TABLE 2.—Measurement of constituents of tobacco smoke in experimenta

Reference, location, and dimensions	Ventilation	Amount of tobacco burned	Level constit
DeRouane and Verduyn (*27*).			
House 50 m³	closed	3 cig in 34 min	7.5 pp
Dublin (*28*).			
Conference room 138 m³	12.0 air changes per hour	2 cig	32.5 p
Harke (*36*).			
Room 57 m³	none	42 cig in 18 min	50 pp .560 n
	7.2 air changes per hour	42 cig in 18 min	10 pp .12 m
	8.4 air changes per hour	42 cig in 18 min	< 10 < .1
	none	9 cigars in 35 min	60 pp 1.04 n
	7.2 air changes per hour	9 cigars in 35 min	20 pp .42 m

TABLE 2.—Measurement of constituents of tobacco smoke in experimental conditions.[1]—continued

Reference, location, and dimensions	Ventilation	Amount of tobacco burned	Level of constituent	Measure of absorption
Harke (*36*).				
Room 57 m^3 (Cont.)	none	9 pipes in 40 min	10 ppm CO .52 mg/m^3 nicotine	
	7.2 air changes per hour	9 pipes in 40 min	< 10 ppm CO < .1 mg/m^3 nicotine	
Room 170 m^3	none	105 cig	30 ppm CO	Smokers 7.5% COHb Nonsmokers 2.1% COHb
	1.2 air changes per hour	107 cig	5 ppm CO	Smokers 5.8% COHb Nonsmokers 1.3% COHb
	2.3 air changes per hour	101 cig	75 ppm CO	Smokers 5.0% COHb Nonsmokers 1.6% COHb
Harke, et al. (*38*).				
Room 38.2 m^3	none	30 cig	.51 mg/m^3 nicotine .65 mg/m^3 acetaldehyde .46 mg/m^3 acrolein	
	none	15 cig	.27 mg/m^3 nicotine .29 mg/m^3 acetaldehyde .23 mg/m^3 acrolein	
	none	10 cig	.13 mg/m^3 nicotine .19 mg/m^3 acetaldehyde .16 mg/m^3 acrolein	

TABLE 2.—Measurement of constituents of tobacco smoke in experimenta

Reference, location, and dimensions	Ventilation	Amount of tobacco burned	Level constit
Harke, et al. (*38*).			
Room 38.2 m³ (Cont.)	none	5 cig	.06 mg
			.13 mg
			.07 mg
Room 170 m³	none	150 cig	58 ppr
		by machine	.72 mg
		in 34 min	.53 mg
			.39 mg
	none	102 cig	28 ppr
		by machine	.18 mg
		in 2 hr	.10 mg
			.09 mg
	2.4 air changes per hour	102 cig	8 ppm
		by machine	.10 mg
		in 2 hr	.5 mg
			.04 mg
	none	108 cig	24.5 p
		by 11 smokers	.14 mg
		in 2 hr	1.0 mg
			.06 mg

TABLE 2.—Measurement of constituents of tobacco smoke in experimental conditions.[1]—continued

Reference, location, and dimensions	Ventilation	Amount of tobacco burned	Level of constituent	Measure of absorption
Harke, et al. (*40*).				
Mid-size European car, engine off, in wind tunnel at 50 km/hr wind speed	none	9 cig	30 ppm CO	
	air jets open and blower off	6 cig	20 ppm CO	
	air jets open and blower on	6 cig	10 ppm CO	
Mid-size European car, engine off, in wind tunnel at zero km/hr wind speed	none	9 cig	110 ppm CO	
	none	6 cig	80 ppm CO	
	air jets open and blower on	6 cig	8-10 ppm CO	
Harmsen and Effenberger (*43*).				
Room 98 m^3	none	62 cig in 2 hr	80 ppm CO, 5,200 $\mu g/m^3$ nicotine	
Hoegg (*45,46*).				
Sealed test chamber 25 m^3	none	4 cig	12.2 ppm CO, 2.28 mg/m^3 TPM	
		8 cig	25.6 ppm CO, 5.39 mg/m^3 TPM	
		16 cig	47.0 ppm CO, 11.41 mg/m^3 TPM	
		24 cig	69.8 ppm CO, 16/65 mg/m^3 TPM	

TABLE 2.—Measurement of constituents of tobacco smoke in experimenta

Reference, location, and dimensions	Ventilation	Amount of tobacco burned	Level constit
Jermini, et al. (*47*).			
Box 30 m³	none	3 cig by machine	.13 pp
			.22 pp
			.011 p
			.041 p
			.013 p
			.023 p
			.45 pp
			.24 pp
			.015 p
			.10 pp
			.17 pp
			.52 pp
			.067 p
			.008 p
			.020 p
			.032 p
			.38 pp
			.10 pp
			.006 p
			.043 p
Lawther and Commins (*52*).			
Room 15 m³	1 air change per hour	7 cig	20 pp
			3 mg/

TABLE 2.—Measurement of constituents of tobacco smoke in experimental conditions.[1]—continued

