The Health Consequences of SMOKING 1977-1978

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The Honorable Thomas P. O'Neill
Speaker of the House of Representatives
Washington, D.C. 20515

Dear Mr. Speaker:

As required by Section 8(a) of the Public Health Cigarette Smoking
Act of 1969, I am submitting the 1977-1978 report on the health
consequences of smoking. The report includes the "Bibliography on
Smoking and Health—1976," the "Bibliography on Smoking and
Health—1977," and "The Health Consequences of Smoking, 1977-
1978."* The report bears a 2-year designation in order to return the
series to an annual timetable which was altered because of the time
required for the clearance processing of the 1976 report. The Bib-
liographies are prepared annually and routinely to reflect the new
acquisitions to the smoking and health data base which operates at
a cost of $200,000.00 per year; the health consequences of smoking
report, which is a review of this new current information and pre-
pared specifically for Congress, this year cost $9,800.00.

"The Health Consequences of Smoking, 1977-1978" includes re-
cently published data from three classic prospective studies of the
mortality resulting from cigarette smoking. These studies, involving
almost one and a half million persons, continue to document excess
mortality among smokers as compared to nonsmokers.

This part of the report also includes data on the established risks
of low birth weight and increased perinatal mortality for offspring
of women who smoke during pregnancy. In addition, the new evi-
dence is reviewed that shows not only a high rate of heart attacks
among women who smoke cigarettes, but that this effect is particu-
larly critical in women who use oral contraceptives.

The data in this report indicate that former smokers show lower
death rates than continuing smokers and within 10 to 15 years after
quitting come close to the low rates of those who never smoked.

One study supports previous evidence that there is a partial
solution to the health problem in the use of cigarettes with lower
emissions of "tar" and nicotine.

As a result of public demand and a responsive industry, there
has been over recent years a continuing decline in the emissions of
"tar" and nicotine in cigarettes in use.

The data in this report and in previous annual reviews of the
health consequences of smoking have established cigarette smoking
as a habit responsible for an overwhelming level of premature death
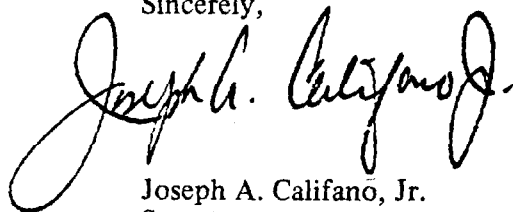and disability in this country. To reduce this preventable and costly

*The bibliographies have been published as DHEW Publication Number (CDC) 78-8309,
January and February 1978.

mortality and morbidity, this Department recently announced a new antismoking program.

The program is one of public education, regulation, and research with special emphasis on children, teenagers, and young women, and on occupations where smoking increases risks from occupational exposure. In undertaking this program, I have invited the cooperation of the major broadcast networks, State and local school officials, the major corporations of this Nation, State Governors and legislators, the Federal Trade Commission, the Federal Communications Commission, the Civil Aeronautics Board, and others whose involvement and cooperation are crucial to the success of this program. In response to the evidence linking the combined use of oral contraceptives and cigarette smoking, the Food and Drug Administration, Public Health Service, HEW, has recently required that a warning statement to that effect accompany oral contraceptives as they are distributed to those who use them. To provide leadership and to coordinate this program, an Office on Smoking and Health has been established in the Office of the Assistant Secretary for Health. As one of its first tasks, this Office will coordinate the production of a comprehensive document which reviews not only the biomedical but also the behavioral and control data about smoking and its effects on health. The report will be submitted to Congress in January 1979.

As the principal health officials of this government, the Surgeon General and I are committed to fulfilling our responsibilities to provide information and direction to permit American citizens to make genuinely free choices about smoking and their own health. In this regard and as I am required by P.L. 91-222 to make such legislative recommendations that I deem appropriate based on the scientific data about the impact of smoking on health, I will submit within the year a legislative package which I hope will meet with your approval. With appropriate coordination of legislative action and program, we can solve this difficult and important public health problem.

