TABLE 7. – *Means of the numerical values given lung sections at autopsy of male former cigarette smokers, standardized for age*

Formerly Smoked	Stopped ≥ 10 yr.		Stopped < 10 yr.	
	<1 Pk.	Pk.	<1 Pk.	Pk.
Number of Subjects	35	66	51	131
Emphysema	0.24	0.70	1.08	1.69
Fibrosis	1.14	1.74	2.44	3.30
Thickening or arterioles	0.57	0.93	1.25	1.59
Thickening of arteries	0.04	0.16	0.36	0.61

NOTE. – Numerical values for each finding were determined by rating each lung section on scales of 0–4 for emphysema and thickening of the arterioles, 0–7 for fibrosis, and 0–3 for thickening of the arteries.

Source: Auerbach, O., et al. (*2*).

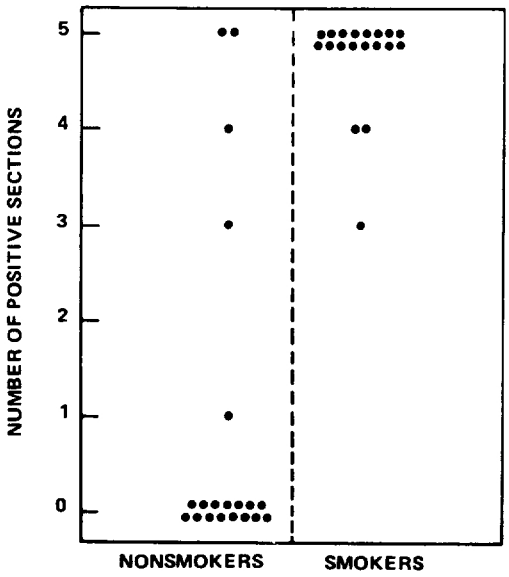
bronchiolitis associated with clusters of pigmented alveolar macrophages in the lungs of smokers. They found these changes only rarely in the lungs of nonsmokers (Fig. 1). The smokers were young (average age 25.7 years), were a heavy smoking population (average 20.1 pack years), but did not differ significantly from the nonsmokers in age, social class, or pollution exposure. However, 12 of the 19 smokers had had productive cough or frequent cold compared to only 3 of the 20 nonsmokers. These authors postulated that bronchiolitis may be responsible for the abnormalities in the tests of small airway function of smokers.

Pathophysiologic Studies in Humans

Yeager, et al. (*48*) showed decreased pinocytosis in human alveolar macrophages obtained from asymptomatic cigarette smoking volunteers when compared to those obtained from nonsmoking controls.

Warr and Martin (*46*) studied alveolar macrophages lavaged from four healthy smokers and four healthy nonsmokers. Only two members of each group were reactive to delayed hypersensitivity skin tests for *Candida albicans.* Macrophages from nonsmokers responded to Migration Inhibitory Factor (MIF) by a depression in migration of at least 30 percent, whereas macrophages from smokers did not respond to MIF. The cells from smokers were noted to migrate three times faster than those from nonsmokers. When *Candida* antigen was added to the medium, cells from the nonreactive subjects (both

FIGURE 1.—Respiratory bronchiolitis in smokers and control group



NOTE.—The position of each symbol represents the number of sections per case in which bronchiolitis was identified.

Source: Niewoehner, D.E., et al. (*24*).

smokers and nonsmokers) were not inhibited, the cells from the reactive nonsmokers were inhibited, but the cells from reactive smokers were not inhibited. Thus, macrophages from smokers did not respond normally to either MIF or antigenic challenge.

Pathophysiologic Studies in Animals

Roszman and Rogers (*27*) noted that either the nicotine or the water soluble fraction of whole cigarettes smoked suppressed the immunoglobulin response of lymphoid cell cultures. When concentrations of over 200 micrograms per milliliter of nicotine of the water soluble fraction were added, they were able to completely suppress the immunoglobulin response and to observe this suppression even in cells exposed for 2 hours prior to the antigenic challenge.