Reference, location, and dimensions	Ventilation	Amount of tobacco burned	Level of constituent	Measure of absorption
McNall (*57*).				
Home 425 m³	3 air changes per hour	12 cig in 1 hr	1.1 mg/m³ TPM	
	.5 air changes per hour	35 cig in 1 hr	2.7 mg/m³ TPM	
Russell, et al. (*65,66*).				
Room 43 m³	none	80 cig & 2 cigars per hr	38 ppm CO	Smokers 9.6% COHb, 1,236 ng/ml urinar nicotine Nonsmokers 2.6% COHb, 80 ng/ml urinary nicotine
Seppanen (*70*).				
Room 37.5 m³	none	126 cig by smokers in 1/5 hr	30 ppm CO	Smokers 9.1% COHb Nonsmokers 2.2% CO
Srch (*78*).				
Car, engine off, 2.09 m³	none	10 cig in 1 hr	90 ppm CO	Smokers 10% COHb Nonsmokers 5% COH

TABLE 2.—Measurement of constituents of tobacco smoke in experimental conditions.[1]—continued

Reference, location, and dimensions	Ventilation	Amount of tobacco burned	Level of constituent	Measure of absorption
Weber, et al. (*79,80,81,82*).				
Box 30 m^3	none	5 cig	12 ppm CO .19 ppm NO .02 ppm NO_2 .23 ppm CH_2O .05 ppm acrolein	
	none	10 cig	24 ppm CO .36 ppm NO .04 ppm NO_2 .46 ppm CH_2O .11 ppm acrolein	

[1]cig = cigarettes, — = unknown, TPM = total particulate matter.

difference in smoke exposure makes the quantification of the involuntary smoking exposure in terms of "cigarette equivalents" confusing and inaccurate. It requires that involuntary smoking be evaluated as a separate problem not subject to simple extrapolation of our understanding of dose-response relationships for cigarette smoking. A more comprehensive review of the chemistry of tobacco smoke is provided in the Chapter on Constituents of Tobacco Smoke in this report.

A number of investigators have attempted to measure the levels of some of the substances in cigarette smoke encountered in experimentally controlled (Table 2) and everyday (Table 3) situations. The type and amount of tobacco product burned, size of the room, amount and type of ventilation or filtration, duration of the smoking, as well as background atmospheric contamination, have all been shown to influence the measured concentrations and absorption by the nonsmoker. A number of substances have been the subject of particular investigative attention.

Carbon Monoxide

Carbon monoxide is one of the major combustion products of cigarettes; mainstream smoke contains 1.5 to 5.5 volumes percent of CO, with levels in sidestream smoke up to three times as high (see Chapter on Constituents of Tobacco Smoke). Carbon monoxide produced by cigarette smoking represents a minor part of the total atmospheric burden of CO but, as can be seen from Tables 2 and 3, it can contribute substantially to the levels found in enclosed spaces. The major determinants of the CO levels in these situations are size of the space in which the smoking occurs (dilution of CO), the number and type of tobacco products smoked (CO production), and the amount and effectiveness of ventilation.

The type of tobacco product smoked is important as a determinant of CO exposure because it has been found that mainstream smoke from regular and small cigars contains more CO per puff and per gram of tobacco burned than that from filter or nonfilter cigarettes (*15*). This greater production of CO by cigars was confirmed by Harke (*36*). He measured the CO produced by 42 cigarettes, 9 cigars, and 9 pipefuls of tobacco, each product evaluated separately but under the same room conditions. The cigars produced the highest CO level (60 ppm).

Carbon monoxide is a gas, does not settle out of the atmosphere in an enclosed space, and is not removed by most of the standard air filtration systems. As a result, the reduction of CO levels requires the replacement of contaminated air with uncontaminated air. Jones and Fagan (*51*) calculated the levels of CO that would result in a 3,000 cubic-foot room populated by 25 smokers when the ventilation was

TABLE 3.—Measurement of constituents of tobacco smoke under natural conditions.[1]

Reference, location, and dimensions	Ventilation	Amount of tobacco burned	Level of constituent	
			Smoking section	Other control section
Brunnemann and Hoffmann (*16*).			dimethylnitrosamine	
Train 1 (Bar Car)			.13 ng/l	
Train 2 (Bar Car)			.11 ng/l	
Bar			.24 ng/l	
Cano, et al. (*19*).				
Submarines 66 m^3	yes	157 cig per day	< 40 ppm CO, 32 ug/m^3 nicotine	
		94–103 cig per day	< 40 ppm CO, 15–35 ug/m^3 nicotine	
Chappel and Parker (*20*).				
General public places		—	3.5 ppm CO	2.0 ppm CO
Government offices	—	—	2.5 ppm CO	2.5 ppm CO
Restaurants	—	—	4.0 ppm CO	2.5 ppm CO
Night clubs and taverns	—	—	13.0 ppm CO	3.0 ppm CO

TABLE 3.—Measurement of constituents of tobacco smoke under natural conditions.[1]—continued

Reference, location, and dimensions	Ventilation	Amount of tobacco burned	Level of constituent	
			Smoking section	Other control section
Cuddeback, et al. (*24*).				
Tavern 1	6 air changes per hour	—	12.5 ppm CO .33 mg/m³ TPM	—
Tavern 2	none	—	17 ppm CO .98 mg/m³ TPM	—
Elliott and Rowe (*30*).				
Arenas	—	—	14.3 ppm CO .367 mg/m³ TPM	3 ppm CO .068 mg/m³ TPM
Galuskinova (*33*).				
Restaurant	—	—	.0002 - .0046 mg/m³ benzopyrene	
Godin, et al. (*35*).				
Ferry boat compartments			18.4 ± 8.7 ppm CO	3.0 ± 2.4 ppm CO
Theater			3.4 ± 0.8 ppm CO	1.4 ± 0.8 ppm CO