Sincerely,

Joseph A. Califano, Jr.
Secretary

Enclosures

Identical letter sent to The Honorable Walter F. Mondale

Preface

This tenth report to the Congress on the health consequences of smoking discusses the special problems incurred by women who smoke and presents recently published overall mortality data on smoking.

Smoking was first recognized as a health problem in the 1930's, when a sharp increase was noted in lung cancer rates for men. No similar increase was noted for women at that time for several reasons. First, as a group, women did not start smoking when men did, since such behavior was socially unacceptable for women at that time. Consequently, until the last decade, there were insufficient numbers of women who had smoked for a long enough period of time to provide the size population necessary for meaningful research.

In recent years, however, the same health risks to men as a result of smoking have been documented for women who smoke. These include cardiovascular disease, lung cancer, cancer of other specific sites, bronchitis, and emphysema. These diseases occur among smokers at rates far greater than those of nonsmokers. Additionally, women have been found to incur unique risks for themselves and for their offspring. For example, women over 30 years of age who smoke and use oral contraceptives have substantially higher risks of myocardial infarction. Moreover, the offspring of women who smoke during pregnancy face greater risks of perinatal mortality and low birth weight. Further understanding of the mechanisms involved in these health consequences continues to evolve.

Three large prospective epidemiologic studies demonstrate that overall mortality rates for cigarette smokers are approximately 70 percent higher than those for nonsmokers. These studies also document a decrease in overall mortality rates for those who quit smoking, provided they were not ill at the time of cessation. There is about a 15 percent reduction in overall mortality risk for smokers of low "tar" and nicotine cigarettes (less than 17.6 mg. "tar" and less than 1.2 mg. nicotine) compared to those who smoke high "tar" and nicotine cigarettes (25.8-35.7 mg. "tar" and 2.0-2.7 mg. nicotine).

Several publications have become available since the last report to Congress which review the social, behavioral, legislative, and health issues related to smoking. A recently published paper by Daniel Horn, Ph.D., as part of his work with the World Health Organization, discusses the major barriers to be overcome if further progress is to be made against the threat of smoking to health. A copy is included as Appendix A to this report. Two other publications of note include the U.S. Public Health Service's "Proceedings of the Third World Conference on Smoking and Health, 1975," DHEW Publication No. (NIH) 77-1413, 1977, Volumes I and II, and the World Health Organization's "Smoking and Its Effects on Health," Technical Report Series No. 568, Switzerland, 1975.

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Preparation of the Report and Acknowledgments

PREVIOUS REPORTS

Reviews of the scientific information linking smoking to health problems began in 1964 with the publication of *Smoking and Health, Report of the Advisory Committee to the Surgeon General of the Public Health Service*, subsequently referred to as the "Surgeon General's Report." Thereafter, Public Law 89-92 was passed requiring supplemental reports to Congress on this subject, and the following three reports were published:

1. *The Health Consequences of Smoking, A Public Health Service Review; 1967.*

2. *The Health Consequences of Smoking, 1968 Supplement to the 1967 PHS Review.*

3. *The Health Consequences of Smoking, 1969 Supplement to the 1967 PHS Review.*

Public Law 91-22 amended the previous law in April 1970 and required a comprehensive review within 18 months, with annual reports to be submitted thereafter. The result of this review was *The Health Consequences of Smoking, A Report of the Surgeon General; 1971.* Since then, the following annual reports on the health effects of smoking have been published:

1. *The Health Consequences of Smoking, A Report of the Surgeon General, 1972.*

2. *The Health Consequences of Smoking, 1973.*

3. *The Health Consequences of Smoking, 1974.*

4. *The Health Consequences of Smoking, 1975.*

5. *The Health Consequences of Smoking, A Reference Edition, 1976.*

Each report since the original "Surgeon General's Report" has reviewed the scientific literature relevant to the association between

smoking and cardiovascular diseases, non-neoplastic bronchopulmonary diseases, and cancer. Smoking as related to the following diseases and conditions has been reviewed periodically in these reports:

