TABLE 2e.—Nitrogen oxides measured under realistic conditions

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels		Nonsmoking controls (ppb)	
					Mean	Range	Mean	Range
Fischer et al. (*1978*) and Weber et al. (*1979*)	Restaurant	50–80/470 m^3	Mechanical	27 × 30 min samples	NO_2: 76	59–105	63 (outdoors)	
					NO: 120	36–218	115 (outdoors)	
	Restaurant	60–100/440 m^3	Natural	29 × 30 min samples	NO_2: 63	24–99	50 (outdoors)	
					NO: 80	14–21	11 (outdoors)	
	Bar	30–40/50 m^3	Natural, open	28 × 30 min samples	NO_2: 21	1–61	48 (outdoors)	
					NO: 195	66–414	44 (outdoors)	
	Cafeteria	80–150/574 m^3	11 changes/hr	24 × 30 min samples	NO_2: 58	35–103	34 (outdoors)	
					NO: 9	2–38	4 (outdoors)	
				Other—non-smokers room			NO_2: 27	15–44
							NO: 5	2–9
Weber and Fischer (*1980*)[a]	44 offices	Varied	Varied	348–354 samples	NO_2: 24 ± 22	115 (peak)	Values not given	
					NO: 32 ± 60	280 (peak)	Values not given	

[a]Control values (unoccupied rooms) have been subtracted.

TABLE 2f.—Nitrosamines measured under realistic conditions

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels (ng/L)	
					Mean	Range
					N-Nitrosodimethylamine	
Brunneman and Hoffmann (*1978*)	Train bar car	Not given	Mechanical	90 min continuous	0.13	
	Train bar car	Not given	Natural	90 min continuous	0.11	
Brunneman et al. (*1977*)	Bar	Not given	Not given	3 hr continuous	0.24	
	Sports hall	Not given	Not given	3 hr continuous	0.09	
	Betting parlor	Not given	Not given	90 min continuous	0.05	
	Discotheque	Not given	Not given	2¾ hr continuous	0.09	
	Bank	Not given	Not given	5 hr continuous	0.01	
	House	Not given	Not given	4 hr continuous	< 0.005	
	House	Not given	Not given	4 hr continuous	< 0.003	

TABLE 2g.—Particulates measured under realistic conditions

Study	Type of premises	Occupancy (active smokers per 100 m³)	Ventilation	Monitoring conditions (min)	Levels (μg/m³) Mean	Levels (μg/m³) Std. dev.	Nonsmoking controls (μg/m³) Mean	Nonsmoking controls (μg/m³) Std. dev.
Repace and Lowrey (*1980*)	Cocktail party	0.75	Natural	15	351	± 38	24	
	Lodge hall	1.26	Mechanical	50	697	± 28	60[1]	
	Bar and grill	1.78	Mechanical	18	589	± 28	63[1]	
	Firehouse bingo	2.77	Mechanical	16	417	± 63	51[1]	
	Pizzeria	2.94	Mechanical	32	414	± 58	40[1]	
	Bar/cocktail lounge	3.24	Mechanical	26	334	± 120	50[1]	
	Church bingo game	0.47	Mechanical	42	279	± 18	30	
	Inn	0.74	Mechanical	12	239	± 9	22[1]	
	Bowling alley	1.53	Mechanical	20	202	± 19	49[1]	
	Hospital waiting room	2.15	Mechanical	12	187	± 52	58[1]	
	Shopping plaza restaurant							
	Sample 1	0.18	Mechanical	18	153	± 8	59[1]	
	Sample 2	0.18	Mechanical	18	163	± 4	36[1]	
	Barbeque restaurant	0.89	Mechanical	10	136	± 17	40[1]	
	Sandwich restaurant A							
	Smoking section	0.29	Mechanical	20	110	± 36	40[1]	
	Nonsmoking section	0	Mechanical	20	55	± 5	30	
	Fast-food restaurant	0.42	Mechanical	40	109	± 38	24[1]	
	Sports arena	0.09[2]	Mechanical	12	94	± 13	55[1]	
	Neighborhood restaurant/bar	0.40	Mechanical	12	93	± 17	55[1]	
	Hotel bar	0.59	Mechanical	12	93	± 2	30	
	Sandwich restaurant B							
	Smoking section	0.13	Mechanical	8	86	± 7	55	
	Nonsmoking section	0	Mechanical	21	51			
	Roadside restaurant	1.12	Mechanical (9.5 ach[3])	18	107[4]		30	
	Conference room	3.54	Mechanical (4.3 ach[3])	6	1947[4]		55	

TABLE 2g.—Continued

Study	Type of premises	Occupancy (active smokers per 100 m^3)	Ventilation	Monitoring conditions (min)	Levels ($\mu g/m^3$)		Nonsmoking controls ($\mu g/m^3$)	
					Mean	Std. dev.	Mean	Std. dev.
Repace and Lowrey (*1982*)	Dinner theater	0.14	Mechanical	44	145	± 43	47	± 10
	Reception hall	1.19	Mechanical	20	301	± 30	33[1]	
	Bingo hall	0.93[2]	Natural	2	1140		40[1]	
		0.93[2]	Mechanical (1.39 ach[3])	6	443[4]		40[1]	

[1] Sequential outdoor measurement (5 minute average).

[2] Estimated.

[3] Air changes per hour.

[4] Equilibrium level as determined from concentration vs. time curve.