TABLE 3.—Measurement of constituents of tobacco smoke under natural conditions.[1]—continued

Reference, location, and dimensions	Ventilation	Amount of tobacco burned	Level of constituent	
			Smoking section	Other control section
Harke (*37*).				
Office Building	air conditioned	—	< 5 ppm CO	
Office Building	not air conditioned	--	< 5 ppm CO	
Room 78.3 m^3	...	3 smokers	15.6 ppm CO	
Harke and Peters (*41*).				
Automobile	35 km/hr speed, no ventilation.	4 cig	24.3 ppm CO	
	80 km/hr speed, no ventilation.	4 cig	12.1 ppm CO	
	30 km/hr speed, no ventilation.	4 cig	21.4 ppm CO	
	30 km/hr speed, air jets open.	4 cig	15.7 ppm CO	
	3 km/hr speed, air jets open & blower on.	4 cig	12.0 ppm CO	
Hinds and First (*44*).	—	--	nicotine:	
Commuter train			.0049 mg/m^3	
Commuter bus			.0063 mg/m^3	
Bus waiting room			.001 mg/m^3	
Airline waiting room			.0031 mg/m^3	
Restaurant			.0052 mg/m^3	
Cocktail lounge			.0103 mg/m^3	
Student lounge			.0028 mg/m^3	

TABLE 3.—Measurement of constituents of tobacco smoke under natural conditions.[1]–continued

Reference, location, and dimensions	Ventilation	Amount of tobacco burned	Level of constituent	
			Smoking section	Other control section
Lefcoe and Inculet (*55*).				
House	—	1 cig	48 x 10^6 particles per cubic foot	.9 x 10^6 particles per cubic foot
Szadkowski, et al. (*75*).				
Offices	—	—	2.7 ppm CO	
Sebben, et al. (*68*).				
Night clubs	—	—	13.4 ppm CO	9.2 ppm CO
Restaurants	—	—	8–28 ppm CO	—
Bus	—	—	7.3 ppm CO	6.2 ppm CO
Slavin and Hertz (*71*).				
Conference room	8 air changes per hour	—	8 ppm CO	1-2 ppm CO
	6 air changes per hour	—	10 ppm CO	1-2 ppm CO

TABLE 3.—Measurement of constituents of tobacco smoke under natural conditions.[1]—continued

Reference, location, and dimensions	Ventilation	Amount of tobacco burned	Level of constituent	
			Smoking section	Other control section
Seiff (*69*).				
Intercity bus	15 air changes per hour	23 cig burning continuously	33 ppm CO	
		3 cig burning continuously	18 ppm CO	
U.S. Dept. Transportation, et al. (*60*).				
Airplane flights:				
Overseas - 100% filled	15–20 air changes per hr		2–5 ppm CO, < .120 mg/m³ TPM	
Domestic - 66% filled			2 ppm CO, < .120 mg/m³ TPM	

[1]cig = cigarettes, — = unknown, TPM = total particulate matter.

varied (Figure 1). They assumed that the smokers would smoke four cigarettes per hour and that each cigarette would produce 74 mg of CO. They then repeated the same calculations for 25 nonsmokers and extrapolated that the room filled with smokers would require a rate of ventilation 10 times higher (1000 cu ft/min versus 100 cu ft/min) than the room with the nonsmokers in order to keep the CO concentration below the Ambient Air Quality Standards set by the Environmental Protection Agency (9 ppm CO) (*31*). These data generate some concern due to the current trend toward more tightly sealed buildings with recirculation and filtration of the air rather than the more energy-costly intake and warming or cooling of uncontaminated outside air. As air conditioning systems become more self-contained the problem of meeting the Ambient Air Quality Standards for CO may become more complex.

Examination of Table 2 reveals that under conditions of heavy smoking and minimal ventilation even the threshold limit value for an 8-hour industrial exposure to CO (50 ppm) (*1*) may be exceeded, but the addition of even modest amounts of ventilation results in a rapid drop in the CO levels. Harke (*40*) also showed that in small enclosed unventilated spaces (an automobile) the CO level is determined more by the number of cigarettes being smoked at one time than by the cumulative number of cigarettes that have been smoked and that the CO level decreases rapidly once the smoking stops.

The level of smoking in these experimental conditions was generally far heavier than is common in everyday situations. Indeed, when levels are measured in everyday situations (Table 3), they are found to be lower than those in the experimental situation. However, cigarette smoking can produce CO levels well above the Ambient Air Quality Standard (9 ppm) in these everyday stiuations.

One must be careful when using the levels recorded in Table 3 as measures of individual exposure because the CO levels were usually measured at points several feet from the nearest smoker. Individuals might be exposed to higher or lower levels depending on their distance from someone actively smoking (*28, 52*). In addition, it is the CO absorbed by the body that causes the harmful effects, not that which is measured in the atmosphere. This absorption can vary from individual to individual, depending on factors such as duration of exposure and cardio-respiratory status.

