TABLE 4.—Number of women in the current workforce, classified by occupation (1978)

Occupation	# of Women in Thousands
White-collar workers	24,594
Professional & Technical	6,083
Biological scientists	22
Chemists	17
Nurses, dieticians, & therapists	1,255
Health technologists and technicians	353
Engineering and science technicians	132
Painters and sculptors	83
Photographers	13
Managers and administrators, except farm	2,365
Sales workers	2,666
Sales clerks, retail trade	1,672
Clerical workers	13,456
Bookkeepers	1,660
Cashiers	1,222
Secretaries	3,561
Typists	1,009
Blue-collar workers	5,770
Craft and kindred workers	694
Printing craft workers	91
Upholsterers	14
Operatives, except transport	4,317
Assemblers	606
Bottling and canning operatives	25
Checkers, examiners, and inspectors; manufacturing	359
Clothing ironers and pressers	101
Cutting operative, n.e.c.	84
Dressmakers, except factory	113
Drillers, earth	2
Dry wall installers and lathers	1
Filers, polishers, sanders and buffers	38
Furnace tenders, smelters, and pourers, metal	3
Garage workers, and gas station attendants	20
Blue-collar workers—cont'd	
Laundry and dry cleaning operatives, n.e.c.	118
Meat cutters and butchers, except manufacturing	13
Meat cutters and butchers, manufacturing	33
Mine operatives, n.e.c.	4
Mixing operatives	3
Packing and wrappers, excluding meat and produce	422
Painters, manufactured articles	30
Photographic process workers	48
Precision machine operatives	43
Drill press operatives	15
Grinding machine operatives	10
Lathe and milling machine operatives	11
Punch and stamping press operatives	47
Sawyer	14
Sewers and stitchers	772
Shoemaking machine operatives	60
Furnace tenders and stokers, except metal	1
Textile operatives	224
Spinners, twisters, and winders	100
Welders and flame cutters	41
Winding operatives, n.e.c.	37
All other operatives, except transport	1,062
Transport equipment operatives	258
Nonfarm laborers	492
Service workers	8,037
Private households	1,135
Child care workers	477
Cleaners and servants	514
Housekeepers	117
Service workers, except households	6,901
Cleaning workers	858

Table 4 (continued)

Occupation	# of Women in Thousands	Occupation	# of Women in Thousands
Service workers—cont'd.		Health service workers—cont'd.	
Lodging quarters cleaners	174	Practical nurses	390
Building interior cleaners, n.e.c.	462	Personal service workers	1,302
Janitors and sextons	222	Attendants	175
Food service workers	2,951	Barbers	11
Bartenders	111	Child care workers	103
Waiters' assistants	45	Hairdresser and cosmetologists	483
Cooks	678	Housekeepers, excluding private households	—
Dishwashers	82	Welfare service aides	92
Food counter and fountain workers	397	Protective service workers	115
Waiters	1,252	Firefighters	1
Food service workers, n.e.c.	384	Guards	53
Health service workers	1,660	Police and detectives	28
Dental assistants	128	Sheriffs and bailiffs	3
Health aides, excluding nursing	238	Farm workers	509
Nursing aides, orderlies, and attendants	902	TOTAL—	38,910

NOTE: n.e.c. is an abbreviation for "not elsewhere classified" and designates broad categories of occupations that cannot be more specifically identified.
SOURCE: U.S. Department of Labor (17).

impact of occupational exposures (or their interactions with smoking) on the health of women.

Because of this cohort effect, any failure to demonstrate an excess risk of a given occupational exposure in women must be interpreted with considerable caution. It may mean only that the women exposed were too young and the exposure too brief for illness to have yet developed. This caution is doubly important for those attempting to demonstrate an interaction between occupational exposure and smoking on the development of disease in women. Thus, little comfort can be taken from the current low prevalence of occupational disease in women. It is reasonable to expect that any movement of large numbers of women into hazardous occupations will be followed, after an appropriate time lag, by a dramatic increase in the prevalence of occupational illness in women.

The Reproductive Role

A third reason for examining the effects of occupational exposures in women separately from those in men is the difference

TABLE 5.—Most common female job categories, by percentage of the female work force employed

Job	Percent of Female Work Force	Job	Percent of Female Work Force
Secretary	8.5	Private Household Worker	2.9
Retail Sales Clerk	4.3	Registered Nurse	2.8
Bookkeeper	4.3	Elementary School Teacher	2.8
Waitress	3.2	Typist	2.6
		Cleaning Workers	2.2
Cashier	3.1	Sewer & Stitcher	2.0

SOURCE: Rones, P. (14).

in their reproductive roles. Toxic occupational exposures in both men and women can reduce fertility and increase frequency of teratogenic effects (see Table 6). In addition, however, the 9-month duration of gestation provides many opportunities for the fetus to share any adverse toxic exposure of its mother. These risks may interact with the well-established risks of cigarette smoking during pregnancy discussed elsewhere in this report. Table 6 provides a list of hazardous substances in the work environment, some of which are suspected of having effects on reproduction.

Another specific concern for women is that of contraception. Substantial numbers of women in the United States use oral contraceptives (18). These drugs have been shown to interact with cigarette smoking to produce a greatly increased risk of cardiovascular disease, as discussed in this report. In addition, it is possible that oral contraceptives may interact in an adverse manner with physical or chemical agents found in the work place, or that the combination of smoking, occupational exposure, and oral contraceptive use may bear special risks. The answers to those questions can be found only through the study of populations of working women.

One study approached this issue by examining the health status of women involved in the manufacture of oral contraceptives. Poller, et al. have shown that women working in the manufacture of oral contraceptives absorb enough of the drugs to influence the clotting mechanism as well as alter menstrual function (12). Unfortunately, the risk of cardiovascular disease—and the effects of smoking in relation to it—could not be estimated in this population. Because of the established excess risk of cardiovascular disease from concurrent smoking

and oral contraceptive use, examination of cardiovascular risk in this group would be of interest.