Allergy (1972)
Exercise Performance (1973)
Harmful Constituents of Cigarette Smoke (1972)
Noncancerous Oral Disease (1969)
Overview: The Health Consequences of Smoking (1975)
Overview: The Health Consequences of Smoking (1976)
Peptic Ulcer Disease (1967, 1971, 1972, 1973)
Pipe and Cigar Smoking (1973)
Pregnancy (1967, 1969, 1971, 1972, 1973)
Public Exposure to Air Pollution from Tobacco Smoke (1972, 1975)
Tobacco Amblyopia (1971)

THE 1977-1978 REPORT

This publication, *The Health Consequences of Smoking, 1977-1978,* contains the most recent data on the health effects of smoking unique to women and on the effects of smoking on overall mortality. Although emphasis is on the most recent data, research from earlier years is included where necessary for clarity.

The report was prepared in the following way by the staff of the National Clearinghouse for Smoking and Health, a division of the Bureau of Health Education, Center for Disease Control, Public Health Service:

1. The Technical Information Center of the Clearinghouse continually monitors and collects the scientific literature on the health effects of smoking by means of several established mechanisms:
    a. An information science corporation is under contract to

extract articles on smoking and health from the scientific literature of the world.

b. The National Library of Medicine, through the MEDLARS system, provides a monthly listing of articles on smoking and health. Articles not provided by the information science corporation are obtained for review.

c. Staff members review current medical literature and identify pertinent articles.

2. Initial drafts for the present report were prepared by the staff of the National Clearinghouse and sent to experts in the content area for review and comment regarding the format, the appropriateness of the articles selected for discussion, and conclusions. The drafts were then revised by the Clearinghouse to incorporate these comments. The final drafts of the complete report were reviewed by the National Cancer Institute, the National Heart, Lung, and Blood Institute, the National Institute of Environmental Health Sciences, the National Institute of Child Health and Human Development, and by additional experts both inside and outside the Public Health Service.

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Chapter 1

Smoking-Related Health Problems
Unique to Women

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Chapter 1

Smoking-Related Health Problems
Unique to Women

INTRODUCTION

Smoking habits and attitudes among women and teenage girls have differed in the past from the habits and attitudes among men and teenage boys. Women tended to smoke fewer cigarettes, were less likely to inhale, and were more likely to smoke low "tar" and nicotine and filter-tipped brands. Surveys have indicated, however, that the smoking habits of women are becoming more like men's. Women are taking up the habit at an earlier age and have become heavier smokers. This has made them more vulnerable not only to lung cancer and other smoking-related diseases, but also to specific health problems that are unique to their sex. For example, research on the relationship between cigarette smoking and the outcome of pregnancy has established that there are definite risks to both the fetus and the mother associated with cigarette smoking during pregnancy. Moreover, women who use oral contraceptives are at greater risk of cardiovascular disease if they smoke cigarettes. There is also evidence that nicotine is present in the breast milk of lactating mothers who smoke. The following is a review of the current information on these and other health consequences of smoking unique to women.

EFFECTS OF SMOKING ON THE OUTCOME OF PREGNANCY

There are definite health risks associated with smoking and pregnancy, including effects on birth weight, perinatal mortality, and long-term physical and intellectual development of the child. This section reviews each of these subjects and also includes information about the likely mechanism of action of smoke and its contents on the mother and the products of conception.