Several investigators have tried to determine the amount of carbon monoxide absorbed in involuntary smoking situations by measuring changes in carboxyhemoglobin levels in nonsmokers exposed to cigarette smoke-filled environments. Anderson and Dalhamn (*3*) found no change in the COHb levels of nonsmokers in a well-ventilated room where the CO level was 4.5 ppm. When Harke (*36*) studied nonsmokers under similar conditions (good ventilation and less than 5 ppm CO), he found an increase in COHb level from 1.1 to 1.6 percent; without

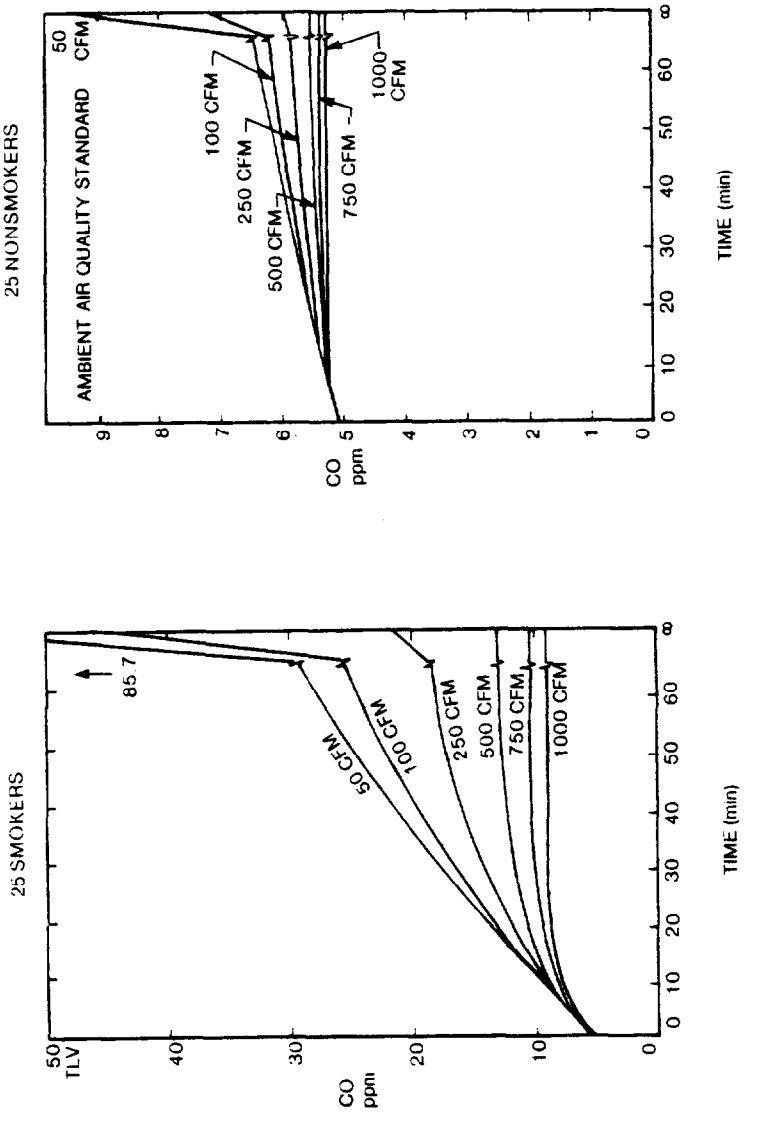

FIGURE 1.—Calculated buildup of CO under varying conditions of ventilation and smoking. Calculated for a room 3000 ft³ with 25 smokers on the left and for 25 nonsmokers on the right. TLV is the threshold limit value for CO (50 ppm). CFM is ventilation in cubic feet per minute.

SOURCE: Jones, R.N. (*51*).

TABLE 4.—Median percent carboxyhemoglobin (COHb) saturation and 90 percent range for nonsmokers by location.

Location	Nonsmokers		No. of nonsmokers	Percent of nonsmokers with COHb >1.5%
	Median	Range		
Anchorage	1.5	0.6–3.2	152	56
Chicago	1.7	1.0–3.2	401	74
Denver	2.0	0.9–3.7	744	76
Detroit	1.6	0.7–2.7	1,172	42
Honolulu	1.4	0.7–2.5	503	39
Houston	1.2	0.6–3.5	240	30
Los Angeles	1.8	1.0–3.0	2,886	76
Miami	1.2	0.4–3.0	398	33
Milwaukee	1.2	0.5–2.5	2,720	26
New Orleans	1.6	1.0–3.0	159	59
New York	1.2	0.6–2.5	2,291	35
Phoenix	1.2	0.5–2.5	147	24
St. Louis	1.4	0.9–2.1	671	35
Salt Lake City	1.2	0.6–2.5	544	27
San Francisco	1.5	0.8–2.7	660	61
Seattle	1.5	0.8–2.7	535	55
Vermont, New Hampshire	1.2	0.8–2.1	959	18
Washington, D.C.	1.2	0.6–2.5	850	35

SOURCE: Stewart, R.D. (74).

ventilation the CO levels rose to 30 ppm and the COHb level increased from .9 to 2.1 percent in 2 hours. Russell, et al. (*65*) found that COHb levels increased from 1.6 to 2.6 percent in nonsmokers present in a smoke-polluted room where the CO level was measured at 38 ppm; however, he cautioned that nearly all persons in the room felt that the conditions were worse than those experienced in most social situations.

Aronow (*4*) exposed 10 patients with coronary artery disease to the smoke from 15 cigarettes smoked by 3 volunteers over 2 hours in a 30.8 m^3 room. He reported that the COHb levels increased in the nonsmokers from a baseline of 1.26 percent to 1.77 percent when the room was ventilated at 11.4 air changes per hour and from 1.30 percent to 2.28 percent when the ventilation was turned off.