TABLE 2g.—Continued

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels (μg/m³)		Nonsmoking controls (μg/m³)	
					Mean	Range	Mean	Range
Cuddleback et al. (*1976*)	Tavern	Not given	6 changes/hr	4 × 8 hr continuous	310	233–346		
	Tavern	Not given	1–2 changes/hr	8 hr continuous	986			
U.S. Dept. of Transportation (*1971*)	18 military planes	165–219 people	Mechanical	72 × 6–7 hr samples		<10–120		
	8 domestic planes	27–113 people	Mechanical	24 × 1¼–2½ hr samples	Not given			
Dockery and Spengler (*1981*)	Residences	Not given	Varied	24 hr samples	32			
Elliott and Rowe (*1975*)	Arena 1	11,806 people	Mechanical	During activities	323		42 (nonactivity day)	
	Arena 2	2,000 people	Natural	During activities	620		92 (nonactivity day)	
	Arena 3 (smoking prohibited)	11,000 people	Mechanical	During activities	148		71 (nonactivity day)	
Harmsen and Effenberger (*1957*)	Trains	15–120 people	Natural	Not given		46–440 particles/cm³		
				Nonsmokers' cars				20–75 particles/cm³
Just et al. (*1972*)	4 coffee houses	Not given	Not given	6 hr averages	1150	500–1900	570 (outdoors)	100-1900
Neal et al. (*1978*)	Hospital unit	Not given	Mechanical	48 hr samples	21 ± 14	3–58	73 ± 25	
	Hospital unit	Not given	Mechanical	48 hr samples	40 ± 21	13–79	72 ± 25	

TABLE 2g.—Continued

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels (μg/m³)		Nonsmoking controls (μg/m³)	
					Mean	Range	Mean	Range
Spengler et al. (*1981*)	Residences	2+ smokers	Natural	24 hr samples	70 ± 43		21 ± 12 (outdoors)	
		1 smoker	Natural	24 hr samples	37 ± 15		21 ± 12 (outdoors)	
Weber and Fischer (*1981*)	44 offices	Varied	Natural and mechanical	429 × 2 min samples	133 ± 130[1]	962[1] (peak)		
Quant et al. (*1982*)	Office No. 1	0.62[2]	Mechanical	Five 10 hr workday averages; continuous monitoring	45	39-54		5–15
	Office No. 2	0.68[2]	Mechanical		45	37–50		15–20
	Office No. 3	1.46[2]	Mechanical		68	42–89		15–20
Brunekreef and Boleij (*1982*)	26 houses	1–3 smokers	Natural	2 mo averages	153[3]	60–340	55	20–90

[1] Values above background.

[2] Habitual smokers per 100 m².

[3] Weighted mean

TABLE 2h.—Residuals measured under realistic conditions

Study	Type of premises	Occupancy	Ventilation	Monitoring conditions	Levels		Nonsmoking controls	
					Mean	Range	Mean	Range
					Acetone (mg/m³)			
Badre et al. (*1978*)[a]	6 cafes	Varied	Not given	100 mL samples		0.91–5.88		
	Room	18 smokers	Not given	100 mL samples	0.51			
	Hospital lobby	12 to 30 smokers	Not given	100 mL samples	1.16			
	2 train compartments	2 or 3 smokers	Not given	100 mL samples		0.36–0.75		
	Car	3 smokers	Natural, open	100 mL samples	0.32			
	Car	2 smokers	Natural, closed	100 mL samples	1.20			
					Sulfates (μg/m³)			
Dockery and Spengler (*1981*)	Residences	Not given	Varied	24 hr samples	4.81			
					Sulfur dioxide (ppb)			
Fischer et al. (*1978*)	Restaurant	50–80/470 m³	Mechanical	27 × 30 min samples	20	9–32	12 ppb	
	Restaurant	60–100/440 m³	Natural	29 × 30 min samples	13	5–18	6	
	Bar	30–40/50 m³	Natural, open	28 × 30 min samples	30	13–75	8	
	Cafeteria	80–150/574 m³	11 ch/hr	24 × 30 min samples	15	1–27	12	
				Other nonsmokers' room			7	3–13
Just et al. (*1972*)	4 coffee houses	Not given	Not given	6 hr continuous	12.0–15.3			

[a] See original paper for nine other residuals.

is also readily available. CO reflects the gas phase components of smoke and thus may not reflect the levels of particulate phase constituents. There are also a number of other CO sources in addition to cigarettes, both in the external environment (e.g., automobiles) and in the indoor environment (e.g., gas stoves). As a result, even the subtraction of external atmospheric levels may not entirely eliminate the contribution of other sources of CO to the indoor environment.

Given these problems, use of several of these measures, or the tailoring of the measurement to the phenomenon being measured, seems appropriate. The measurement of total particulate matter may be a reasonable indicator of exposure to the particulate phase of smoke, once the measurement is limited to respirable particulates and once background levels with the same level of activity, but without smoke, are subtracted. Relatively precise methods have been developed to predict the levels of exposure to carbon monoxide (Jones and Fagan 1975; Coburn et al. 1965) and total particulate matter (Repace and Lowrey 1980) that would be expected in rooms of different size and ventilation with different rates of smoking. Stewart et al. (1974), using blood donors, found the median blood carboxyhemoglobin level for smokers and nonsmokers in selected populations to be 5.0 and 1.2 percent, respectively. This corresponds to a steady state ambient CO level of 7 ppm, which represents a combination of atmospheric pollution from cigarette smoke and the background level of urban pollution and is consistent with the levels described in Table 2. Exposure levels to carbon monoxide are highly dependent on ventilation, occupancy, smoking rates, and background levels in the ambient air. The half life of carboxyhemoglobin is approximately 4 hours, making blood carboxyhemoglobin a useful biologic monitor of acute exposure to passive smoking, but one that does not provide useful data for chronic exposure.