The preceding discussion presents several areas where female–male differences may significantly limit the direct applicability of the results of male smoking studies to the female population. These areas of potential difference present research questions that justify significant, ongoing research activities.

Specific Interactions Between Occupational Exposure and Smoking

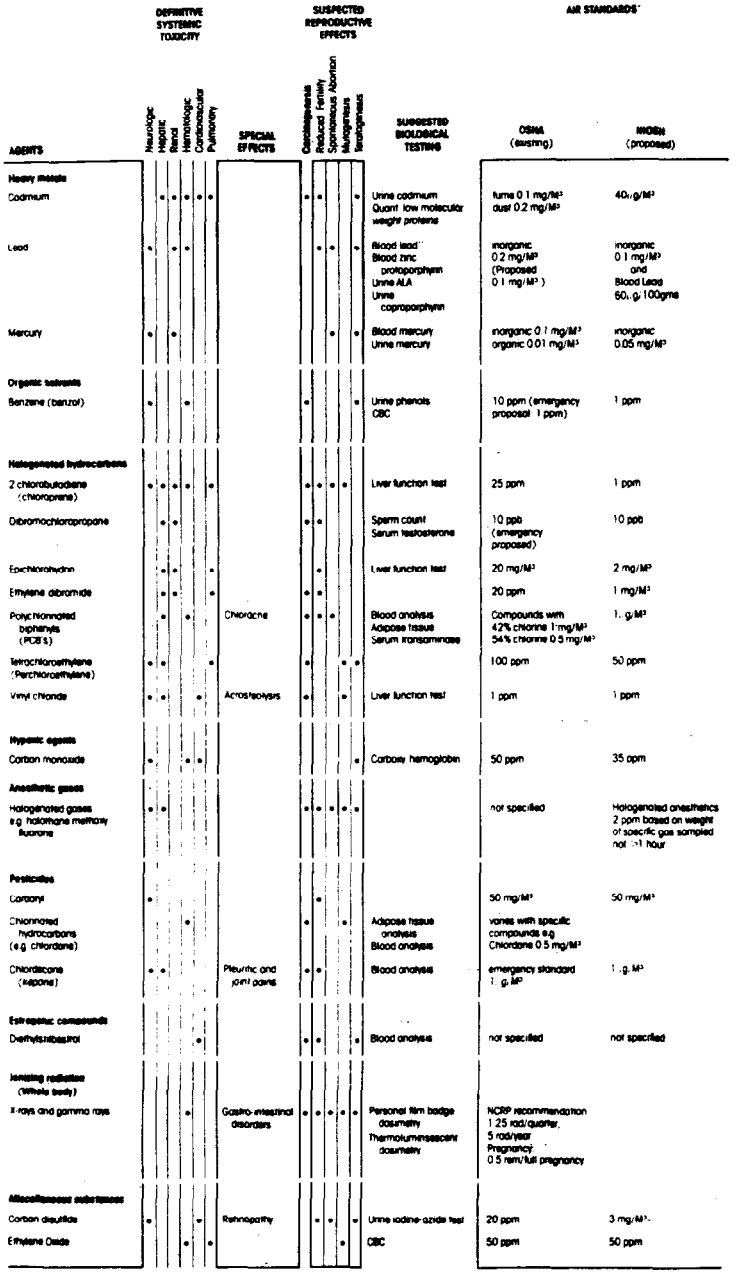
A review of all the potential risks of occupational exposure for women is beyond the scope of this section. Table 6 lists a number of agents found in the occupational environment and their observed organ toxicity. Table 7 presents selected pulmonary irritants and sensitizers in specific occupational settings in relation to the number of women employed in those settings.

There is little specific data on the health effects of a given occupational exposure in women. Two clear exceptions exist—exposure to asbestos and to cotton dust. The data from studies of women exposed to these two compounds provide examples of established interactions between smoking and occupational exposure in women.

ASBESTOS

Selikoff, et al. prospectively followed a group of 370 male asbestos insulation workers. They demonstrated a multiplicative effect of asbestos exposure and cigarette smoking on the risk of development of lung cancer (4,13). Workers who smoked cigarettes developed lung cancer at a rate 92 times that of non-exposed nonsmokers. They observed no deaths from lung cancer among 87 nonsmokers, and 24 deaths from bronchogenic cancer among 283 regular smokers, a number well in excess of the 3 deaths expected. Newhouse, et al. followed a cohort of 900 women first employed between 1936 and 1942 in an asbestos factory making both textiles and insulation materials (2,10,11). They analyzed the group's mortality experience between first employment and 1968, with a minimum of 26 years' follow-up. There was an excess overall mortality partly accounted for by deaths from cancer, observed even among those who worked in jobs with low-to-moderate exposure to asbestos. An excess of cancer of the lung and pleura was found among those who were severely exposed and who had worked less than 2 years. In the group with severe exposure for more than 2 years in the factory, excess deaths from cancer of the lung, pleura, and non-