Stewart, et al. (*74*) measured COHb levels in a group of nonsmoking blood donors from several cities and found that 45 percent exceeded the Clean Air Act's Quality Standard of 1.5 percent, with the 90 percent range as high as 3.7 percent for individual cities (Table 4).

These levels represent the total body burden of CO for the nonsmoker due to endogenous production as well as to all forms of environmental exposure (industrial and automobile as well as smoking). They are also the levels from which any increase would occur

when the nonsmoker encounters an environment in which smoking has raised the ambient CO levels.

Nicotine

Nicotine in the atmosphere differs from CO in that it tends to settle out of the air with or without ventilation, thereby decreasing its atmospheric concentration, whereas the CO level will remain constant until the CO is removed. The concentrations of both substances are decreased substantially by ventilation. As can be seen from data in Tables 2 and 3, under conditions of adequate ventilation, neither exceeds the maximum threshold limit values for industrial exposure (nicotine, 500 $\mu g/m^3$; CO, 50 ppm) (*1*); whereas in conditions without ventilation, smoking produces very high concentrations of both nicotine (up to 1,040 $\mu g/m^3$) and CO (110 ppm).

Nicotine in the environment is of concern because nicotine absorbed by cigarette smokers is felt to be one factor contributing to the development of atherosclerotic cardiovascular disease. Several researchers have attempted to measure the amount of nicotine absorbed by nonsmokers in involuntary smoking situations. Cano, et al. (*19*) studied urinary excretion of nicotine by persons on a submarine. Despite very low levels measured in the air (15 to 32 $\mu g/m^3$), nonsmokers showed a small rise in nicotine excretion; however, the amount excreted was still less than 1 percent of the amount excreted by smokers. Harke (*36*) measured nicotine and its main metabolite, cotinine, in the urine of smokers and nonsmokers exposed to a smoke-filled environment and reported that nonsmokers excreted less than 1 percent of the amount of nicotine and cotinine excreted by smokers. He concluded that at this low level of absorption nicotine is unlikely to be a hazard to the nonsmoker.

Russell and Feyerabend (*66*) examined the plasma and urinary nicotine values for smokers and nonsmokers under conditions of severe tobacco smoke pollution (CO 38 ppm). They demonstrated a rise in the plasma nicotine in nonsmokers to 90 ng/ml and in urinary nicotine to 80 ng/ml—values which are substantially below those for urinary nicotine found in smokers (1236 ng/ml).

Other Substances

In two studies environmental levels of the experimental carcinogen benzo(*a*)pyrene were measured. Galuskinova (*33*) found levels of benzo(*a*)pyrene from 2.82 to 14.4 $\mu g/m^3$ in smoky restaurants, but it is not clear how much of this was due to cooking and how much was due to smoking. In a study of the concentration of benzo(*a*)pyrene in the atmosphere of airplanes (*60*), only a fraction of a microgram per cubic meter was detected. The effect of chronic exposure to very low levels of this carcinogen has not been established for humans.

Brunnemann and Hoffmann (*16*) measured the levels of dimethylnitrosamine in a small room under very heavy experimental smoking and found levels of this potent carcinogen of .23 to 2.7 ng/l. When levels were measured under ambient conditions in two train bar-cars and in one bar, levels from .11 to .24 ng/l were measured. The authors state that these levels would result in the nonsmoker inhaling air containing the same quantity of nitrosamine in 1 hour as there is in the mainstream smoke of 5 to 30 cigarettes. However, it is not clear that the absorption of nitrosamine from environmental conditions is equivalent to the absorption by smoking, and it is also not established that nitrosamines can act as carcinogens at these levels delivered by inhalation.

Acrolein, acetaldehyde, and a number of other irritating substances have been measured in experimental smoking conditions (*38*, *47*, *79*, *80*, *81*, *82*) and may contribute to the eye irritation experienced in these conditions. Acrolein was the only substance that exceeded the threshold limit values even under conditions of very heavy smoke pollution.

Effects of Tobacco Smoke on the Nonsmoker

General Population

The effect of involuntary smoking on an individual is determined not only by the qualitative and quantitative aspects of the smoke-filled environment but also by the characteristics of the individual. Reactions may vary with age as well as with the sensitivity of an individual to the components of tobacco smoke. The possible effects range from minor eye and throat irritations experienced by most people in smoke-filled rooms to the anginal attacks in some persons with coronary artery disease.

In 1975, a national probability sample of U.S. telephone households was asked to agree or disagree with the statement, "It is annoying to be near a person who is smoking cigarettes" (*59*). Of "never smokers," 77.0 percent of the males and 80.5 percent of the females agreed with the statement; of current smokers, 35.o percent of the males and 34.5 percent of the females also agreed with the statement.

Speer (*72*) assessed the nature of this annoyance by interviewing 250 nonallergic patients about their reaction to cigarette smoke; 69.2 percent reported eye irritation, 31.6 percent headache, 29.2 percent nasal symptoms, and 25.2 percent cough.

Two government-sponsored studies have attempted to evaluate the degree of minor irritation due to cigarette smoke experienced by bus and plane passengers. The U.S. Department of Transportation (*69*) studied the environment on two ventilated buses—one with simulated unrestricted smoking and another with simulated smoking limited to the rear 20 percent of the seats. In one bus, lighted cigarettes were

placed at every other seat (23 cigarettes) to simulate a bus filled with smokers. In the other bus, cigarettes were placed only in the rear 20 percent of the bus (5 cigarettes) to simulate a bus where smoking was limited to the rear 20 percent of the seats. When smoking was limited, the CO level at the driver's seat was 18 ppm (ambient air 13 ppm), compared to the level of 33 ppm (ambient air 7 ppm) measured in the unrestricted smoking situation. Four of the six subjects seated in the bus reported eye irritation during the unrestricted smoking simulation. None of the six subjects, including those seated in the rear 20 percent of the bus, reported any eye irritation in the restricted smoking situation.