Assessment of chronic exposure with a biologic marker requires the ability to measure some accumulating product of smoke. To date, substances such as cotinine (Matsukura et al. 1979; Langone et al. 1973; Williams et al. 1979; Feyerabend and Russell 1980; Russell et al. 1982), thiocyanate (Bottoms et al. 1982; Cohen and Bartsch 1980), and polonium-210 (Radford and Hunt 1964; Little and McGendy 1966) have been measured in active smokers. Plasma and urinary nicotine, plasma and urinary cotinine, and salivary nicotine and cotinine have been reported in nonsmokers exposed to tobacco smoke (Jarvis and Russell 1984; Russell and Feyerabend 1975; Feyerbend et al. 1982). Of these measures, it would appear that urinary cotinine offers the most promise as an index of exposure. However, there are no published data using these measures as biologic markers of chronic involuntary smoke exposure.

In contrast to physiologic investigations, epidemiologic studies have used the number of smokers in the home or in the working environment as the principal exposure variable. These relatively crude indices, in general, ignore time spent with the smoker and the environmental factors known to influence ambient smoke concentration noted above.

In summary, involuntary smoking research deals with an exposure that is qualitatively and quantitatively different from that of active smoking. Adequate characterization of passive exposure in both epidemiologic and physiologic studies is substantially more difficult for involuntary exposure than for active smoking exposure. While the active smoker's total current cigarette consumption is relatively easily quantitated, the lower dose and greater influence of ventilation and ambient environment for involuntary smoke exposure makes assessment of exposure one of the most important methodologic issues of this research. Clearly, a biologic marker of chronic exposure that reflects the amount of tobacco smoke to which nonsmoking persons are exposed would be a useful tool. In addition, carefully formulated questionnaires quantifying passive smoking are also necessary, and may prove equally valid for assessing exposure. No single index has yet been accepted by all investigators, and comparison between studies remains difficult. However, Repace and Lowrey (1983) have estimated that the nonsmoking population may be exposed to from 0 to 14 mg of tar per day, with an average exposure of 1.43 mg per day.

Acute Physiologic Response of the Airway to Smoke in the Environment

Relatively little acute exposure data exist concerning the effects of passive inhalation of cigarette smoke on pulmonary function (Table 3). The data that are available have been obtained in exposure chambers under carefully monitored and controlled circumstances (Pimm et al. 1978; Shephard et al. 1979; Dahms et al. 1981).

Pimm and colleagues (1978) exposed nonsmoking adults to smoke in an exposure chamber. Relatively constant levels of carbon monoxide (approximately 24 parts per million) were achieved in the chamber during involuntary smoking. Peak blood carboxyhemoglobin levels were always less than 1 percent in subjects before smoke exposure, but were significantly greater during the study exposure. Lung volumes, flow volume curves, and heart rate were measured for all subjects. Measurements were made at rest and following exercise under control conditions and smoke-exposure conditions. Flow at 25 percent of the vital capacity decreased significantly with smoke exposure at rest in men and with exercise in women. The magnitude of the change was small: a 7 percent decrease in flow in

TABLE 3.—Acute effects on pulmonary function of passive exposure to cigarette smoke

Study	Type of exposure	Magnitude of exposure	Effects	Comments
Pimm et al. (*1978*)	Chamber 14.6 m^3 with sparse furniture; smoking machine in room	Peak [CO] ~ 24 ppm; particulates >4 mg/m^3	Men: 5% increase FRC, 11% increase RV, 4% decrease $\dot{V}_{max25}$ during exercise Women: 7% decrease $\dot{V}_{max25}$ post exercise; no effects on VC, TLC, FVC, FEV_1, $\dot{V}_{max50}$	Nonsmokers; average age of men = 22.7, women = 21.9; sham exposure as control
Shepard et al. (*1979*)	As above	Low exposure: peak [CO] ~ 20 ppm, particulates ~ mg/m^3; high exposure: [CO] ~ 31 ppm	Low exposure: 3% decrease FEV_1, 4% decrease $\dot{V}_{max50}$, 5% decrease $\dot{V}_{max25}$ with exercise; no increased effect with high exposure	Nonsmokers; average age of men = 23, women = 25; sham exposure as control; subjects estimated to have inhaled ~ 1/2 cigarette/2 hours
Dahms et al. (*1981*)	Chamber 30 m^3; climate controlled	Room levels not measured; estimated at peak [CO] ~ 20 ppm	0.9% increase in FVC, 5.2% increase in FEV_1, 2.2% increase in FEF_{25-75} at 1 hour	10 nonsmokers; age range 24–53 years; not blinded; no sham exposure

men and 14 percent in women. No other consistent changes in lung function were observed. Shepard and coworkers (1979) utilized a similar crossover design in a chamber of exactly the same size as Pimm's. Their results were almost identical, with a small (3 to 4 percent) decrease in FVC, FEV_1, $\dot{V}_{max50}$, and $\dot{V}_{max25}$. They concluded that these changes were of the magnitude anticipated from an exposure of less than 1/2 cigarette in 2 hours (the exposure anticipated for a passive smoker).

Dahms et al. (1981) used a slightly larger chamber with an estimated peak CO level of approximately 20 parts per million. They found no change in FVC, FEV_1, or FEF_{25-75} after 1 hour of exposure in normal subjects. This experiment was not blinded and had no sham exposure.

The data from these studies suggest that involuntary smoke exposure can probably produce measurable, albeit small, changes in the airways of normal individuals. This response is consistent with the acute response to the inhalation of cigarette smoke by the active smoker, and it is not surprising that high dose involuntary exposure to tobacco smoke might produce similar results. The magnitude of these changes is small, even at moderate to high exposure levels, and it is unlikely that this change in airflow per se results in symptoms; however, it may be only one manifestation of a broader irritant response to smoke in nonsmokers.