TABLE 6.—Chart of toxins and effects



AGENTS	DEFINITIVE SYSTEMIC TOXICITY: Neurologic	Hepatic	Renal	Hematologic	Cardiovascular	Pulmonary	SPECIAL EFFECTS	SUSPECTED REPRODUCTIVE EFFECTS: Osteosclerosis	Reduced Fertility	Spontaneous Abortion	Mutagenesis	Teratogenesis	SUGGESTED BIOLOGICAL TESTING	AIR STANDARDS: OSHA (ceiling)	NIOSH (proposed)
Heavy metals															
Cadmium	•		•		•	•		•	•			•	Urine cadmium Quant low molecular weight proteins	fume 0.1 mg/M³ dust 0.2 mg/M³	40 μg/M³
Lead	•			•	•				•	•		•	Blood lead* Blood zinc protoporphyrin Urine ALA Urine coproporphyrin	inorganic 0.2 mg/M³ (Proposed 0.1 mg/M³)	inorganic 0.1 mg/M³ and Blood Lead 60 μg/100gms
Mercury	•			•						•		•	Blood mercury Urine mercury	inorganic 0.1 mg/M³ organic 0.01 mg/M³	inorganic 0.05 mg/M³
Organic solvents															
Benzene (benzol)	•				•			•				•	Urine phenols CBC	10 ppm (emergency proposal 1 ppm)	1 ppm
Halogenated hydrocarbons															
2 chlorobutadiene (chloroprene)	•	•	•	•	•	•		•	•	•	•	•	Liver function test	25 ppm	1 ppm
Dibromochloropropane		•	•					•	•				Sperm count Serum testosterone	10 ppb (emergency proposed)	10 ppb
Epichlorohydrin		•	•			•			•				Liver function test	20 mg/M³	2 mg/M³
Ethylene dibromide		•	•			•		•	•					20 ppm	1 mg/M³
Polychlorinated biphenyls (PCB's)		•			•		Chloracne	•	•	•			Blood analysis Adipose tissue Serum transaminase	Compounds vary 42% chlorine 1 mg/M³ 54% chlorine 0.5 mg/M³	1. g/M³
Tetrachloroethylene (Perchloroethylene)	•	•				•		•			•	•		100 ppm	50 ppm
Vinyl chloride	•	•			•		Acroosteolysis	•			•		Liver function test	1 ppm	1 ppm
Hypoxic agents															
Carbon monoxide	•				•	•						•	Carboxy hemoglobin	50 ppm	35 ppm
Anesthetic gases															
Halogenated gases e.g. halothane methoxy fluorane	•	•						•	•	•	•	•		not specified	Halogenated anesthetics 2 ppm based on weight of specific gas sampled not >1 hour
Pesticides															
Carbaryl	•								•					50 mg/M³	50 mg/M³
Chlorinated hydrocarbons (e.g. chlordane)					•			•				•	Adipose tissue analysis Blood analysis	varies with specific compounds e.g. Chlordane 0.5 mg/M³	
Chlordecone (kepone)	•	•					Pleuritic and joint pains	•	•				Blood analysis	emergency standard 1. g/M³	1. g/M³
Estrogenic compounds															
Diethylstilbestrol						•		•	•			•	Blood analysis	not specified	not specified
Ionizing radiation (Whole body)															
X-rays and gamma rays					•		Gastro-intestinal disorders	•	•	•	•	•	Personal film badge dosimetry Thermoluminescent dosimetry	NCRP recommendation 1.25 rad/quarter, 5 rad/year Pregnancy 0.5 rem/full pregnancy	
Miscellaneous substances															
Carbon disulfide	•					•	Retinopathy	•	•			•	Urine iodine-azide test	20 ppm	3 mg/M³·
Ethylene Oxide					•	•					•		CBC	50 ppm	50 ppm

- Animal and/or human data
- Time weighted average 40-hr. week, 8-hr. day (OSHA), 10-hr. day (NIOSH).
- In pregnancy, blood lead less than 40μ g/100 gms is suggested
- Evidence only male infertility; no data on females

SOURCE: National Institute for Occupational Safety and Health (9).

neoplastic respiratory disease were observed. The authors calculated the excess annual mortality due to lung cancer. When workers with low-to-moderate exposure experienced a mean excess lung cancer mortality of 63 deaths (per 100,000 years' exposure). Those severely exposed for less than 2 years experienced an excess of 44 deaths, and those severely exposed for 2 years or longer experienced an excess of 241 deaths. Interestingly, an examination of deaths did not reveal any significant association with age at first employment in the asbestos factory. In the sub-sample of workers whose smoking histories were available, those women who had both smoked and were heavily exposed had a risk of developing lung cancer over 30 times that of nonexposed nonsmoking women. The authors concluded that the data suggested that asbestos and cigarette smoking exert multiplicative rather than merely additive effects.

In summary, the data on smoking and asbestos exposure in women closely resemble the findings demonstrated for men.

COTTON DUST

Approximately 250,000 women were employed in the textile industry in 1978; that population included approximately 100,000 women engaged in spinning, twisting, and winding operations. Byssinosis is a syndrome characterized by tightness of the chest and shortness of breath in workers exposed to dust of cotton, flax, and hemp. In addition to these acute symptoms, workers have been found to develop chronic bronchitis, and some become severely disabled by their obstructive lung disease (3). Berry, et al. studied the workers in 14 cotton and 2 man-made fiber mills in England (1). They found that men had a greater prevalence of byssinosis than women, and that smokers of both sexes had 1.4 times greater prevalence of byssinosis than nonsmokers. Byssinosis prevalence was also positively associated with length of exposure to cotton dust in both women and men and was positively associated with dust level in the working environment in women. Berry, et al. were unable to determine if the observed difference in prevalence by sex represented a difference in physiologic response or differences in occupational exposure. They also found a higher prevalence of bronchitis in exposed versus nonexposed workers of both sexes. Smoking workers had higher bronchitis rates than nonsmoking workers.

Bouhuys, et al. studied 645 active and retired cotton textile workers (including 372 women), aged 45 and older, who had worked an average of 35 years. Their respiratory symptoms and flow-volume curves were compared to those of community resi-

TABLE 7.—Example of pulmonary irritants and inorganic sensitizers in various occupations where women work

Severe pulmonary irritant	Inorganic sensitizers	Occupation	# of women employed in thousands
Beryllium & Compounds Phosphorous Trichloride Tellurium (Hexaflouride) Zinc (Chloride fume)	Platinum Salts	Electronic Machinery, Equipment & Supplies —Household Appliances —Radio, T.V. & Communication Equipment —Electrical Machinery, Equipment & Supplies	890 67 216 604
Ammonia Chlorine Ozone Sulfuric Acid Uranium Compounds Vanadium Compounds (Pentoxide)	Phthalic Anhydride	Professional & Photographic Equipment & Watches —Scientific & Controlling Instruments —Optical & Health Services Supplies —Photographic Equipment & Supplies	238 65 119 36
Acrolein Ammonia Cadmium dust Chlorine Chromates Dichloroethyl ether Ethylene Oxide Hydrogen Chloride	Cobalt, metal fumes & dust Phthalic Anhydride	Rubber & Misc. Plastic Products —Rubber Products —Misc. Plastic Products	257 86 171