Several Federal agencies (*60*) cooperated to survey the symptoms experienced by travelers on both military and commercial aircraft. They distributed a questionnaire to passengers on 20 military and 8 commercial flights; 57 percent of the passengers on the military flights and 45 percent of the passengers on the commercial flights were smokers. The planes were well ventilated and CO levels were always below 5 ppm, with low levels of other pollutants as well. In spite of the low level of measurable pollution, over 60 percent of the nonsmoking passengers and 15 to 22 percent of the smokers reported being annoyed by the other passengers' smoking. These feelings were even more prevalent among those nonsmokers who had a history of respiratory disease. Seventy-three percent of the nonsmoking passengers on the commercial flights and 62 percent of the nonsmoking passengers on the military flights suggested that some remedial action be taken; 84 percent of those suggesting remedial action felt that segregating the smokers from nonsmokers would be a satisfactory solution.

Weber, et al. (*80*) found an increasing frequency of reported eye, nose, and throat irritation with increasing concentrations of smoke in a sealed chamber. Eye and nose irritation was much more frequent than throat or respiratory irritation, and self-reported eye irritation was very clearly related to objective signs such as tear flow, eye closing, and eye rubbing. The authors felt that acrolein was the major offending substance, but high concentration of other substances were also present. Artho and Koch (*10*) have reported 11 unpleasant smelling constituents in the volatile and 50 in the semivolatile phase of cigarette smoke.

The eye and nose irritation experienced by nonsmokers in a smoke-filled environment is influenced by the humidity of the air as well as by the concentration of irritating substances found in the atmosphere. Johansson and Ronge (*48, 49*) have shown that eye and nose irritation due to cigarette smoke is maximal in warm, dry air and decreases with a small rise in relative humidity. A change from acceptable to unpleasant was reported at 4.7 mg/m^3 of particulate matter for nonsmokers, and eye irritation was noted at 9 mg/m^3 for both smokers and nonsmokers. The authors concluded that a ventilation rate of 12 m^3

/hr/cig was necessary to avoid eye irritation and 50 m^3/hr/cig was necessary to avoid unpleasant odors.

The effects of cigarette smoking on the cardiovascular system of the smoker are reviewed in the Chapter on Cardiovascular Diseases. The response of the nonsmoker to cigarette smoke will be examined here. Harke and Bleichert (*39*) studied 18 adults (11 smokers and 7 nonsmokers) in a 170 m^3 room in which 150 cigarettes were smoked or allowed to burn in ashtrays for 30 minutes. They noted that the subjects who smoked during the experiment had a significant lowering of skin temperature and a rise in blood pressure. Nonsmokers who were exposed to the same smoke-contaminated environment showed no change in either of these parameters. Luquette, et al. (*56*) performed a similar experiment with 40 children exposed alternately to smoke-contaminated and clean atmospheres, but otherwise they were under identical experimental conditions. They found that exposure to the smoke was associated with increases in heart rate (5 beats per minute) and in systolic (4 mm Hg) and diastolic (5 mm Hg) blood pressure. The differences in results between these studies may be due, in part, to the age of the subjects, i.e., children may be more sensitive to the cardiovascular effects of involuntary smoking than adults; or, the increase in heart rate and blood pressure may be due to a difference between children and adults in the psychologic response to being in a smoke-filled atmosphere.

Rummel, et al. (*64*) examined this question with a group of 56 students exposed to cigarette smoke. They found a slight increase in systolic blood pressure on exposure to smoke for the entire group. When the group was divided into those who were indifferent to cigarette smoke and those who expressed a dislike for smoke, both groups had a rise in systolic blood pressure on exposure to smoke. However, the "dislike" group also had a significantly higher heart rate at the start of the study and during the entire course of the study, suggesting that psychological factors may play a role in the physiologic response to involuntary smoking.

Several authors have found small decrements in the exercise time until exhaustion (*5*), ventilation-V_{O2} max (*62*), and an increase in heart rate with exercise (*34*) after exposure to low levels of carbon monoxide. These effects are more pronounced in older than in younger populations (*5, 34*).

Pimm, et al. (*61*) examined the effect of exposure to machine-produced smoke on ventilatory function in healthy adults. They were able to show no significant changes in subdivisions of lung volume, maximum expiratory flow-volume curves, and single-breath nitrogen washout curves following exposure.

Schilling, et al. (*67*) examined the presence of self-reported symptoms and pulmonary function tests (FVC, $FEV_{1.0}$, PEF, MEF_{50}, and MEF_{25}) in 376 families with 816 children aged 1 to 17. The data did

not show any significant association between parental smoking habits and either symptoms or pulmonary function tests in spouses or children.

In summary, a substantial proportion of the normal population experiences irritation and annoyance on being exposed to cigarette smoke. The eyes and nose are the areas most sensitive to irritation, and the level of irritation increases with increasing levels of smoke contamination. Healthy nonsmokers exposed to cigarette smoke have little or no physiologic response to the smoke, and what response does occur may be due to psychological factors. There probably is a slight reduction in the maximum exercise capacity in older nonsmokers exposed to levels of CO occasionally found in involuntary smoking situations.