Symptomatic Responses to Chronic Passive Cigarette Smoke Exposure in Healthy Subjects

Eye irritation is the most common complaint experienced by normal people acutely exposed to cigarette smoke. In one study, 69 percent of subjects reported ever experiencing this symptom (Speer 1968). Headache, nasal irritation, and cough were reported by approximately one-third of the subjects in this and other investigations (Weber and Hertz 1976; Slavin and Hertz 1975). Several factors may alter the prevalence of irritant symptoms, including the amount of smoking, the size of the area involved, the humidity and temperature of ambient air, and the extent of ventilation (Johansson 1976). No longitudinal studies of these irritant effects (e.g., development of increased sensitivity or tolerance) have been reported.

Weber (1984) has examined the effect of dose and duration of exposure to environmental tobacco smoke on subjective reporting of eye irritation and objective measurement of eye blink rate. Figure 1 reveals that both eye irritation and blink rate increase with increasing dose of smoke exposure, and that substantial subjective irritation and objective increase in blink rate occur at levels of smoke exposure (CO levels of 20 to 24 ppm) equivalent to those used to evaluate pulmonary function changes in response to environmen-

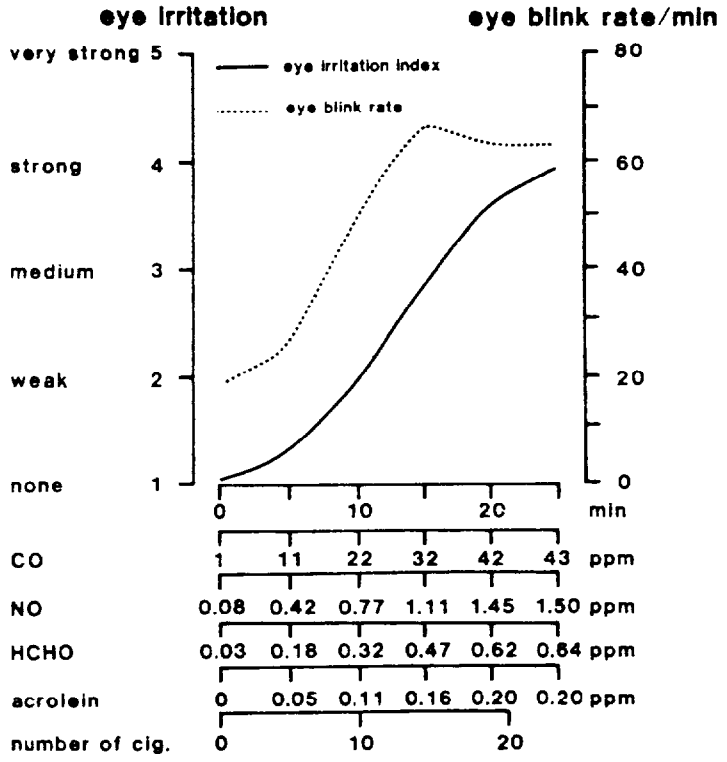

FIGURE 1.—Mean subjective eye irritation, mean eye blink rate, and concentrations of some pollutants during continuous smoke production in an unventilated climatic chamber

NOTE: Thirty-three subjects; ventilation rate 0.01 h^{-1}; eye irritation index calculated from the answers to four questions concerning eye irritation; 0 min = measurement before smoke production.

SOURCE: Weber (1984).

tal tobacco smoke exposure. Both irritation and blink rate increase with duration of exposure to environmental tobacco smoke (Figures 2 and 3). After 60 minutes of exposure, distinct changes are evident in level of irritation with a smoke exposure of 1.3 ppm CO, and the blink rate increased with smoke exposures as low as 2.5 ppm CO. These levels of smoke exposure (1.3 to 2.5 ppm CO) are well within those measured under realistic conditions (see Table 1). Therefore, it is possible to demonstrate an objective irritant response in normal subjects at levels of smoke exposure substantially lower than the levels where an airway response (also presumably an irritant response) has been demonstrated. Whether this difference represents a difference in threshold for irritation in the eye and airway or a limitation in the ability to measure subtle changes in the airway is uncertain.

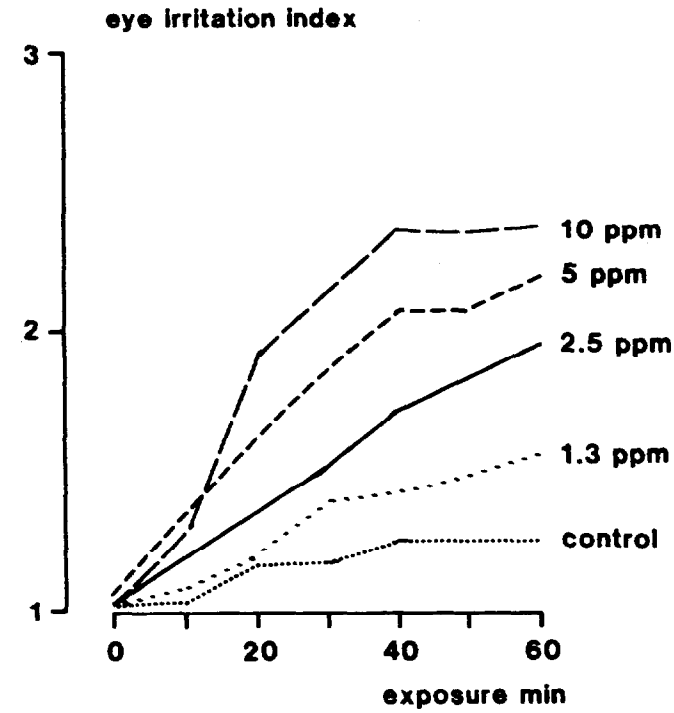

FIGURE 2.—Subjective eye irritation due to environmenta tobacco smoke, related to smoke concentratio and duration of exposure

NOTE: CO values are levels during smoke production minus background level before smoke production; 32 t subjects; 0 min = measurements before smoke production.
SOURCE: Weber (1984).