Guinea pigs (*29*) exposed to the smoke of five cigarettes and then lavaged 2 hours later had fewer pulmonary macrophages and leukocytes in the lavage fluid than did controls not exposed to smoke. The decrease in the number of macrophages was highly correlated with acetaldehyde, tar, nicotine, hydrogen cyanide, and

acrolein concentrations in the cigarette smoke. The decrease in the number of leukocytes was more closely correlated with pH of the particulate phase and concentrations of acetaldehyde and tar.

Tracheal mucous velocity has been shown to be decreased in purebred beagle dogs (*45*) exposed to 100 cigarettes per week for 13.5 months. In donkeys (*1*) low level exposure to whole cigarette smoke accelerated tracheobronchial clearance, whereas at intermediate and high levels of exposure, clearance was decreased. At high exposure levels whole cigarette smoke had twice the effect of filtered smoke in decreasing clearance.

SUMMARY OF RECENT BRONCHOPULMONARY FINDINGS

1. Cigarette smokers with mild viral respiratory illnesses have been shown to develop abnormal but reversible changes in certain pulmonary function tests while nonsmokers show no changes in these tests. Cigarette smokers have also been shown to have a significantly longer duration of respiratory symptoms following mild viral illness than nonsmokers.

2. Cigarette smoking is more closely related to COPD than is air pollution under the conditions of air pollution encountered by the average person. The possibility remains that the two kinds of exposure may interact to increase the total effect beyond that contributed by each exposure.

3. Cigarette smokers without respiratory symptoms have evidence of small airway dysfunction (elevated closing volumes) more frequently than do nonsmokers without respiratory symptoms.

4. Autopsy studies have shown a dose-response relationship between cigarette smoking and the microscopic changes of COPD. Data from one study indicate that bronchiolitis may be a far more common finding in cigarette smokers than in nonsmokers.

5. Pulmonary macrophages from cigarette smokers' lungs have a decreased ability to respond to in vitro antigenic stimuli as compared to macrophages from smokers.

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CHAPTER 4.
Involuntary Smoking

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INTRODUCTION

The effects of smoking on the smoker have been extensively studied, but the effects of tobacco smoke on nonsmokers have received much less attention. The 1972 Health Consequences of Smoking (*49*) reviewed the effects of public exposure to the air pollution resulting from tobacco smoke. This exposure has been called "passive smoking" by many authors, but will be referred to in this report as "Involuntary Smoking." The term involuntary smoking will be used to mean the inhalation of tobacco combustion products from smoke-filled atmospheres by the nonsmoker. This type of exposure is, in a sense, "smoking" because it provides exposure to many of the same constituents of tobacco smoke that voluntary smokers experience. It is also "involuntary" because the exposure occurs as an unavoidable consequence of breathing in a smoke-filled environment.

The chemical constituents found in an atmosphere filled with tobacco smoke are derived from two sources – mainstream and sidestream smoke. Mainstream smoke emerges from the tobacco product after being drawn through the tobacco during puffing. Sidestream smoke rises from the burning cone of tobacco. Mainstream and sidestream smoke contribute different concentrations of many substances to the atmosphere for several reasons: Different amounts of tobacco are consumed in the production of mainstream and sidestream smoke; the temperature of combustion differs for tobacco during puffing or while smouldering; and certain substances are partially absorbed from the mainstream smoke by the smoker. The amount of a substance absorbed by the smoker depends on the characteristics of the substance and the depth of inhalation by the smoker. As discussed in the 1972 Report, when the smoker does not inhale the smoke into his lungs, the smoke he exhales contains less than half its original amount of water-soluble volatile compounds, four-fifths of the original nonwater-soluble compounds and particulate matter, and almost all of the carbon monoxide (*15*). When the smoker inhales the mainstream smoke, he exhales into the atmosphere less than one-seventh of the amount of volatile and particulate substances that were originally present in the smoke and also reduces the exhaled CO to less than half its original concentration (*16*). As a result, different concentrations of substances are found in exhaled mainstream smoke depending on the tobacco product, composition of the tobacco, and degree of inhalation by the smoker.

