer (1992) measured RSP from SHS in a 1986 Mazda 626 4-door sedan at 20 miles per hour with the windows closed and in which 3 Marlboro filter cigarettes had been smoked by a smoker at a rate of ~3 cigarettes in 40 minutes, or 4.5 cigarettes per hour. The car's air exchange rate was measured at a = 7.27 h^{-1}, and its volume was v = 3.7 m³. RSP deposition rates were not measured. These investigators found amazingly high peak RSP levels of 3000 µg/m³ and valleys of about 1000 µg/m³ between cigarettes. Ott, Langan, and Switzer (1992) also reported peak carbon ~~dioxide~~ (CO) levels at 12 ppm with valleys between cigarettes at 6 ppm compared to a measured roadway CO background of 1.5 ppm. Also applying the sequential cigarette exposure model (SCEM) to this experiment, the authors calculated an emission rate of 88 mg CO/cigarette. To place the measured concentration in perspective, the CO level in this car is higher than that observed by Otsuka et al. (2001), who showed that exposure to secondhand smoke carbon monoxide (SHS-CO) at levels of 6.02 ppm for 30 minutes induces acute endothelial dysfunction of the

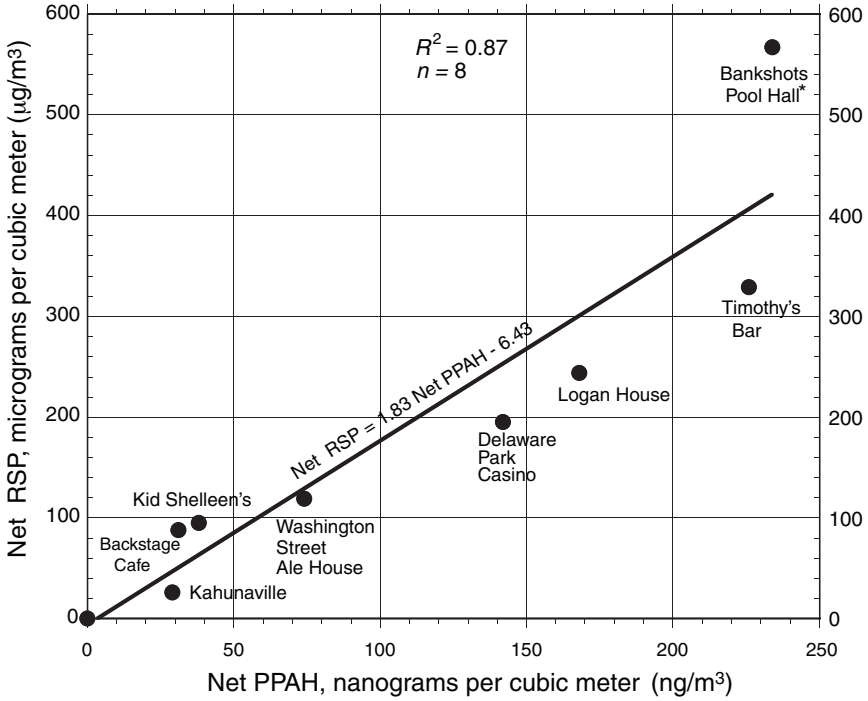

FIGURE 9.10 Net RSP and PPAH (indoor–outdoor levels) for eight Wilmington, DE locations (Figure 9.9, top) are correlated during smoking. Net PPAH is about 0.05% of net RSP. *(Data calculated from pre-ban RSP and post-ban indoor RSP [Figure 9.9, bottom] to account for an apparent non-SHS indoor source.) (From Repace 2004a, with permission.)

coronary circulation in nonsmokers. Thus, these observed SHS-CO levels are physiologically significant. Mulcahy and Repace (2002) reported 6.36 ppm median SHS-CO levels in 14 Galway, Ireland pubs. Recently, the Centers for Disease Control cautioned persons at risk of cardiovascular disease to avoid exposure to SHS (Pechacek and Babb 2004).

What do the peaks and valleys generated by CO emissions of multiple smoldered cigarettes look like? Figure 9.11 illustrates the CO emissions measured in the same Silicon Valley experiment of Figure 9.8. Using the SCEM model of Ott, Langan, and Switzer (1992), these data fit a SHS CO emission rate of 6.5 mg/min, or 65 mg CO/cigarette.