Smoking and Birth Weight

In 1957, Simpson published her original finding that babies born to women who smoke during their pregnancy weigh on the average 200 grams (g) less than the babies born to women who do not smoke

(34). Since then, more than 100 articles on this relationship have led to the general acceptance that smokers' babies generally weigh 150 to 250 g less than nonsmokers' babies, and twice as many of the former weigh less than 2500 g (13). The 1973 report of *The Health Consequences of Smoking* presented evidence to support a causal association between cigarette smoking and fetal growth retardation (39). A strong dose-response relationship was also established in that report, with differences in weight being in direct proportion to the number of cigarettes smoked.

The following additional points were summarized in the 1973 report to further support the causal association between cigarette smoking during pregnancy and lower birth weight:

1. Results are consistent in all studies, retrospective and prospective, from many different countries, races, cultures, and geographic settings.

2. The relationship between smoking and reduced birth weight is independent of other factors that influence birth weight, such as race, parity, maternal size, socioeconomic status, sex of child, and all others that have been studied.

3. If a woman gives up smoking by the fourth month of pregnancy, her risks of delivering a low-birth-weight baby is similar to that of a nonsmoker.

Subsequent to the 1973 report, additional reports have further discussed and corroborated the association between smoking in pregnancy and low birth weight (19, 25, 33, 35).

Smoking and Perinatal Mortality

A strong, probably causal, association between cigarette smoking and higher late fetal and infant mortality rates among smokers' infants is now well established (38). Retrospective and prospective studies have revealed a statistically significant relationship between cigarette smoking and an elevated mortality risk among the infants of smokers. In three of these studies of sufficient size to permit adjustment for other risk factors, a highly significant independent association between smoking and mortality was established. Part of the discrepancy in results between these studies and those in which a significant association between smoking and infant mortality was not demonstrated may be explained by a lack of adjustment for risk factors other than smoking.

The 1973 report also presented evidence indicating that the higher relative risks occurred among populations with risk factors other than smoking being present, such as socioeconomic status, age, parity, race, and previous pregnancy history.

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Since 1973, a series of articles by Meyer, et al. analyzed data from the Ontario Perinatal Mortality Study of all single births in ten Ontario teaching hospitals in 1960-61 (26, 27, 28). The study involved 51,490 births, including 701 fetal deaths and 655 early neonatal deaths, and was supplemented by clinical records with interviews of mothers in the hospital, interviews with anesthetists and attending physicians, and autopsy records (29). Perinatal mortality increased significantly with smoking and was also affected by such factors as maternal age, parity, socioeconomic status, previous pregnancy history, hemoglobin level, and other risk factors (29). Smoking frequencies also varied by many of these characteristics. Smoking and other risk factors were cross-tabulated among 52 data subgroups. In all subgroups, the mortality increase with smoking was dose related, but not in a simple, linear way. The increased risk of perinatal mortality associated with light smoking among young, low-parity, nonanemic mothers was less than 10 percent. At the other extreme, mothers with other risk factors of high parity, public hospital status, with previous low-birth-weight infants, or with hemoglobin less than 11 g had further increased perinatal mortality risks of 70-100 percent when they were smokers. The most significant risk factor (mortality rate of 78 per 1,000 total births) was anemia, defined as a hemoglobin of less than 8.0 g. The failure of some earlier studies to find a significant increase in perinatal mortality with maternal smoking may be due to selection of study populations from the end of the spectrum, where light smoking is associated with only a slight increase in perinatal risk. This evidence points up how population selection could influence study findings and shows that exposure to the effects of smoking during pregnancy is much more dangerous for the babies of some women than for others. These findings are corroborated by a number of studies in which fetal, neonatal, or perinatal mortality rates are compared for smoking and nonsmoking women, controlling for the effects of various risk factors previously mentioned (1, 12, 22, 36).