Several minor symptoms (conjunctival irritation, dry throat, etc.) are caused by levels of cigarette smoke encountered in everyday life, and serious allergic-like reactions to cigarette smoke may occur in some sensitive individuals. A major concern, however, about atmospheric contamination by cigarette smoke has been due to the production of significant levels of carbon monoxide. Cigarette smoking in poorly ventilated enclosed spaces may generate carbon monoxide levels above the acceptable 8-hour industrial exposure limits (50 ppm) – set by the American Conference of Government Industrial Hygienists (*1*). Exposure to this level of carbon monoxide even for short periods of time has been shown to reduce significantly the exercise tolerance of some persons with symptomatic cardiovascular disease. There is also some evidence that prolonged exposure to this level of carbon monoxide in combination with a high cholesterol diet can enhance experimental atherosclerosis in animals (Chapter 1, Cardiovascular Diseases).

In the present chapter, the effects of cigarette smoke on the environment and on the nonsmoker in that environment will be examined by reviewing data on (1) the constituents of cigarette smoke measured under various conditions, and (2) the physiologic effects of this "involuntary smoking" on individuals.

CONSTITUENTS OF TOBACCO SMOKE

In a recent workshop on the effects of environmental tobacco smoke on the nonsmoker (*41*), Corn (*14*) presented a compilation adapted from Hoegg (*32*) of some of the substances in mainstream cigarette smoke and the ratio of sidestream to mainstream levels for some of these substances (Table 1). The actual numerical value of the sidestream to mainstream concentration ratio will vary with different types of tobacco tested, but Table 1 gives values generally consistent with those found by others (*34, 42*). Many of these substances including nicotine and carbon monoxide are found in much higher concentrations in sidestream smoke than in mainstream smoke, establishing that the smoke exposure received by both the smoker and nonsmoker due to breathing in a smoke-filled environment differs qualitatively as well as quantitatively from the smoke exposure received by the smoker who inhales through a lighted cigarette. A more comprehensive recent review of the constituents of mainstream and sidestream smoke has also been provided by Schmeltz, et al. (*42*) and Johnson, et al. (*34*).

TABLE 1. – *Comparison of mainstream and sidestream cigarette smoke*[1,2]

	Compound	Mainstream (mg/cig)	Sidestream (mg/cig)	Ratio Sidestream/ Mainstream	Comment
A	General characteristics				
	Duration of smoke production	20 sec	550 sec	27	
	Tobacco burnt	347	411	1.2	
	Particulates, no. per cigarette	1.05×10^{12}	3.5×10^{12}	3.3	
B	Particulate phase				
	[2]Tar (chloroform extract)	20.8	44.1	2.1	
		10.2	34.5	3.4	Filter cigarette
	Nicotine	0.92	1.69	1.8	
		0.46	1.27	2.8	Filter cigarette
	Benzo(a)pyrene	3.5×10^{-5}	13.5×10^{-5}	3.7	
	Pyrene	13×10^{-5}	39×10^{-5}	3.0	
	Total phenols	0.228	0.603	2.6	
	Cadmium	12.5×10^{-5}	45×10^{-5}	3.6	
C	Gases and vapors				
	Water	7.5	298	39.7	3.5 mg of Mainstream and 5.5 mg of Sidestream in particulate phase, rest in vapor phase
	Ammonia	0.16	7.4	46	
	Carbon monoxide	31.4	148	4.7	
	Carbon dioxide	63.5	79.5	1.3	
	Oxides of Nitrogen	0.014	0.051	3.6	

[1] Adapted from Hoegg, U.R. (*31, 32*).

[2] For 35 ml puff volume, 2 sec puff duration, one puff per minute and 23 or 30 mm butt length and 10 percent tobacco moisture.

Source: Corn, M. (*14*).

A number of other researchers have attempted to measure the levels of some of the substances in cigarette smoke encountered in everyday situations (Table 2). They have also tried to determine the factors controlling the atmospheric concentrations of these substances as well as the amount absorbed by nonsmokers under these conditions. Carbon monoxide, nicotine, benzo(a)pyrene, acrolein, and acetaldehyde have been of particular concern.

Carbon Monoxide

Levels of carbon monoxide (CO), a major product of tobacco combustion, have been studied in a variety of situations, and concentrations ranging from 2 to 110 ppm have been measured (Table 2). 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 (CO removal).

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 pre puff and per gram of tobacco burned than filtered or unfiltered cigarettes (*8*). This greater production of CO by cigars was confirmed by Harke (*23*). 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).