9.13 APPLICATIONS — DOSIMETRY: TRANSLATION OF EXPOSURE INTO DOSE VIA COTININE ANALYSIS

The concentration of one major SHS atmospheric marker can be estimated from the concentration of another (e.g., nicotine, RSP, and CO), and therefore are all relatable to the major body-fluid biomarker, cotinine, because it is derived from nicotine (Repace and Lowrey 1993; Repace et al. 1998; Repace, Al-Delaimy, and Bernert 2006). This allows a greater understanding of how the dose distribution in Figure 9.3 comes about. The reader is referred to the excellent article by Benowitz (1999) for a detailed discussion of nicotine and cotinine pharmacokinetics. Because cotinine has a 19-hour half-life, Figure 9.3 essentially represents a daily dose "snapshot" of the cross-sectional SHS exposure for the U.S. population for the time period 1988–1991, and illustrates a two-order of magnitude spread in SHS exposure among the population. Can the models and data given in this chapter shed any light on this distribution? As part of a risk assessment of passive smoking–induced lung cancer, Repace and Lowrey (1985) modeled the average SHS-RSP exposure of

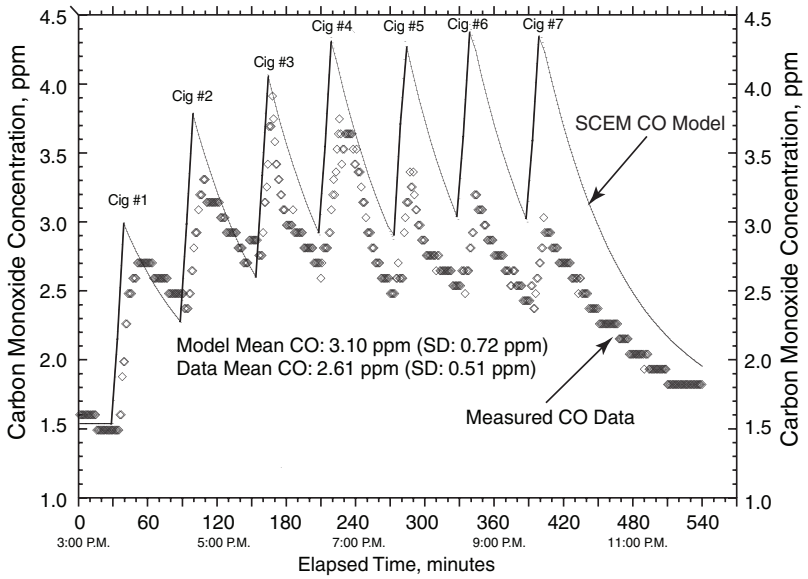

FIGURE 9.11 The simultaneous CO emissions for the seven smoldered cigarettes experiment shown in Figure 9.8. Using the SCEM model of Ott, Langan, and Switzer (1992), these data fit a SHS CO emission rate of 6.5 mg/min, or 65 mg CO/cigarette (Data from Ott and Repace 2003). The room volume is 41 m^3 and the air exchange rate is 0.82 h^{-1}.

a typical person in the U.S. population using the HSM, incorporating time-activity pattern studies, exposure probabilities for home and workplace exposure derived from surveys, coupled with respiration rates for home and work activities, using the default $\phi = 1.3a$. Using these methods, we assumed that the typical person was exposed only at home or at work, which covered 88% of a person's time. Based on the survey-grounded assumption that 86% of the U.S. population was exposed to SHS either at work or at home, or in both microenvironments, Repace and Lowrey (1985, 1993) estimated that the average American circa 1985 inhaled 1430 micrograms of SHS-RSP daily. Repace and Lowrey (1993), assuming a 10:1 ratio for RSP to nicotine in SHS, translated the modeled SHS-RSP inhaled exposure for this hypothetical person into an estimated daily nicotine exposure of 143 micrograms daily, and by developing a pharmacokinetic model translating inhaled nicotine into its biomarker, cotinine, estimated that the typical U.S. adult nonsmoker was exposed to 1 nanogram of cotinine per milliliter (ng/ml), for the most-exposed persons, 10 ng/ml. When updated with an improved 78% nicotine-to-cotinine conversion efficiency (Benowitz 1999; Repace et al. 1998), these values are reduced by a factor of 0.86, to 0.86 ng/ml and 8.6 ng/ml, respectively.

The NHANES III national cotinine dosimetry survey shown in Figure 9.3 later found an 88% SHS exposure probability. When compared to the NHANES III "reported home or work ETS exposure" distribution of Figure 9.3, the cotinine doses estimated by Repace and Lowrey (1993) yield estimates strikingly consistent with both the median and upper extreme values observed for those exposed at work or at home for the U.S. population in 1989–1991 (Pirkle et al. 1996; Repace, Al-Delaimy, and Bernert 2006).