Chronic respiratory symptoms have been reported most common in children. Studies from several different countries (Table 4) ha shown a positive relationship between parental cigarette smoki and the reporting of the symptoms of chronic cough, chronic phleg and persistent wheeze (Colley et al. 1974; Bland et al. 1978; Lebow and Burrows 1976; Weiss et al. 1980; Ware et al. 1984; Schilling et 1977; Kasuga et al. 1979; Schenker et al. 1983). Some of these studi may be confounded by an increased reporting of symptoms in t child by parents who smoke and have symptoms (Colley et al. 19 Bland et al. 1978; Kasuga et al. 1979) or by the child's own smoki habits (Colley et al. 1974; Bland et al. 1978; Kasuga et al. 1979). N all studies show statistical significance for all symptoms (Lebow and Burrows 1976; Schilling et al. 1977; Schenker et al. 198 However, a consistent finding in all reported data is an increase symptoms with an increased number of smoking parents in t

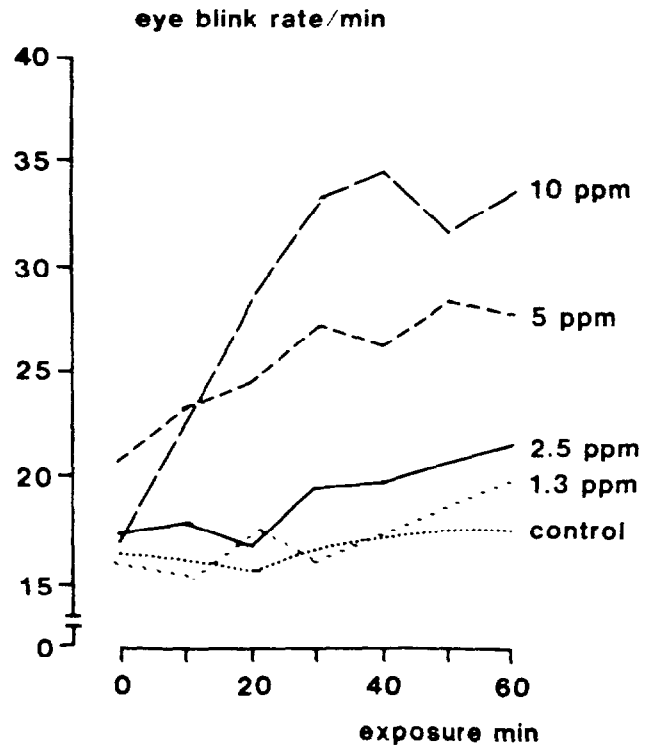

FIGURE 3.—Effects of environmental tobacco smoke on eye blink rate

NOTE: CO values are levels during smoke production minus background level before smoke production; 32 to 43 subjects; 0 min = measurements before smoke production.

SOURCE: Weber (1984).

home. This effect persists after controlling for parental cough and is most marked in the first year of life.

British researchers, studying a birth cohort, demonstrated an increased incidence of wheezing over a 5-year period among nonasthmatic children who had two parents who smoked. However, when examined by logistic regression, parental smoking was not a significant predictor of occurrence of wheeze or the future occurrence of asthma (Bland et al. 1978). In a subgroup of the cohort—861 children of asymptomatic parents, Leeder and colleagues (1976a) found no significant trend in asthma-wheeze symptoms with increasing levels of parental smoking over a 5-year period. In a study of 650 children aged 5 to 10 years (Weiss et al. 1980), a significant trend in the reported prevalence of chronic wheezing with current parental smoking was found; the rates were 1.85 percent, 6.85 percent, and 11.8 percent for zero, one smoking parent, and two smoking parents, respectively. Although the data given are for all

TABLE 4.—Respiratory symptoms in children in relation to involuntary smoke exposure

Study	Subjects	Respiratory symptoms or illness	Rates per 100 by number of smoking parents 0	1	2	Comment
Colley et al. (*1974*)	2,426 children, aged 6–14, England	Chronic cough assessed by questionnaire completed by parent	15.6	17.7	22.2	Trend significant; possible that symptoms in parents could result in reporting bias; active smoking in children could also bias results; bias unlikely to explain full effect of trend
Bland et al. (*1978*)	3,105 children, aged 12–13, who did not admit to ever smoking cigarettes, England	Cough during day or at night	16.4	19.0	23.5	Self-reported symptoms and smoking history collected simultaneously from children; difference between morning and daytime cough suggested as different diseases, but could be difference in exposure, in that exposure more likely in daytime than when asleep
		Morning cough	1.5	2.8	2.9	
Weiss et al. (*1980*)	650 children, aged 5–9, United States	Chronic cough and phlegm	1.7	2.7	3.4	Trend not significant
		Persistent wheeze	1.8	6.8	11.8	Trend significant
Ware et al. (*1984*)	8,528 children, aged 5–9, with two parents of known smoking status, six U.S. cities	Chronic cough	7.7	8.4	10.6	Adjusted for age, sex, and city cohort effects; significant trends
		Persistent wheeze	9.9	11.0	13.1	