In addition to the effect of type of tobacco product on CO levels, data on the effects of room size, amount of tobacco burned, and ventilation are included in Table 2. Only under conditions of unusually heavy smoking and poor ventilation did CO levels exceed the maximum permissible, 8-hour industrial exposure limit of 50 ppm CO (*1*); however, even in cases where the ventilation was adequate, the measured CO levels did exceed the maximum acceptable ambient level of 9 ppm (*18*).

Harke (*27*) also showed that in small enclosed ventilated spaces (an automobile) the CO level is determined more by the number of cigarettes being smoked at one given time than by the cumulative number of cigarettes that have been smoked; also the CO level decreases rapidly once the smoking stops.

TABLE 2. – Measurements of constituents released by the combustion of tobacco products in various situations

[Cig = cigarettes; – = unknown; TPM = total particulate matter]

Reference, Location, and Dimensions If Known	Ventilation	Amount of Tobacco Burned	Constituents
Harke, H.-P., et al. (*27*)			
Mid-size European car, engine off, in wind tunnel at 50 km/hr wind speed	None	9 cig	30 ppm CO
	Air jets open & blower off	6 cig	20 ppm CO
	Air jets open & 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 & blower on	6 cig	8-10 ppm CO
Harke, H.-P., Peters, H. (*28*)			
Car in traffic	None	4 cig	21.4 ppm CO
Srch, M. (*45*)			
Car, engine off– 2.09 m^3	None	10 cig in 1 hr	90 ppm CO, Smokers 10% COHb Nonsmokers 5% COHb
Seiff, H.E. (*44*)			
Intercity buses	15 air changes per hr	23 cig (burning continuously)	33 ppm CO (at driver's seat)
		3 cig (burning continuously)	18 ppm CO (at driver's seat)

TABLE 2. – *Measurements of constituents released by the combustion of tobacco products in various situations – Continued*

[Cig = cigarettes; – = unknown; TPM = total particulate matter]

Reference, Location, and Dimensions If Known	Ventilation	Amount of Tobacco Burned	Constituents
U.S. Dept. Transportation, et al. (*48*)			
Airplane flights:			
Overseas–100% filled	15-20 air changes per hr	–	2-5 ppm CO, <.120 mg/m^3 TPM
Domestic–66% filled	do.	–	<2 ppm CO, <.120 mg/m^3 TPM
Cano, J.P., et al. (*11*)			
Submarines–66 m^3	Yes	157 cig per day	<40 ppm CO, 32 μg/m^3 Nicotine
		94-103 cig per day	<40 ppm CO, 15-35 μg/m^3 Nicotine
Godin, G., et al. (*21*)			
Ferry boat compartments:			
Smoking	–	–	18.4 ±8.7 ppm CO
Nonsmoking	–	–	3.0±2.4 ppm CO
Theater:			
Foyer	–	–	3.4±0.8 ppm CO
Auditorium	–	–	1.4±0.8 ppm CO
Bridge, D.P., Corn, M. (*7*)			
Party rooms:			
145 m^3	7 air changes per hr	50 cig & 17 cigars in 1.5 hr	7 ppm CO
101 m^3	10.6 air changes per hr	63 cig & 10 cigars in 1.5 hr	9 ppm CO

TABLE 2. – *Measurements of constituents released by the combustion of tobacco products in various situations – Continued*

[Cig = cigarettes; – = unknown; TPM = total particulate matter]