Severe pulmonary irritant	Inorganic sensitizers	Occupation	# of women employed in thousands
Hydrogen Fluoride			
Hydrogen Sulfide			
Phosgene			
Phosphorous Trichloride			
Phthalic Anhydride			
Sulfuric Acid			
Tellurium (Hexafluoride)			
Zinc Compounds			
Ammonia	Phthalic Anhydride	Leather & Leather Products	177
	Polyvinyl Chloride	—Footwear, except rubber	13
Chromic Acid & Chromates		—Leather Products, except	
Chromium, metals &		footwear	40
insoluble salts			
Hydrogen Sulfide			
Phthalic Anhydride			
Sulphur Dioxide			
Ammonia		Fabricated Metal Products	299
Cadmium dust/fumes		—Cutlery, hand tools, &	
Chromic Acid & Chromates		other hardware	52
Chromium, metal &		—Fabricated structural	
insoluble salts		metal products	78
Fluorine		—Screw machine products	26
Hydrogen Chloride		—Metal stamping	43
Nitrogen Dioxide		—Misc. fabricated metal	
Sulfuric Acid		products	101
Zinc Chloride fumes			

TABLE 7.—(Continued)

Severe pulmonary irritant	Inorganic sensitizers	Occupation	# of women employed in thousands
Chlorine Hydrogen Fluoride	Detergents (Enzymatic)	Personal Services —Laundering, Cleaning, & other Garment Services	231
Chlorine Chlorine Dioxide Chromium, metal & insoluble salts Nitric Acid Sulfur Dioxide Sulfuric Acid	Detergents (Enzymatic) Cobalt	—Beauticians	492
Ammonia Chlorine		Private Households Hotels & Motels	1,217 424
Beryllium & Beryllium compounds Chromic Acid & Chromates Chromium, metal & insoluble salts Iodine Selenium Hexafluoride Zinc Chloride fumes	Cobalt, metal fumes & dust Detergentes (Enzymatic) Platinum Salts	Professional & Related Services —Hospital Workers —Offices of Physicians —Offices of Dentists —Health Services —Convalescent Institutions	11,931 2,866 506 242 473 869

Severe pulmonary irritant	Inorganic sensitizers	Occupation	# of women employed in thousands
Ammonia	Cobalt dust	Textile Mill Products	409
Antimony	Phthalic Anhydride	—Knitting Mills	126
Bromine		—Yarn, thread & fabric mills	229
Cadmium dust/fumes		—Misc. Textile mill products	23
Chlorine		Apparel & other fabricated textile products	
Chromates			
Cotton dust, raw		—Apparel & Accessories	995
Dichloroethyl ether		—Misc. fabricated	898
Dimethylamine		textile products	97
Ethylene Chlorohydrin			
Ethylene Oxide			
Hydrogen Sulfide			
Methyl Bromide			
Nitric Acid			
Nitrogen Dioxide			
Sulfur Dioxide			
Sulfuric Acid			
Zinc Chloride fumes			
Hydrogen Sulfide		Meat Products	12,986

SOURCE: National Clearinghouse for Smoking and Health (8), Rones, P. (14), Stellman, J. (16).

dents who acted as controls (3). Textile workers of both sexes had significantly increased prevalence of chronic cough, wheezing, and dyspnea. Work in the textile mills was the major variable associated with symptom prevalence, with smoking as an additional significant variable. The lung function data confirmed the association of both smoking and working in the mills with decreased lung function. Nonsmoking female workers were slightly more likely to report chronic cough than nonsmoking men, but smoking male workers were almost twice as likely to report this symptom as smoking women. A similar pattern was seen for wheezing and chest tightness, but not for dyspnea.

Kilburn, et al. studied the prevalence of byssinosis and bronchitis in 1,046 women textile workers and showed an interaction of smoking and work exposure in producing a higher prevalence rate of both byssinosis and bronchitis at a given dust level (5).

In summary, women have clearly been shown to have a higher risk of developing byssinosis, chronic bronchitis, and chronic obstructive lung disease because of exposure to cotton dust in the workplace. Cigarette smoking has been shown to interact with some work exposures to increase this risk, although it is not established whether this interaction is additive or multiplicative. Men employed in occupations where they are exposed to cotton dust have a greater prevalence of bronchitis and respiratory disability than women. Clarification is necessary to determine whether this is a sex difference or a difference in exposure (either occupational or smoking).

Summary

1. The 1979 Surgeon General's Report identified the ways in which smoking cigarettes may interact with the occupational environment. They include:
   a) Facilitation of absorption of physical contamination of cigarettes,
   b) Transformation of workplace chemicals into more toxic substances,
   c) Addition of the exposure to a toxic constituent of tobacco smoke to a concurrent exposure to the same constituent present in the workplace,
   d) Addition of a health effect due to environmental exposure to a similar health effect due to smoking,
   e) Synergy of exposures, and
   f) Causation of accidents.
2. Women are entering occupational environments with greater frequency, and thus may be experiencing greater exposures to physical and chemical agents.

3. Cohorts of women with a greater prevalence of smoking are currently reaching the ages of maximal disease occurrence, replacing earlier cohorts with lower cigarette exposures.

4. Physiologic differences in hormonal status between males and females constitute a potential source of differing responses.

5. In the workplace women who are pregnant present a 9-month exposure opportunity, including potential teratogenic and perinatal mortality effects.

6. Concurrent exposure of women to smoking and asbestos resulted in a clear excess of cancer of the lung.

7. Women smokers exposed to cotton dust run a higher risk of developing byssinosis, bronchitic syndromes, and abnormal pulmonary function tests than nonsmoking women.