TABLE 4.—Continued

Study	Subjects	Respiratory symptoms or illness	Rates per 100 by number of smoking parents 0	1	2	Comment
Dodge (*1982*)	628 children, grades 3–4, in two-parent households; questionnaire response of parents, United States	Wheeze	27.6	27.9	40.0	All trends significant; some of effect might relate to parental symptoms, but not likely to influence trends
		Phlegm	6.4	10.9	12.0	
		Cough	14.6	23.0	27.8	
Schenker et al. (*1983*)	4,071 children, aged 5–14, in western Pennsylvania	Chronic cough	6.3	7.0	8.3	None of these rates significant; data not adjusted for parental symptoms
		Chronic phlegm	4.1	4.8	4.0	
		Persistent wheeze	7.2	7.7	5.4	
			Never smoking		Parent smoking	
Lebowitz and Burrows (*1976*)	1,252 children, <15 years old, United States	Persistent cough	3.7		7.2	Higher rates in symptomatic households with trends persisting, but not significant for asymptomatic households
		Persistent phlegm	10		12.8	
		Wheeze	23.4		24.1	
Schilling et al. (*1977*)	816 children, age 7+, United States	Cough, phlegm, wheeze	No significant effect			Specific data not provided
Kasuga et al. (*1979*)	1,937 children, aged 6–11, Japan	Wheeze, asthma	Increased prevalence in families with a heavy smoker (≥21 cig/day); less clear effect in family with a light smoker (<21 cig/day)			Data adjusted for distance of home from main traffic, highway

households, when the analysis was restricted to those households where neither parent reported symptoms, the results were identical, suggesting that in this population, significant reporting bias was not responsible for the observed results. Lebowitz and Burrows (1976), in a group of 463 current-smoking and never-smoking households with children below age 15, found trends—but no statistically significant differences—for a variety of symptoms, including wheeze most days, in households with smokers. In the same study, among 849 households with older children and adults, there were no significant differences for any symptom prevalence between current-smoking and never-smoking household members. In a general population study, Schilling et al. (1977) reported no association between wheeze and involuntary smoking.

A preliminary report from one of the largest studies currently under way (Speizer et al. 1980) indicated no association of persistent wheeze with the presence of smoking in the household for approximately 8,000 children aged 6 to 11 in six communities. However, subsequent analyses of these same cohorts with the addition of approximately 2,000 more children and a more detailed assessment of the smoking behavior of each parent revealed a positive relationship that increased with the amount of maternal smoking and was only modestly affected by taking into account the parents' own symptoms (Ware et al. 1984). Dodge (1982), studying third and fourth grade children, found that symptoms, including wheeze, were related to both the presence of symptoms in the parents and the number of smokers in the household. The gradient of the wheeze effect persisted even after excluding the potential effect of reporting bias by symptomatic parents. Few data are available on the level of exposure necessary to produce symptoms or on the implication of these symptoms for future lung growth and development. No data are currently available on the relationship of passive smoking to other putative risk factors for wheezing such as atopy, respiratory infection, and increased levels of airways responsiveness, nor are sufficient data available to estimate whether these early exposures affect the occurrence of respiratory disease later in life. The characteristics of the child who may be susceptible to this type of exposure are unknown. However, the data are sufficiently consistent to suggest that pediatricians should routinely inquire about smoking habits of parents when caring for children with chronic or recurrent respiratory symptoms and illnesses. It would also be prudent to advise parents of children who are suffering from recurrent respiratory illnesses or persistent wheeze or asthma not to smoke.

Respiratory Infections in Children of Smoking Parents

Bronchitis and pneumonia and other lower respiratory illnesses are significantly more common in the first year of life in children who have one or two smoking parents (Table 5). Bonham and Wilson (1981) showed that in 1970 the majority of homes with children under 17 years of age had at least one smoker. Thus, passive smoking by children, even in early childhood, is widespread. Harlap and Davies (1974) studied 10,672 births in Israel between 1965 and 1968 and observed that infants whose mothers said they smoked (as determined at a prenatal visit) experienced a 27.5 percent greater hospital admission rate for pneumonia and bronchitis than children of nonsmoking mothers. In addition, they demonstrated a dose–response relationship between the amount of maternal smoking and the number of hospital admissions for these conditions. It should be noted that the mothers were reporting prenatal smoking and not postnatal smoking for the first year of life.

British investigators studying live births between 1963 and 1965 in London also observed an increased frequency of bronchitis and pneumonia in the first year of life associated with involuntary smoking that did not carry over to years 2 to 5 (Colley et al. 1974). This effect was independent of parents' own symptoms and increased with the amount of smoking by parents. Bronchitis and pneumonia also increased with an increased number of siblings, and this was not controlled in the analysis.

Fergusson et al. (1981), studied 1,265 New Zealand children from birth to age 3. They demonstrated an increase in both bronchitis and pneumonia and lower respiratory illness during the first 2 years of life in children whose mothers smoked. Corrections for maternal age, family size, and socioeconomic status did not affect the linear relationship between the degree of maternal smoking and the rate of respiratory illness. This effect declined with the increasing age of the child.

Leeder and colleagues (1976b) studied a British cohort of children born between 1963 and 1965 and demonstrated that parental cigarette smoking was associated significantly with bronchitis and pneumonia during the first year of life. A dose–response association persisted after correction for parental respiratory symptoms, sex of the child, number of siblings, and a history of respiratory illness in the siblings.

Pullan and Hey (1982) studied children who were hospitalized with documented respiratory syncytial virus (RSV) infection in infancy. They found a significant difference in the smoking habits of mothers at the time of the infection, compared with children hospitalized for other illnesses—including respiratory diseases for which RSV infection was not documented. These children reported an excess occurrence of wheeze and asthma and had lower levels of pulmonary

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TABLE 5.—Early childhood respiratory illness and involuntary cigarette smoking