Reference, Location, and Dimensions If Known	Ventilation	Amount of Tobacco Burned	Constituents
Harke, H.-P., et al. (*25*)			
Room – 38.2 m^3	None	30 cig per 13 min (by machine)	64 ppm CO, 510 $\mu g/m^3$ Nicotine .46 mg/m^3 Acrolein 6.5 mg/m^3 Acetaldehyde
		5 cig per 13 min (by machine)	11.5 ppm CO, 60 $\mu g/m^3$ Nicotine, .07 mg/m^3 Acrolein, 1.3 mg/m^3 Acetaldehyde
Harke, H.-P. (*24*)			
Office Bldg	Air conditioned	–	<5 ppm CO
Office Bldg	Not air conditioned	–	<5 ppm CO
Room – 78.3 m^3	–	3 smokers	15.6 ppm CO
Harke, H.-P., (*23*)			
Room – 57 m^3	None	42 cig (by machine)	50 ppm CO, 530 $\mu g/m^3$ Nicotine
	7.2 air changes per hr	42 cig do.	10 ppm CO, 120 $\mu g/m^3$ Nicotine
	8.4 air changes per hr	42 cig do.	<10 ppm CO, <100 $\mu g/m^3$ Nicotine
	None	9 cigars do.	60 ppm CO, 1040 $\mu g/m^3$ Nicotine
	7.2 air changes per hr	9 cigars do.	20 ppm CO, 420 $\mu g/m^3$ Nicotine
	None	9 pipes do.	10 ppm CO, 520 $\mu g/m^3$ Nicotine
	7.2 air changes per hr	9 pipes do.	<10 ppm CO, <100 $\mu g/m^3$ Nicotine

TABLE 2. – *Measurements of constituents released by the combustion of tobacco products in various situartions – Continued*
[Cig = cigarettes; – = unknown; TPM = total particulate matter]

Reference, Location, and Dimensions If Known	Ventilation	Amount of Tobacco Burned	Constituents
Harke, H.-P. (*23*) Room–170 m^3	None	105 cig	30 ppm CO, Smokers 7.5% COHb Nonsmokers 2.1% COHb
	1.2 air changes per hr	107 cig	5 ppm CO, Smokers 5.8% COHb Nonsmokers 1.3% COHb
	2.3 air changes per hr	101 cig	75 ppm CO, Smokers 5.0% COHb Nonsmokers 1.6% COHb
Anderson, G., Dalhamn, T. (*3*) Room–80 m^3	6.4 air changes per hr	46 cig & 3 pipefuls	4.5 ppm CO, 377 $\mu g/m^3$ Nicotine, 3.0 mg/m^3 TPM
Russell, M.A.H., et al. (*40*) Room–43 m^3	None	80 cig & 2 cigars per hr	38 ppm CO, Smokers 9.6% COHb Nonsmokers 2.6% COHb
Harmsen, H., Effenberger, E. (*30*) Room–93 m^3	None	62 cig in 2 hrs	80 ppm CO, 5200 $\mu g/m^3$ Nicotine
Hoegg, U.R. (*31, 32*) 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

One must be careful when using the levels recorded in Table 2 as measures of individual exposure because the CO levels were usually measured at points several feet from the nearest smoker and probably would have been higher if measured at points corresponding to the position of a person sitting next to someone actively smoking (*17, 35*). In addition, it is the CO absorbed by the body that causes the harmful effects and not that which is measured in the atmosphere. This absorption can vary from individual to individual, depending on factors such as duration of exposure, volume of air breathed per minute, and cardio-respiratory function.

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*) were unable to find any change in the COHb levels of nonsmokers in a well ventilated room where the CO level was 4.5 ppm. When Harke (*23*) studied nonsmokers under similar conditions (good ventilation and less than 5 ppm CO), he was able to show an increase in COHb level from 1.1 to 1.6 percent; without 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. (*40*) also found that COHb levels increased from 1.6 to 2.6 percent in nonsmokers exposed to a smoke-filled 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.

Stewart, et al. (*46*) 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 3). These levels represent the total CO exposure from all sources, involuntary smoking, and other sources of pollution as well as establishing the levels which would be added to any new involuntary smoking exposure.

Increases in the COHb levels of this magnitude are probably functionally insignificant in the healthy adult, but in persons with angina pectoris, any reduction of oxygen-carrying capacity is of great importance. In this disease, the volume of blood able to be pumped through the diseased coronary artery is already unable to meet the demands of the heart muscle under exercise stress. Aronow, et al. (*4*) examined the effect of exposure to carbon monoxide on persons with angina pectoris. They exercised persons with angina

TABLE 3. – *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., et al. (*46*).

pectoris before and after exposure to carbon monoxide. The average amount of exercise that was able to be performed before a person developed chest pain was significantly shortened from 226.7 seconds before exposure to 187.6 seconds after CO exposure. This change occurred after a 2-hour exposure to 50 ppm CO and with an increase in COHb level from 1.03 percent to 2.68 percent; these COHb levels are within the range produced by involuntary smoking.