Effects of Carbon Monoxide in Psychomotor Tests

There has been some concern over the effects of relatively low levels of carbon monoxide on psychomotor functions (the ability to perceive and react to stimuli), especially on those functions related to driving an automobile. Yabroff, et al. (*85*) recently reviewed this topic extensively. They concluded that "experimenters have found some performance tasks associated with driving affected by low levels of carboxyhemoglobin, some as low as 2 percent. However, disagreement exists regarding the levels at which particular tasks are affected. These tasks include:

1. Vigilance—both visual and acoustical—needed for defensive driving.
2. Color vision and discrimination, especially important in discerning taillight or brake light usage and traffic lights.
3. Brightness discrimination, important to driving as a clue used in distance estimation.
4. Peripheral vision, used in surveying the environment, signs, and other traffic.
5. Glare recovery, which is the ability to recover visual acuity after being subjected to bright lights of another motor vehicle at night or in going from bright sunshine into a shaded area (e.g., a tunnel).
6. Speech linkage"(*85*).

A number of authors have tested driving ability directly. Ray and Rockwell (*63*) found that as COHb increased time estimates were shorter, distance estimates were longer, and taillight discrimination and determination of velocity change in the lead car took longer. There were also slight changes in normal driving and cornering. Weir and Rockwell (*84*) also found slight deterioration in driving performance; measurements of visual acuity showed that drivers required more time to retrieve visual information and spent less time looking outside the forward direction (20 degrees x 20 degrees visual angle). These changes were noted at 6 to 8 percent COHb and are similar to those

found in drivers under low alcohol concentrations. The combined effect of alcohol and CO has been studied and no additional impairment due to CO could be demonstrated for tests of coordination or cognitive function (*58*). When actual driving skills were tested (*83*), significant interactions between CO and alcohol occurred for tasks which demanded higher information processing such as curve negotiation and car following (at 12 percent COHb).

In summary, it is possible to demonstrate changes in psychomotor function at levels of CO found in involuntary smoking conditions, but these effects generally are measurable only at the threshold of stimuli perception. Effects of CO on driving performance and interactive effects of CO and alcohol have been demonstrated only for levels of COHb above those found in involuntary smoking conditions.

Special Populations

The above studies examined the effects of involuntary smoking on relatively healthy populations. An exposure that is harmless for someone who is healthy may have a very different effect on someone with heart or lung disease or hypersensitivity to substances found in smoke. Children are also a group in which effects may differ, due to their greater ventilation per body weight. This section will review the evidence on the effects of involuntary smoking for each of these special populations.

Cardiovascular Disease

Carbon monoxide, which has 230 times the affinity of oxygen for hemoglobin, impairs oxygen transport in two ways. First, it competes with oxygen for hemoglobin binding sites. Second, it increases the affinity of the remaining hemoglobin for oxygen, thereby requiring a larger gradient in PO_2 between the blood and tissue to deliver a given amount of oxygen. Carbon monoxide also binds to other heme-containing pigments, most notably myoglobin, for which it has an even greater affinity than for hemoglobin under conditions of low PO_2. The significance of this binding is unclear but may be important in tissues such as heart muscle, which have both high oxygen requirements and large amounts of myoglobin.

In healthy individuals, the levels of COHb due to involuntary smoking are probably functionally insignificant, with small changes demonstrable only under extreme exertion. In individuals with a limited cardiovascular reserve, however, any reduction in the oxygen-carrying capacity of the blood may be of greater importance.

Ayres, et al. (*11, 12*) exposed a group of patients to various concentrations of CO (COHb 9 percent), and found that they had lower arterial and mixed venous PO_2's, decreased lactate extraction, and decreased coronary sinus PO_2.

Aronow and Isbell (*9*) and Anderson, et al. (*2*) have shown a decrease in the mean duration of exercise before onset of pain in patients with angina pectoris exposed to low levels of carbon monoxide (50 and 100 ppm). Carboxyhemoglobin levels were significantly elevated (2.9 percent after 50 ppm; 4.5 percent after 100 ppm), and the systolic blood pressure, heart rate, and product of systolic blood pressure times heart rate (a measure of cardiac work) were all significantly lower at the onset of angina pectoris.

In a continuation of this work, Aronow, et al. (*6, 8*) studied eight patients with angiographically demonstrated coronary artery disease (> 75 percent obstruction of at least one coronary artery) during two separate cardiac catheterizations. During the first, each patient smoked three cigarettes; during the second, each patient inhaled carbon monoxide until the maximal coronary sinus COHb level equaled that produced by smoking during the first catheterization. Smoking increased the systolic and diastolic blood pressure, heart rate, left ventricular end-diastolic pressure (LVEDP), and coronary sinus, arterial, and venous CO levels. No changes were noted in left ventricular contractility (dp/dt), aortic systolic ejection period, or cardiac index; decreases were found in stroke index and coronary sinus, arterial, and venous PO_2. When carbon monoxide was inhaled, increased LVEDP and coronary sinus, arterial, and venous CO levels were noted; there were no changes in systolic and diastolic blood pressure, heart rate, or systolic ejection period; and decreases in left ventricular dp/dt, stroke index, cardiac index and coronary sinus, arterial and venous PO_2 were found. These data suggest that carbon monoxide has a negative ionotropic effect on myocardial tissue resulting in the decreased contractility (dp/dt) and stroke index. When the positive effect of nicotine on contractility and heart rate is added by smoking, the net effect is increased cardiac work for the same cardiac output.