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PREGNANCY AND INFANT HEALTH.

PREGNANCY AND INFANT HEALTH

Introduction

A woman who smokes during pregnancy not only risks her own health, but also changes the conditions under which her baby develops. Studies have identified specific areas in which the effects of maternal smoking during pregnancy may occur. These include fetal growth, most often determined by comparing birth weights of smokers' babies with those of otherwise similar nonsmokers' babies; spontaneous abortions, fetal deaths, and neonatal deaths; pregnancy complications, including those that predispose to preterm delivery; possible effects on lactation; and long term effects on surviving children. The relationships between maternal smoking and these outcomes have been established by clinical, pathological, and especially epidemiological studies. Understanding of mechanisms by which smoking may produce the observed effects has been gained by physiological studies in humans and experimental studies in animals.

The Chapter on Pregnancy and Infant Health in the 1979 Surgeon General's Report is a detailed review of past studies of the effects of smoking in pregnancy, with a comprehensive bibliography. This section summarizes current knowledge in major areas of study, describes important new studies, and points out areas requiring further research (146).

Smoking, Birth Weight, and Fetal Growth

Babies born to women who smoke during pregnancy are, on the average, 200 grams lighter than babies born to comparable women who do not smoke. Since 1957, when Simpson reported this finding from her original study (138), it has been confirmed in more than 45 studies of more than half a million births (146). Results of these studies are expressed as mean birth weights of smokers' and nonsmokers' babies or, alternatively, as the percentage of babies who weigh less than a specified amount, usually 2,500 grams.

To illustrate the association between maternal smoking and an increased proportion of low-birth-weight infants, the results of five studies with an aggregated total of almost 113,000 births in Wales, the United States, and Canada are summarized in Table 1. In these populations, 34 to 54 percent of the mothers smoked during pregnancy and on the average the smokers had twice as many low-birth-weight babies as the nonsmokers. Also in these populations, from 21 to 39 percent of the incidence of

TABLE 1.—Birth weight under 2,500 grams by maternal smoking habit, relative and attributable risks derived from published studies

Study	Nonsmokers	Smokers		Births < 2,500 grams		Relative risk smoker: nonsmoker	Attributable risk* (%)
	No.	No.	Proportion	Non-smoker (%)	Smoker (%)		
Cardiff	7,176	6,238	.465	4.1	8.1	1.98	31
US Collaborative							
White	8,466	9,781	.536	4.3	9.5	2.21	39
Black	11,252	7,777	.409	10.7	17.5	1.64	21
California, Kaiser Permanente							
White	3,189	2,145	.402	3.5	6.4	1.83	25
Black	934	479	.338	6.4	13.4	2.09	27
Montreal	3,954	3,004	.432	5.2	11.4	2.19	34
Ontario	27,316	21,062	.435	4.5	9.1	2.02	31

*Percentage of total birth weights < 2,500 gm attributable to maternal smoking. Attributable risk in population = $b(r-1)$ divided by $b(r-1)+1$ where b = proportion of mothers who smoke and r = relative risk of low weight = smoker rate/nonsmoker rate.
SOURCE: Meyer, M.B. (86).

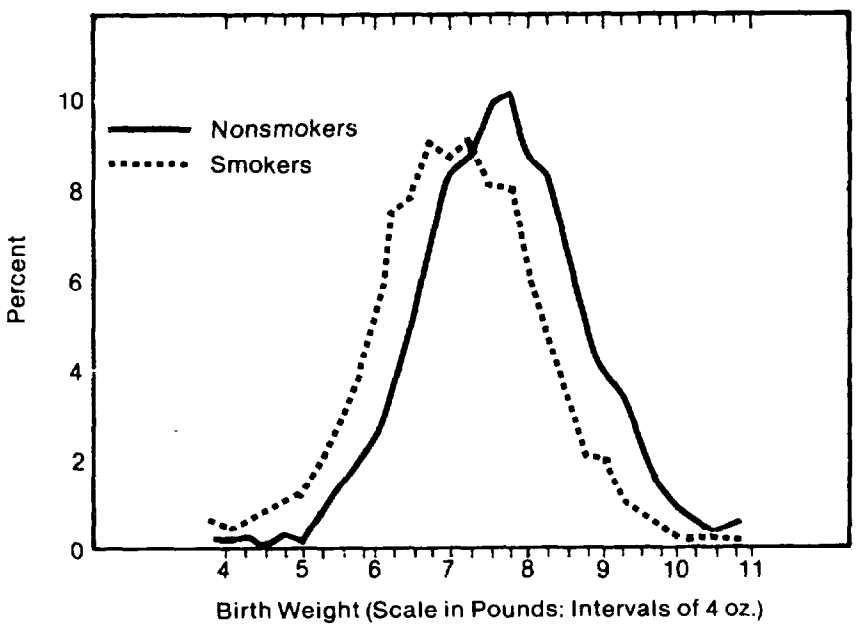

FIGURE 1.—Percentage distribution by birth weight of infants of mothers who did not smoke during pregnancy and of those who smoked one pack or more of cigarettes per day

SOURCE: MacMahon, B. (77).

low birth weight could be attributed to maternal smoking (3,15,38,86,102,106,107).

One study in which rates of low birth weight were simultaneously adjusted for multiple factors showed that maternal smoking had a more significant relationship to birth weight than did previous pregnancy history, hospital pay status, mother's prepregnant weight, height, age-parity, or sex of child. Adjusted rates of birth weights under 2,500 grams were 49 per thousand for nonsmokers, 76 per thousand for smokers of less than a pack per day, and 114 per thousand for smokers of a pack per day or more. The risk of having a low-birth-weight baby therefore increased 53 percent and 130 percent for light and heavy smokers, respectively, compared with nonsmokers (86).

Population studies that illustrate whole distributions of birth weights by maternal smoking levels show a downward shift of all birth weights in proportion to the amount smoked (74, 77,83,114,136,160) (see Figure 1).