What exposure scenarios might lead some unfortunate nonsmokers to be at the upper end of the SHS dose distribution of Figure 9.3 (dark gray)? A serum cotinine value of 8 ng/ml corresponds to saliva cotinine value of about 9.3 ng/ml (Repace et al. 1998). In the early 1990s, London pub workers had measured *median* salivary cotinine levels of 8 ng/ml (Jarvis, Foulds, and Feyerabend 1992). By 2000 a sample ($n = 44$) of London bar-worker salivary cotinines manifested mean values of 6.16 ng/ml, seven times higher than the mean values for a sample of all English households of 0.86 ng/ml (Jarvis 2001). This suggests that bar staff may populate the upper dose levels of the

cotinine distribution. What daily average SHS air pollution concentration does a serum cotinine value at the upper extreme represent? The Repace et al. (1998) model equates a saliva cotinine concentration of 9.36 ng/ml to an 8-hour time-weighted average (TWA) SHS nicotine exposure concentration of 109 $\mu g/m^3$, which in turn equates to an estimated 8-hour TWA SHS-RSP concentration of 1090 $\mu g/m^3$ (Repace and Lowrey 1993). Levels this high are consistent with the most extreme SHS values encountered in workplace exposures in the hospitality industry shown in Figure 9.4 and Figure 9.9.

Application of such models permits forensic risk assessment. For example, Repace (2004b) analyzed a cotinine dosimetry study of flight attendants conducted by the National Cancer Institute (Mattson et al. 1989) on several Air Canada flights in 1989, and estimated that the median serum cotinine dose of typical flight attendants in aircraft cabins, at 2.88 ng/ml, was about 6-fold that of the average U.S. worker and about 14-fold that of the average person at that time. As there are in excess of 3,000 flight attendant SHS personal-injury lawsuits currently in litigation, such estimates are indispensable for assisting the court in placing such exposures in perspective. If a flight attendant develops a disease associated with SHS, exposure or dose models permit incomparably better evaluation of risk probabilities than is possible simply by saying Ms. X worked as a flight attendant and was exposed to SHS in aircraft cabins for 20 hours per week. Application of such models also permits improvements in exposure assessment in passive smoking epidemiology, which has been based almost wholly on spousal smoking status, which ignores concentration, duration, and respiration rates, and relates only tangentially to activity patterns, and thus is a weak surrogate for exposure (Repace, Al-Delaimy, and Bernert 2006).

9.14 FUTURE ISSUES

Secondhand smoke pollution will continue to be a major public health problem for many people throughout much of the world for many decades. To place this into perspective, in 1986, 50 million U.S. cigarette smokers smoked 584 billion cigarettes, burning an estimated 424,330 metric tons of tobacco in indoor microenvironments, with another estimated 29,700 metric tons burned by pipe and cigar smokers (Repace and Lowrey 1990). Fourteen years later, in 2000, the number of cigarette smokers had declined only by 7% to 46.5 million. Table 9.4 gives the number of U.S. smokers, cigarettes consumed by them, and estimated smoking rates for various years from 1965–2000. The number of smokers has been slowly declining since its peak in 1983. Both the number of cigarettes consumed and the cigarette smoking rate peaked in 1979 and have also slowly declined since. The decline in average cigarette smoking rate may actually reflect fewer opportunities to smoke because of the increased prevalence of workplace and home smoking bans rather than a decline in personal smoking rate when an individual is at liberty to smoke freely; in turn, the reduced smoking prevalence may facilitate the growth of home and workplace smoking bans. However 18 state legislatures currently preclude total workplace smoking bans (American Nonsmokers Rights 2005), and this decline may occur largely outside those states.

Half of all U.S. children still grow up in homes with smokers. In 2003, 94% of locations measured in seven countries in Latin America were observed to have detectable levels of SHS nicotine (Navas-Acien et al. 2004). The California Environmental Protection Agency (CalEPA 2005) estimates that indoor air pollution from SHS in California — even after banning all workplace smoking — still causes more than $25 billion in associated healthcare costs. More research is needed in several areas. Smoking in multifamily dwellings continues to be a significant problem due to infiltration of tobacco smoke from one unit to another; little research has been done on the concentrations and mitigation, but this is a major complaint expressed to local health departments and has been the subject of litigation.