These data indicate that exposure to CO at levels found in some involuntary smoking situations may well have a significant impact on the functional capacity of persons with angina pectoris. Carbon monoxide has also been shown to decrease cardiac contractility in persons with coronary heart disease at COHb levels similar to those produced due to involuntary smoking situations (*5*). It is reasonable to assume that any significant CO exposure to the diseased heart reduces its functional reserve.

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 Table 2, 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 $\mu g/m^3$; 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. (*11*) 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 did show a small rise in nicotine excretion; however, the amount excreted was still less than 1 percent of the amount excreted by smokers. Harke (*23*) measured nicotine and its 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 feels that at this low level of absorption nicotine is unlikely to be a hazard to the nonsmoker.

Other Substances

In two studies environmental levels of the experimental carcinogen benzo(a)pyrene were measured. Galuskinova (*20*) found levels of benzo(a)pyrene from 2.82 to 14.4 mg/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 (*48*), 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.

Acrolein and acetaldehyde have also been measured in smoke-filled rooms (*25*, Table 2) and may contribute to the eye irritation commonly experienced in these situations.

EFFECTS OF EXPOSURE TO CIGARETTE SMOKE

Cardiovascular Effects of Involuntary Smoking

The effects of cigarette smoking on the cardiovascular system of the smoker are well established, but very little is known about the cardiovascular response of the nonsmoker to cigarette smoke. Harke and Bleichert (*26*) studied 18 adults (11 smokers and 7 nonsmokers) in a room 170 m^3 large 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. Non-smokers who were exposed to the same smoke-contaminated environment showed no change in either of these parameters. Luquette, et al. (*36*) performed a similar experiment with 40 children exposed alternately to smoke-contaminated and clean atmospheres, but otherwise under identical experimental conditions. They found that exposure to the smoke caused 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.

Effects of Carbon Monoxide on Psychomotor Tests

Carbon monoxide from tobacco smoke, automobile exhaust, and industrial pollution is an important component of air pollution. There has been some concern over the effect of relatively low levels of carbon monoxide on psychomotor functions (the ability to perceive and react to stimuli), especially those functions related to driving an automobile (Table 4).

Carbon monoxide levels occasionally reached in some involuntary smoking situations result in measurable cognitive and motor effects, but these effects generally are measurable only at the threshold of stimuli perception. One study (Wright, et al., (*50*)) found that the safe driving habits measured on a driving simulator did not improve as much with practice in a group exposed to CO as did the habits of a control group. Another study (*37*) with a different experimental design but at the same levels of CO did not find any effect on complex psychomotor activity such as driving a car. Thus, the role of CO alone in motor vehicle accidents remains unclear. The effect on judgement and reactions of CO in combination with factors such as fatigue and alcohol, conditions known to influence judgement and reaction time, has not been determined.

Pathologic Effects of Exposure to Cigarette Smoke

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 largely 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 severity of possible effects range from minor eye and throat irritations experienced by most people in smoke-filled rooms, to the anginal attacks of some persons with cardiovascular disease.

The minor symptomatic irritation experienced by nonsmokers in a smoke-filled environment is influenced by the humidity of the air as well as the concentration of irritating substances found in the atmosphere. Johansson and Ronge (*33*) have shown that 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.

TABLE 4.– *Effects of carbon monoxide on psychomotor functions*

Reference	Test or Measurement	CO level (ppm)	COHb level (Percent)	Effect
McFarland, R.A. (*37*)	Ability of drivers to stay between two-lane markers while being permitted only brief glimpses of the road		6	None
			11	None
			17	None
Ray, A.M., Rockwell, T.H. (*39*)	Reaction time to car taillights		10	Prolonged
McFarland, R.A. (*38*)	Performance of two tasks at same time	700	17	None
	Dark adaptation and glare recovery	700	17	None
	Peripheral vision at 10° and 30°	700	17	None
	Peripheral vision at 20°	700	17	Decreased
	Depth perception	700	17	None
Stewart, R.D., et al. (*47*)	Time perception	500	20	None