These studies show that the relationship between smoking and reduced birth weight is independent of all other factors that influence birth weight, such as race, parity, maternal size, socioeconomic status, sex of child, and other factors that have been studied. It is also independent of gestational age. There is a dose-response relationship: that is, the more the woman smokes during pregnancy, the greater the reduction in birth weight. If a woman gives up smoking by her fourth month of gestation her risk of delivering a low-birth-weight baby is similar to that of a nonsmoker.

PLACENTAL RATIOS

Analyses of placental weights by maternal smoking habits have noted that these weights were either not affected or were less affected by maternal smoking than were birth weights (57,61,91,104,155). The placental ratio, the ratio of placental weight to birth weight, tended to be larger for smokers than for nonsmokers, mainly because of the dose-related reduction in birth weights with increasing number of cigarettes smoked.

Wingerd and colleagues have studied placental ratios based on data from 7,000 pregnancies among members of the Kaiser Foundation Health Plan in Oakland, California (156). Smoking information was obtained early in pregnancy, and placentas were handled according to Benirschke's standardized protocol. Figure 2 shows placental ratios by smoking level and gestation for single live births. At each gestational age, from 37 through 43 weeks, the more the mother smoked during pregnancy, the higher was the placental ratio. These ratios were higher for black than for white women and tended to increase as maternal hemoglobin level decreased (156).

Christianson's recent report, based on standardized examinations of these placentas, has shown that the increase in placental ratio with maternal smoking level was due to considerable decreases in mean birth weight, accompanied by slight increases in mean placental weight. In addition, smokers' placentas were significantly thinner than those of nonsmokers, and their minimum diameters were larger (19).

Maternal smoking leads to significant increases in carboxyhemoglobin in maternal and fetal blood, with a consequent reduction in the oxygen carrying capacity of both, and a reduction of the pressure at which oxygen is delivered to the fetal tissues (70,72,146). Christianson discusses the similarity between studies of placental ratios by smoking level, altitude, maternal anemia, and maternal cyanotic heart disease. She suggests that the changes in placental ratio represent an adap-

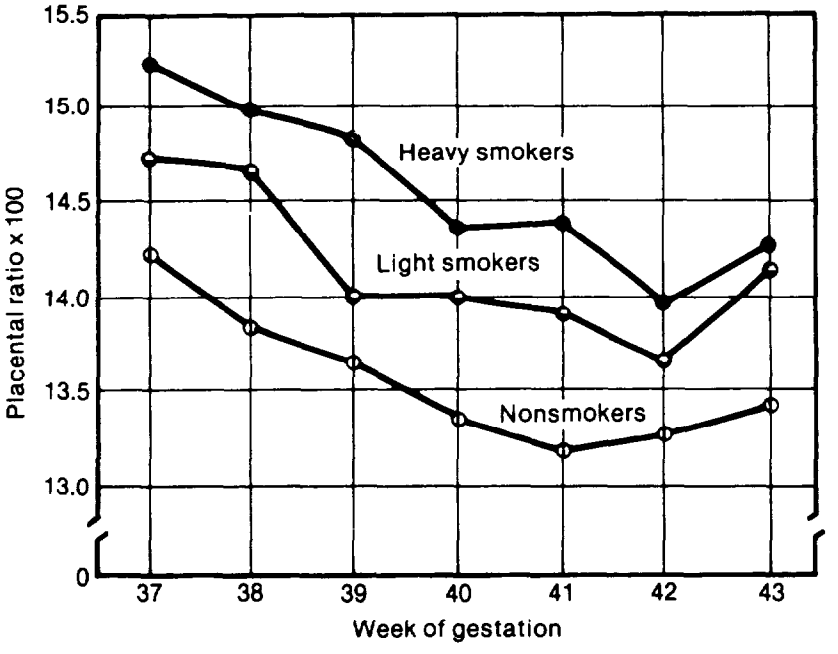

FIGURE 2.—Ratio of placental weight to birth weight by length of gestation and maternal smoking category

SOURCE: Wingerd, J. (156).

tation to relative fetal hypoxia (19). An adaptive advantage for survival might occur because a larger placenta with an increased area of attachment would deliver more oxygen, and a smaller fetus would have a decreased oxygen demand. If so, it is extremely important to know whether this reduction in size is accompanied by any long-term costs in later growth and development.

GESTATION AND FETAL GROWTH

In early studies the consistent finding that mean birth weights were lower and the frequency of births under 2,500 grams higher for women who smoked during pregnancy than for similar nonsmokers raised the obvious question of whether this might be due to a smoking-related reduction in gestation. This is not the case. Studies consistently show that mean gestation is minimally reduced by maternal smoking (less than 2 days) (3,13,146,159) and that birth weight is lower for infants of smokers than for infants of nonsmokers at each gestational age (3,15,83,146).

The finding that maternal smoking does not cause an overall downward shift in the distribution of gestational ages, as was shown for birth weights of smokers' infants, leads to the conclusion that the lower weight must be due to direct retardation of fetal growth. In other words, these infants are small-for-dates rather than preterm. The type of fetal growth retardation associated with maternal smoking is characterized by an abnormally short crown–heel length for gestational age (89,90). Smokers' babies are smaller than corresponding nonsmokers' babies in all dimensions measured, including length, head circumference, chest circumference, and shoulder circumference (10,30,31,52,57,61,102,104,146,157).

Previous studies of these measurements at birth have inferred that birth size reflects the rate of fetal growth; this has been confirmed by a definitive study in which fetal biparietal diameters were measured serially during gestation. Persson and coworkers studied 5,715 pregnancies prospectively, making ultrasonic measurements of biparietal diameters (BPD) from 18 to 20 weeks through term. Separate growth curves of BPD were constructed for fetuses of smokers and nonsmokers who were delivered between 266 and 294 days after the last menstrual period. The BPD increased faster in the nonsmoking group; the difference from the smoking group was significantly apparent from the 28th week and was positively correlated with the average number of cigarettes smoked (Figure 3). Measurements taken at birth showed that the distributions of birth weights and lengths shifted downwards in proportion to the level of smoking. Figure 4 illustrates this shift (114). These findings corroborate Miller's characterization of smokers' babies as normally proportioned but short as well as light for dates, and smaller in all dimensions than babies of nonsmokers (90). The data are also consistent with the speculation that relative fetal hypoxia results in a slower mitotic rate, a baby with fewer cells, and a reduced oxygen demand.