Outgassing of deposited SHS tars is another major concern of nonsmokers moving into homes or apartments formerly occupied by heavy smokers, as is re-emission from surfaces in elementary school multipurpose rooms time-shared with bingo games. Johansson, Olander, and Johansson

TABLE 9.4
Estimated Number of U.S. Smokers, Cigarettes Smoked and Smoking Rates, 1965–2000, for 8 Available Years

Year	1965	1970	1974	1979	1983	1988	1995	2000
Millions of U.S. smokers[a]	50.1	48.1	48.9	51.1	53.5	49.4	47.0	46.5
Billions of cigarettes consumed[b]	521.1	534.2	594.5	621.8	603.6	560.7	482.2	430
Cigarettes smoked per smoker per day[c]	28.5	30.4	33.3	33.3	30.9	31.0	28.1	25.3

[a] CDC TIPS: Number (in millions) of adults 18 years and older who are current, former, or never smokers, overall and by sex, race, Hispanic origin, age, and education, National Health Interview Surveys, selected years — United States, 1965–2000.

[b] Federal Trade Commission sales by manufacturers to wholesalers and retailers within the U.S. and Armed forces personnel stationed outside the U.S. National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention.

[c] Leap-year adjusted.

(1993) reported that the surfaces in a room where cigarette smoking occurs become secondary particulate-phase pollution sources. Singer et al. (2004) found that lower volatility gas-phase SHS compounds (nicotine, 3-ethenylpyridine, phenols, cresols, naphthalene, and methylnapthalenes) outgassed from surfaces where they had sorbed, suggesting widespread contamination of buildings and air-handling systems with deposited SHS. Such outgassing contributes to indirect exposure to SHS contaminants even when smokers are not present or not smoking in microenvironments with daily smoking, particularly where infants are concerned. Further research is needed into the range of deposition and re-emission rates, and persistence of SHS tars on room surfaces. Smoking in hotels exposes guests in lobbies and maids cleaning smokers' rooms to unknown levels of outgassed SHS, which as Table 9.1 shows, contains a wide variety of regulated carcinogens. Smoking in outdoor cafes also continues to expose waitstaff to SHS; preliminary research into this problem shows that outdoor levels of SHS-RSP and SHS-PPAH in the proximity of smokers may be as high as indoor microenvironments (Klepeis, Ott, and Switzer 2004; Repace 2004 abstract). SHS in prisons and psychiatric institutions has been little studied. Additional studies of cotinine dose in specific worker groups, such as bar and casino workers, and its relationship to SHS exposure and ventilation and air cleaning would be useful to evaluate claims of efficacy for these putative SHS control measures relative to smoke-free workplaces. Finally, there will be an expected 1 billion tobacco deaths worldwide in the 21st century if current global smoking patterns continue — a tenfold increase of the 20th century toll of 100 million (CRC 2002; WHO 2005). Based on the ratio of estimated U.S. passive smoking to active smoking deaths (Surgeon General 2004; CalEPA 2005), one out of eight of those deaths will be nonsmokers exposed to SHS.

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9.16 QUESTIONS FOR REVIEW

1. A bitter epidemiological dispute developed when Enstrom and Kabat (2003), funded by the tobacco industry, published a re-analysis and 40-year follow-up of the American Cancer Society's (ACS) Cancer Prevention Study I cohort that concluded that "the results

do not support a causal relation between environmental tobacco smoke (ETS) and tobacco-related mortality." Thun (2003) of ACS responded that Enstrom and Kabat's (2003) analysis was "misleading science" and was fundamentally flawed in major part because of exposure misclassification of CPS I subjects. Enstrom and Kabat replied that in this cohort, in 1959, "the majority of female never smokers married to never smokers were not exposed to ETS." The U.S. adult smoking prevalence in 1965 was 42.4%, 37% in 1974, and by 1990 had declined to 25.5%. If Figure 9.3 represents the exposure distribution of the nonsmoking U.S. population in 1990, who is more likely to be correct? Why?