Study	Subjects	Findings	Illness rates per 100				Comments
			By cigarettes per day				
			0	1-10	11-20	20+	
Harlap and Davies (*1974*)	10,672 births, 1965-1968, West Jerusalem, Israel	Hospitalized for bronchitis/pneumonia in first year of life RR[1]=1.38	9.5	10.8	16.2	31.7	Smoking history obtained antenatally; maternal smoking only
Colley[2] (*1974*)	2,205 births, 1963-1965, London, England	Questionnaire on bronchitis/pneumonia in first year of life RR=1.73 for one parent smoker RR=2.60 for two parent smokers	7.6 10.3	10.4 15.1	11.1 14.5	15.2 23.2	= Asymptomatic parents = Symptomatic parents Neither controlled for number of siblings or sex of smokers
Fergusson et al. (*1981*)	1,265 births, 4 months, 1977, Christchurch, New Zealand	Questionnaires on doctor or hospital visits for bronchitis/pneumonia; check by hospital records Assessment at 4 months, 1, 2, and 3 years RR=2.04 if mother smoked	7.0 7.0	12.8 4.6	13.4 8.8	Maternal only Paternal only	Combined effect significant for maternal smoking in first year of life only
			By number of smoking parents				
			0	1	2		
Ware et al. (*1984*)	8,528 children, aged 5-9, with two parents of known smoking status, six U.S. cities	Respiratory illness in last year	12.9	13.7	14.8		Adjusted for age, sex, and city cohort effect; significant trends

TABLE 5.—Continued

Study	Subjects	Findings	Illness rates per 100			Comments
Said et al. (*1978*)	3,920 children, aged 10–20, France	Tonsillectomy and/or adenoidectomy, generally before age 5, as indicator of frequent respiratory tract infection	28.2	41.4	50.9	Self-reporting by children; not clear that smoking habits of parents at time of reporting directly related to exposure approximately 10+ years earlier
Schenker et al. (*1983*)	4,071 children, aged 5–14, western Pennsylvania	Chest illness before age 2 Chest illness >3 days in past year	6.7 8.8	7.9 11.8	11.5 13.6	Trends for both significant
Cameron et al. (*1969*)	158 children, aged 6–9; parents completed telephone questionnaire, United States	Respiratory illness with restricted activity and/or medical consultation in last year	1.33		7.4	Illness reporting not verified; not clear how reporting adult was related to child
Leeder et al. (1976a, b)	2,149 infants, born 1963–1965, Harrow, England	RR ~ 2.0 for infants with two smoking parents	Not provided			Parents answered for children, but response bias seems unlikely because effects were observed for infants of asymptomatic parents; effects of maternal vs. paternal smoking not investigated
Sims et al. (*1978*)	35 children hospitalized with RSV bronchiolitis, 35 controls, England	Borderline significant increase in maternal smoking during first year of life RR=2.65	Not provided			No significant effect for paternal smoking; average amount smoked greater for parents of cases than for controls

TABLE 5.—Continued

Study	Subjects	Findings	Illness rates per 100	Comments
Rantakallio (*1978*)	1,821 children of smoking mothers, 1,823 children of nonsmoking mothers	Significant increase in hospitalization for respiratory illness during first 5 years of life RR = 1.74	Not provided	Prospective followup of doctor visits, hospitalizations, deaths up to age 5; only maternal smoking evaluated
Pullan and Hey (*1982*)	130 children admitted to hospital during first year of life with RSV infection, 111 nonhospitalized controls	Significant effect of maternal (RR = 1.96) and paternal (RR = 1.53) smoking at time of study; significant maternal effect of smoking during first year of life (RR = 1.55)	Not provided	

[1] Relative risk for children of smoking mothers versus children of nonsmoking mothers calculated from published data provided by J.M. Samet, M.D.

[2] These data are considered in a more expanded analysis provided by Leeder et al. (*1976*).

function that persisted to age 10. The authors could not distinguish between the possibilities that infection caused damage that persisted and affected the maturation of the lung or that these children were already more susceptible to severe RSV infection. Greenberg et al. (1984) examined the tobacco smoke exposure of infants in the first year of life by measuring urinary cotinine-to-creatinine ratios. They found that infants of mothers who smoked had a ratio of 351 ng per mg, as contrasted with a ratio of 4 ng per mg in infants of mothers who did not smoke. Breast-fed infants were excluded because of the presence of nicotine in the breast milk of mothers who smoke. A dose–response relationship was present between the cotinine-to-creatinine ratio and the reported level of maternal smoking in the previous 24 hours. This study suggests that infants of mothers who smoke absorb measurable amounts of the smoke from this environmental exposure.

Rantakallio (1978) studied over 3,600 children for 5 years, half of whom had mothers who smoked and half of whom did not. Children of mothers who smoked had a 70 percent greater chance of being hospitalized for a respiratory illness than children of nonsmoking mothers.

Some of these studies may be confounded by the increased reporting of symptoms in the child by parents who smoke and have symptoms (Cameron et al. 1969; Said et al. 1978; Leeder et al. 1976b), but in those studies in which parental symptoms were controlled, the effects persisted. Other studies may be influenced by the child's own smoking habits (Said et al. 1978), although the majority of research examined children in an age range in which smoking would be unlikely.

In summary, several studies suggest important increases in severe respiratory illnesses, particularly in the very young (less than 2 years old) children of smoking parents. Young children may represent a more susceptible population for adverse effects of involuntary smoking than older children and adults. The amount of time spent with active smokers, particularly by children under 2 years of age with smoking mothers, may be an important factor. How in utero exposure influences this risk is unknown.

Pulmonary Function in Children of Smoking Parents

In recent years, a number of studies have examined the relationship of parental cigarette smoking to pulmonary function in children (Table 6). The majority of these studies have been cross sectional (Tager et al. 1979; Weiss et al. 1980; Vedal et al., in press; Burchfiel et al., 1983; Tashkin et al. 1983; Hasselblad et al. 1981; Ware et al. 1984) and have demonstrated decreases in level of pulmonary function ($FEV_{0.75}$, FEV_1, FEF_{25-75}, and flows at low lung volumes) in

children of smoking mothers compared with children of nonsmoking mothers.