LONG-TERM GROWTH AND DEVELOPMENT

Possible long-term consequences of maternal smoking during pregnancy are also of concern. Several long-term studies provide evidence that children of smoking mothers have slight but measurable deficiencies in physical growth, intellectual and emotional development, and behavior (95).

Because these complex outcomes are affected by many known and unknown factors, it is important to take these other factors into account in any attempt to measure long-term effects of maternal smoking. Several well-controlled studies have shown

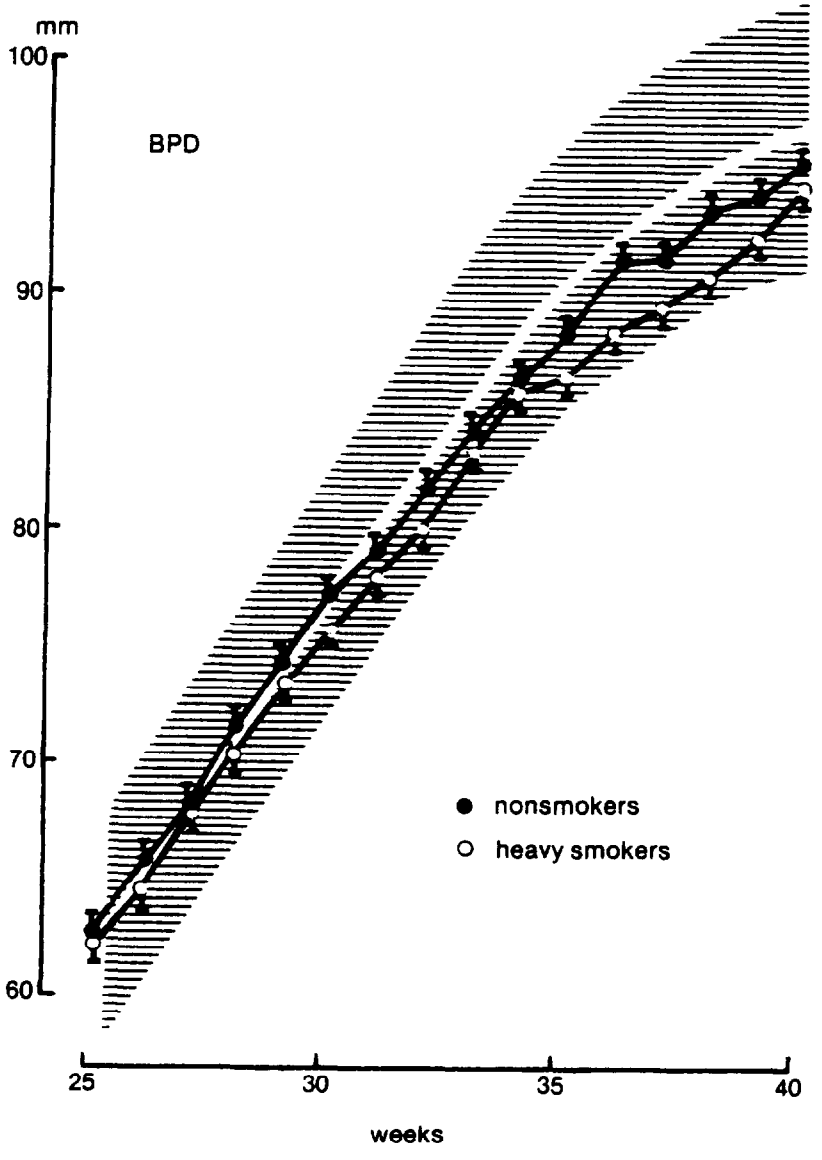

FIGURE 3.—Fetal Biparietal Diameters (BPD) values [means and standard error of means (SEM)] of nonsmokers and heavy smokers (10 cigarettes/day) plotted in relation to postmenstrual age against the normal range (shaded area depicts 95% confidence interval)

SOURCE: Persson, P.H. (114).

that the physical growth of smokers' babies remains behind that of nonsmokers' babies as measured at 7 to 14 days (31); 1 year, 4 years, and 7 years (pairs of births matched for race, date of delivery, maternal age and education, and sex of child) (52); 5 years (adjusted for other factors) (157); up to 6½ years (prospective study) (35); and at ages 7 and 11 (follow-up studies of the 17,000 children from the British Perinatal Mortality Study, with the adjustment for other social and biological factors) (16,30,33).

Associations have also been noted between maternal smoking and deficiencies in neurological and intellectual development of the child. Hardy and Mellits analyzed findings for 88 pairs of children of smokers and nonsmokers, matched for race, date of delivery, maternal age and education, and sex of the child. Although they reported no significant differences in intellectual function between children born to smoking and nonsmoking mothers, the direction of difference on almost all tests was in favor of the nonsmokers' babies. Fewer smokers' than nonsmokers' children had normal neurological status at age 1 year, both in the original 88 matched pairs and in the additional set of 55 pairs of children of smokers and nonsmokers, matched for birth weight as well as for the other cited factors. In both sets, smokers' children had lower scores on the majority of tests of intelligence and intellectual function at ages 4 and 7 (52,146).

Similarly, Dunn evaluated neurological, intellectual, and behavioral status in a prospective study of low-birth-weight infants, including 76 who were "small-for-dates" (term and preterm), 92 "truly premature" (preterm with birth weight between 11 and 89 percentile) and 151 full-birth-weight control infants. Neurological abnormalities, including minimal cerebral dysfunction and abnormal or borderline electroencephalograms, were slightly more common among children born to women who smoked (Table 2).