2. Air quality control authorities recommend that citizens remain indoors during outdoor air pollution alerts. Is this likely to be good advice for nonsmokers who live with smokers?
3. It is clear that smoke-free workplace laws can reduce indoor air pollution in casinos, bars, and restaurants virtually to outdoor levels. Opponents of such laws argue that ventilation can produce acceptable indoor air quality. In Figure 9.9, the SHS-RSP level in Timothy's Bar averages 337 $\mu g/m^3$ above the 10 $\mu g/m^3$ outdoor background concentration at an active smoker density of 1.44 burning cigarettes per 100 m^3. Holding the smoker density and outdoor background concentration constant, how many air changes per hour would it take to reduce the total indoor RSP to 15 $\mu g/m^3$, the level of the Annual National Ambient Air Quality Standard which defines clean air for RSP? Is this value possible to attain at the 18 h^{-1} air exchange rate recommended by the ASHRAE Standard 62-2001? The bar's existing particle removal rate was calculated using the ASM model to be $\phi = 0.87\ h^{-1}$. The actual indoor RSP level in the bar after the ban was 24 $\mu g/m^3$. How much ventilation would it take to attain the NAAQS in this case? [Answer: In the case of a 10 $\mu g/m^3$ background particle concentration, one would need to attain a level of (15–10) = 5 $\mu g/m^3$ of SHS-RSP, which results in the equation (337–10) $(0.87/\phi) = 5$ $\mu g/m^3$, and solving for ϕ gives a particle decay rate of $\phi = a + k = 75.2\ h^{-1}$. Assuming the particle deposition rate k is small compared with the air change rate a, the ventilation rate would need to be about 4 times the level recommended by ASHRAE. In the case of a 24 $\mu g/m^3$ background level, attainment of the NAAQS would be impossible.]
4. One of the most common complaints for local health departments is infiltration of SHS from a neighboring apartment. If a nonsmoker resides in a mid-level apartment in a three or more story building, how many apartments might surround the nonsmoker? What is the probability of having at least one smoker in a neighboring apartment if the smoking prevalence in the building is 25%? About one half to two thirds of the air in a multifamily dwelling appears to infiltrate from neighboring apartments in older buildings. What methods can you think of to minimize or eliminate this infiltration? [Answer 1: The nonsmoker's apartment will typically be surrounded by four neighboring apartments. If two people reside in each neighboring apartment, of the eight neighbors, chances are that at least two smoke. Therefore, one to two neighboring apartments will have a smoker. Answer 2: To minimize infiltration, plugging cracks behind electrical outlets and around plumbing pipes, plus positively pressurizing the nonsmoker's apartment or depressurizing the wall between apartments are possible engineering controls. Smoking and nonsmoking buildings may be possible future policies.]
5. Numerous flight attendants have filed lawsuits against the tobacco industry and against individual airlines over their exposure to SHS. An important question arising in such litigation is what are SHS exposures like on a typical aircraft? Assume that a Boeing 747 flying from the U.S. to Europe has a cabin volume $v = 790\ m^3$ and an air exchange rate $a = 14.7\ h^{-1}$. Assume a complement of 288 passengers, with an average smoking prevalence of 13.7%, and a smoking rate of 1.5 cigarettes per hour. Using the mass-balance model, estimate the well-mixed SHS-RSP level. Compare your result with the average of four measurements taken on eight international flights sampling four locations

on the aircraft: (133 + 36 + 21 + 11)/4 = 50 μg/m^3, by Nagda et al. 1989). Assume that flight attendants moving about the aircraft will approximate this average. [Answer: Using the HSM of Equation 9.3, the habitual smoker density is D_{hs} = 100({1.5 cigarettes/h}/{2 cigarettes/h})(0.137 smoking prevalence)(288 passengers) / (790 m^3) = 3.74 *HS*/100 m^3. Assume *k* small compared to *a*. Thus, Equation 9.3 becomes: ETS-RSP = 280(3.74/14.7) = 71 μg/m^3. With the default of $\phi = 1.3a$, ETS-RSP = 55 μg/m^3.]

6. Table 9.1 shows a list of 172 toxic compounds in SHS for which SHS atmospheric markers are surrogates. Fowles and Dybing (2004) used toxicological methods, which in principle provide a plausible and objective framework for estimating cancer risk, applied to a list of toxic substances in tobacco smoke such as those in Table 9.1. They found that this method underestimates observed cancer risk in smokers by a factor of 5. Can you think of a reason why? [Answer: This may be due to synergy between chemicals. Other reasons include omission of some chemicals; unknown toxicity of many chemicals; inadequacy of risk assessment methods; difficulty in extrapolating from animals to humans and from low dose to high dose, etc.]
7. Average house volumes in the U.S. were about 350 m^3 in the late 1990s. For an air exchange rate of 0.6 h^{-1}, a $PM_{2.5}$ deposition rate of 0.4 h^{-1}, and 1 smoker spending 8 waking hours in the home smoking 2 cigarettes per hour with a $PM_{2.5}$ emission rate of 14 mg/cigarette, calculate the 24-hour average $PM_{2.5}$ concentration. How does your answer compare with the observed range in three large studies of 25 to 45 μg/m^3? [Answer: 26.7 μg/m^3.]

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