In some studies, there seems to be a dose–response relationship (Tager et al. 1979; Weiss et al. 1980); i.e., the greater the number of smokers in the home, the lower the level of function. When analyzed by multiple regression techniques, maternal smoking has the greatest impact (as would be expected from the greater contact time with the child), and a dose–response relationship with the amount smoked seems to exist (Weiss et al. 1980; Tager et al. 1979; Ware et al. 1984; Vedal et al., in press). Younger children seem to be more adversely affected than older children (Tager et al. 1979; Weiss et al. 1980), and clearly there is an added effect in older children if they themselves smoke (Tager et al. 1979).

Tager and colleagues (1983) followed 1,156 children for 7 years to determine the effect of maternal smoking on growth of pulmonary function in children. After correcting for previous level of FEV_1, age, height, personal cigarette smoking, and correlation between mother's and child's pulmonary function, maternal smoking was associated with a reduced rate of annual increase in FEV_1 and FEF_{25-75}. The magnitude of the effect was consistent with a 3 to 5 percent decrease in expected lung growth due to the maternal smoking effect, constant over the time period of the study. Because so few mothers changed their smoking habits, the study did not attempt to differentiate between postnatal and in utero effects of involuntary smoke exposure.

Ware et al. (1984) followed 10,106 white children for two successive annual examinations. The FEV_1 was 0.6 percent lower in the children of smoking mothers at the first examination and 0.9 percent lower at the second examination. These differences were statistically significant, but represent very small absolute differences. In this study, and in the other studies that show small changes in pulmonary function, it is not clear whether these changes represent small changes occurring uniformly among the children of smoking mothers or somewhat larger changes occurring in a small subpopulation of susceptible children.

The available data demonstrate that maternal smoking affects lung function in young children. However, the absolute magnitude of the difference in lung function is small; it is unlikely that this small difference, per se, is of functional significance. The concern generated by the demonstration of even small differences is directed at the future lung function of those children, particularly if they become active cigarette smokers as adults. The possibility that this difference in lung function may result from pathophysiologic mechanisms similar to those present in active smokers raises the concern that these children may be "sensitized" to smoke at an early age, and that this "sensitization" may result in a more rapid decline in lung

TABLE 6.—Pulmonary function in children exposed to involuntary smoking

Study	Subjects	Pulmonary function measure	Outcome	Comments
Schilling et al. (*1977*)	816 children, aged 7–17, Connecticut and South Carolina	FEV_1 as percent predicted	No effect of parental smoking	No control for sibship size or correlation of siblings' pulmonary function; when analysis restricted to children who never smoked, $\dot{V}_{max50}$ significantly less in children with smoking mothers
Tager et al. (*1979*)	444 children, aged 5–19, East Boston, Massachusetts	MMEF in standard deviation units	Significant effect of parental smoking	Analysis controlled for sibship size and correlation of siblings' pulmonary function
Weiss et al. (*1980*)	650 children, aged 5–9, East Boston, Massachusetts	MMEF in standard deviation units	Significant effect of parental smoking	Analysis controlled for sibship size and correlation of siblings' pulmonary function
Vedal et al. (in press)	4,000 children, aged 6–13	FEV_{75}, FVC, $\dot{V}_{max50}$, $\dot{V}_{max75}$, $\dot{V}_{max90}$	FVC positively associated, flows negatively associated	Flows dose–response with amount smoked by mother
Lebowitz and Burrows (*1976*)	271 households with complete histories of parents' smoking and of pulmonary function of children $\geq$ age 6, Tucson, Arizona	FEV_1, FVC, $\dot{V}_{max50}$, $\dot{V}_{max75}$ derived from MEF_1 $\dot{V}$ curves, expressed as standard deviation units	No effect of parental smoking	Suggestion that real differences in indoor levels of exposure compared with more northerly climates may be occurring

TABLE 6.—Continued

Study	Subjects	Pulmonary function measure	Outcome	Comments
Dodge (*1982*)	558 children, aged 8–10, Arizona	FEV_1 by age change FEV_1/H^3 per year	No effect of parental smoking	Potential bias in participation rates; cross-sectional data not controlled for children's height; annual change in FEV_1/H^3 at ages 8, 9, and 11 consistently greater in nonsmoking households than in two-parent smoking households; statistical test not significant, however
Tager et al. (*1983*)	1,156 children, aged 5–19 at initial survey, East Boston, Massachusetts	FEV_1, FEF_{25-75}	Significant decreased rate of growth in FEV_1 and FEF_{25-75} for children of smoking mothers	7-year followup; no effect of paternal smoking; maximum effect of maternal smoking on fully developed lung not more than 4 or 5 percent
Burchfiel et al. (*1983*)	4,378 children, aged 0–19, Tecumseh, Michigan	FVC, FEV_1, $\dot{V}_{max50}$	Decreased FEV_1 and FVC for boys and $\dot{V}_{max50}$ for girls with increased number of smoking parents	Abstract; no distinction between effects of maternal and paternal smoking; effects most prominent for boys and youngest age groups
Tashkin et al. (*1983*)	1,070 nonsmoking, nonasthmatic children, Los Angeles	$\dot{V}_{max}$, $\dot{V}_{max75}$, $\dot{V}_{max25}$, FEF_{25-75}	Decreased $\dot{V}_{max}$, $\dot{V}_{max25}$ for boys and FEF_{25-75}, $\dot{V}_{max75}$ for girls with at least a smoking mother	No effect of paternal smoking
Hasselblad et al. (*1981*)	16,689 children, aged 5–17, seven geographic regions, United States	FEV_{75} as percent predicted	Significant effect of maternal smoking, but not paternal smoking	Large number of children excluded because of invalid pulmonary function data or missing parental smoking data