In a battery of psychological tests, the mean scores of children of nonsmoking mothers were better than those of smokers' children in 45 out of 48 correlations, and the difference was significant in 14 of these. Some significant differences in favor of nonsmokers' children were also demonstrated with respect to behavior ratings and school placement (35). These results are very similar to those of Hardy and Mellits in that the direction of the differences was almost always in favor of the nonsmoker's child.

Small numbers and population selection factors were not a problem in the longitudinal follow-up of the population originally included in the British Perinatal Mortality Study, comprising approximately 17,000 births, an estimated 98 percent of all births in England, Scotland, and Wales during the week of

TABLE 2.—Incidence of neurological abnormalities at about 6½ years, by maternal smoking habits

Diagnosis	Percent of Children with Diagnosis Maternal Smoking Habits		
	Smoker	Nonsmoker	P
Minimal cerebral dysfunction	20.0	11.0	<.05
Total neurological abnormalities	29.4	19.5	<.05
EEG borderline or abnormal			
Low-birth-weight children	46.3	32.4	NS
Full-birth-weight children	28.2	21.6	NS

NS = not significant.
SOURCE: Dunn, H.G. (35).

March 3 to 9, 1958. These children have been traced and studied again at ages 7 and 11, to describe their behavior, their health, their physical development, their educational standards, and their home environment. At ages 7 and 11 years, physical and mental problems due to maternal smoking during pregnancy were found, and these increased with the number of cigarettes smoked.

Children whose mothers smoked 10 or more cigarettes a day during pregnancy were on average 1.0 centimeter shorter and 3 to 5 months retarded in reading, mathematics, and general ability, as compared with the offspring of nonsmokers. After allowing for associated social and biological factors, all of these differences were highly significant, as illustrated in Figure 5 ($p < 0.001$) (16,30).

Denson's case-control study of hyperkinesis reported a highly significant association of hyperkinesis with heavy maternal smoking, which at a mean level of 23.3 cigarettes per day was more than three times the average for two control groups. The authors concluded that their findings were "consistent with the hypothesis that smoking during pregnancy is an important cause of the hyperkinetic syndrome" (31).

A recent comparison by Saxton of behavioral patterns of infants of mothers who smoked during pregnancy with infants of mothers who did not smoke found that these patterns can be influenced by smoking in pregnancy, and that the auditory senses are particularly affected. Fifteen smokers of more than 15 cigarettes per day and 17 nonsmokers were selected for study, matched for maternal age, social class, and parity. All infants were spontaneous term deliveries of normal birth weight. Sex distribution, length of labor, analgesia, and obstetrical factors were similar for the two groups. Examiners who did

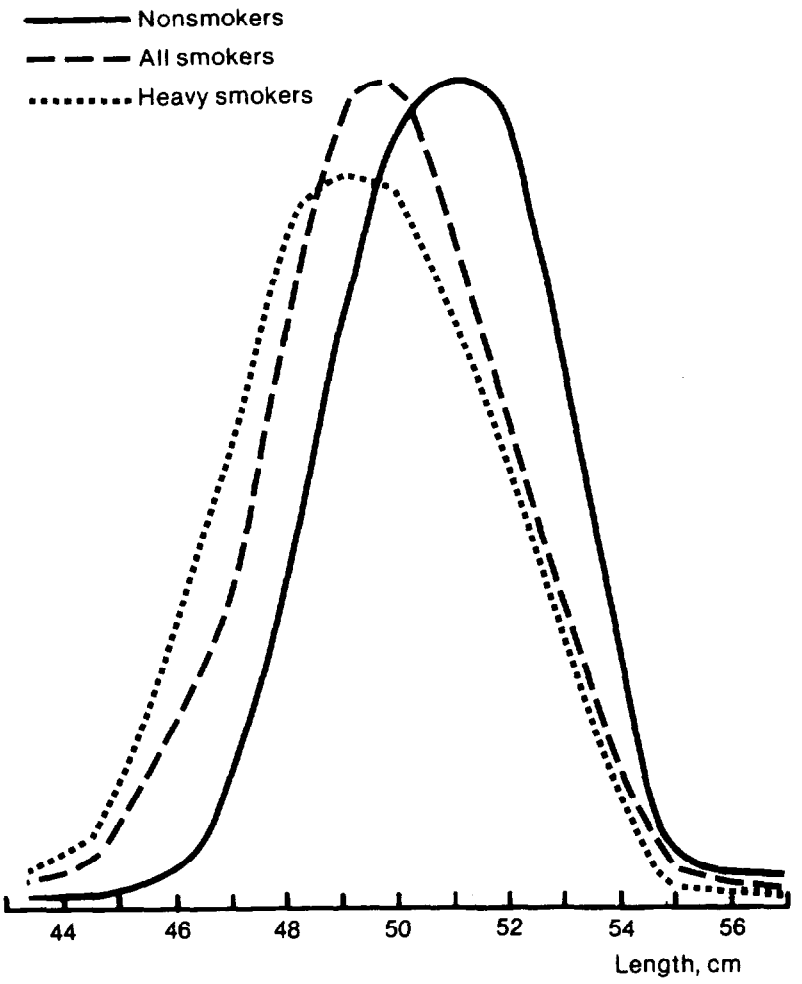

FIGURE 4.—Distribution of birth lengths

SOURCE: Persson, P.H. (114).

not know the smoking status of the mother evaluated the infants at 4 to 6 days of age, using the Brazelton Neonatal Behavioral Assessment Scale. The scale includes a total of 20 tests and maneuvers. While many of these showed no statistically significant differences, auditory tests or tests with auditory components were significantly different. Recorded "overall impressions" of the infants at the end of the test showed that the smokers' infants tended towards "irritability, decreased ability for self-control, and a general lack of interest, whereas the nonsmokers, infants tended to be less irritable and better oriented." The author concluded that some effect on the normal