Aronow (*4*) also examined the effect of involuntary smoking on patients with angina pectoris. Ten patients (two smokers and eight nonsmokers) were exercised after a control exposure to uncontaminated air, after exposure to 15 cigarettes smoked over 2 hours in a well ventilated (30.8 m^3) room, and after exposure to 15 cigarettes smoked over 2 hours in an unventilated (30.8 m^3) room. He reported that the carboxyhemoglobin levels rose from 1.25 percent in the control situation to 1.77 percent after exposure in the ventilated room, and to 2.28 percent in the unventilated room. He found that the mean time of exercise until onset of angina decreased 22 percent after exposure in the ventilated room and 38 percent after exposure in the unventilated room. The patients also had onset of angina at a lower heart rate and systolic blood pressure. He also noted that the patients had an elevation in their heart rate and systolic and diastolic blood pressures. He attributed this to the possible absorption of nicotine (no nicotine

levels were measured). The very low levels of nicotine absorption documented under these conditions (see the previous section) make it unlikely that nicotine would be responsible for these physiologic changes. Another explanation would be the anxiety or aggravation induced by the smoke-filled room resulting in a stress response (*78*). The combination of elevated blood pressure and pulse at the start of exercise and the elevation in carboxyhemoglobin levels resulted in a greater decline in exercise time to produce angina for the measured level of carboxyhemoglobin than had been shown for carbon monoxide exposure alone.

In summary, there is evidence that elevations in carboxyhemoglobin levels capable of being produced by involuntary smoking can reduce the exercise duration required to induce angina in some patients with coronary artery disease.

Chronic Obstructive Lung Disease

Patients with chronic lung disease represent a second group who are limited in their ability to exercise and who might be particularly susceptible to involuntary smoking exposures. Aronow, et al. (*7*) exercised 10 patients with hypoxic chronic lung disease (PO_2 less than 70 torr) before and after a 1-hour exposure to 100 ppm CO (COHb increased from 1.43 percent to 4.08 percent). There was a significant reduction in the mean exercise time, from 218.5 seconds to 146.6 seconds, until marked dyspnea. There was no difference in exercise mean systolic or diastolic blood pressure, heart rate, product of systolic blood pressure times heart rate/100, or arterial P_{O2}, P_{CO2}, or pH before or after CO exposure. The mechanism for this earlier induction of dyspnea remains unclear because decreased oxygen transport to the exercising tissues should have been reflected in a shift to anaerobic metabolism and the development of acidosis.

Hypersensitivity

The evidence for possible immunologic reactions to tobacco smoke is reviewed in the allergy chapter of this report; the existence of a true tobacco allergy has not been clearly established. It does seem clear, however, that those patients with a history of allergies to other substances are more likely to report the irritating effects of tobacco smoke (*32*, *72*).

Children

Children have a higher incidence of respiratory infections than adults and may be more susceptible to air pollutants than adults due to their greater minute ventilation per body weight. Several researchers have investigated the effects of parental smoking on the health of children. Cameron, et al. conducted two telephone surveys of Detroit families to

determine the relationship between children's respiratory illness and parental smoking habits. In the first survey (*17*), they found a statistically significant relationship between the prevalence of children's respiratory infection and parental smoking habits only when all children under 16 were considered but not when only those under 9 or under 5 were considered. In a larger survey of the same city (*18*), they found a relationship between parental smoking and prevalence of respiratory illness in the 10- to 16-year age group and in the birth to 5-year age group. Neither study was controlled for smoking by the children, which might be a factor in the 10- to 16-year age group, or for socioeconomic status, which has an effect on both smoking habits and illness. However, the data suggested a higher prevalence of respiratory disease in families where there are smokers than in nonsmoking families.

Colley, et al. (*21*) also found a relationship between parental smoking habits and the prevalence of respiratory illness in the children. However, an even stronger relationship was found between parental cough and phlegm production and respiratory infections in children. They postulated that this latter relationship resulted from the greater infectivity of these parents due to their cough and phlegm production. The relationship between parental cigarette smoking and respiratory infection in their children would then occur because cigarette smoking caused the parents to cough and produce phlegm and would not be indicative of a direct effect of cigarette smoke-filled air on the children. Lebowitz and Burrows (*53*) found a similar relationship, but Schilling, et al. (*67*) did not.

Harlap and Davies (*42*) studied infant admissions to Hadassah Hospital in West Jerusalem and found a relationship between admissions for bronchitis and pneumonia in the first year of life and maternal smoking habits during pregnancy. Data on maternal smoking habits after the birth of the child were not obtained, but it can be assumed that most of the mothers who smoked during pregnancy continued to smoke during the first year of the infant's life. A relationship between infant admission and maternal smoking habits was demonstrable only between the sixth and ninth months of infant life and was more pronounced during the winter months. Mothers who smoke during pregnancy are known to have infants with a lower average birth weight than the infants of nonsmoking mothers. The relationship between maternal smoking and their infants' admission to the hospital found in this study was greater for low birth-weight infants, but the same relationship was found for normal birth-weight infants (Table 5) (*42*). Harlap and Davies (*42*) demonstrated a dose-response relationship for maternal smoking and infant admission for bronchitis and pneumonia; however, they also found a relationship between maternal smoking and infant admissions for poisoning and injuries. This may indicate a bias in the study due to relationships