Additional data were published in 1976−1977 (26, 27) and revealed that frequencies of low birth weight (under 2500 g), preterm delivery (< 38 weeks), perinatal mortality, abruptio placentae, placenta previa, bleeding during pregnancy, and prolonged and premature rupture of the membranes increased directly and significantly (p< 0.00001) as the level of maternal smoking increased (Tables 1 and 2). The 1976 paper used multiple regression analysis to measure the independent effect of smoking on the various risk factors. The probabilities of these complications were also compared (Figure 1). Risks of placenta previa and abruptio placentae were higher for smokers than for nonsmokers at all gestations, with

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TABLE 1. Adjusted rates and F ratios for maternal smoking and other important factors affecting birth weight, gestation, placental complications, and perinatal mortality

Factor	*Adjusted Rates of Outcome	†F Ratio
Birth Weight < 2500 Grams **Per 1000 Births**		
Maternal Smoking Level		
None	49.4	182.8
< 1 Pack Per Day	75.7	
> 1 Pack Per Day	113.7	
Previous Pregnancy History		
No Previous Pregnancy	70.0	123.5
Previous Pregnancy, 0 Loss	57.8	
Previous Pregnancy, Loss	134.8	
Hospital Pay Status		
Private	60.0	84.0
Public	87.4	
Gestation < 38 Weeks **Per 1000 Births**		
Maternal Smoking Level		
None	77.1	50.6
< 1 Pack Per Day	92.2	
> [1] Pack Per Day	115.9	
Previous Pregnancy History		
No Previous Pregnancy	69.1	182.6
Previous Pregnancy, 0 Loss	85.7	
Previous Pregnancy, Loss	193.9	
Hospital Pay Status		
Private	78.9	120.3
Public	116.2	
Placenta Previa **Per 1000 Births**		
Maternal Smoking		
None	6.5	11.7
< 1 Pack Per Day	8.1	
> 1 Pack Per Day	12.5	
Previous Pregnancy History		
No Previous Pregnancy	8.8	14.4
Previous Pregnancy, 0 Loss	6.6	
Previous Pregnancy, Loss	15.8	

(Hospital pay status not a significant factor)

TABLE 1. Adjusted rates and F ratios for maternal smoking and other important factors affecting birth weight, gestation, placental complications, and perinatal mortality *(continued)*

	Abruptio Placentae Per 1000 Births	
Maternal Smoking		
None	16.4	**17.1**
< 1 Pack Per Day	20.3	
> 1 Pack Per Day	27.6	
Previous Pregnancy History		
No Previous Pregnancy	18.8	**25.6**
Previous Pregnancy, 0 Loss	17.6	
Previous Pregnancy, Loss	37.4	
Hospital Pay Status		
Private	17.5	**20.7**
Public	25.0	
	Perinatal Mortality Per 1000 Births	
Maternal Smoking		
None	23.5	**8.4**
< 1 Pack Per Day	28.2	
> 1 Pack Per Day	31.8	
Previous Pregnancy History		
No Previous Pregnancy	23.1	**97.4**
Previous Pregnancy, 0 Loss	23.6	
Previous Pregnancy, Loss	68.7	
Hospital Pay Status		
Private	23.3	**44.2**
Public	36.1	

*Adjusted rates show independent effect of the factor given, adjusted for all other factors in regression. They are: maternal smoking, hospital pay status, mothers' birthplace, height, prepregnant weight, sex of child, previous pregnancy history, and age-parity.

†F ratio degrees of freedom: numerator = number of subgroups −1, denominator = infinity. (All differences shown are highly significant. F ratios indicate the relative importance of the factor.)

SOURCE: Personal correspondence, based on data in Meyer, M.B., et al. (26).

FIGURE 1.—Risks of selected pregnancy complications for smoking and nonsmoking mothers, by period of gestational age at delivery for A, abruptio placentae, B, placenta previa, C, premature rupture of membranes (PROM)

SOURCE: Meyer, M.B., et al. (27).

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y-axis: Probability of Prom (0.0001 to 0.1, logarithmic)
x-axis: Gestation: Weeks (20 to 44)

Curves labeled: Smokers, Nonsmokers
Circled: C
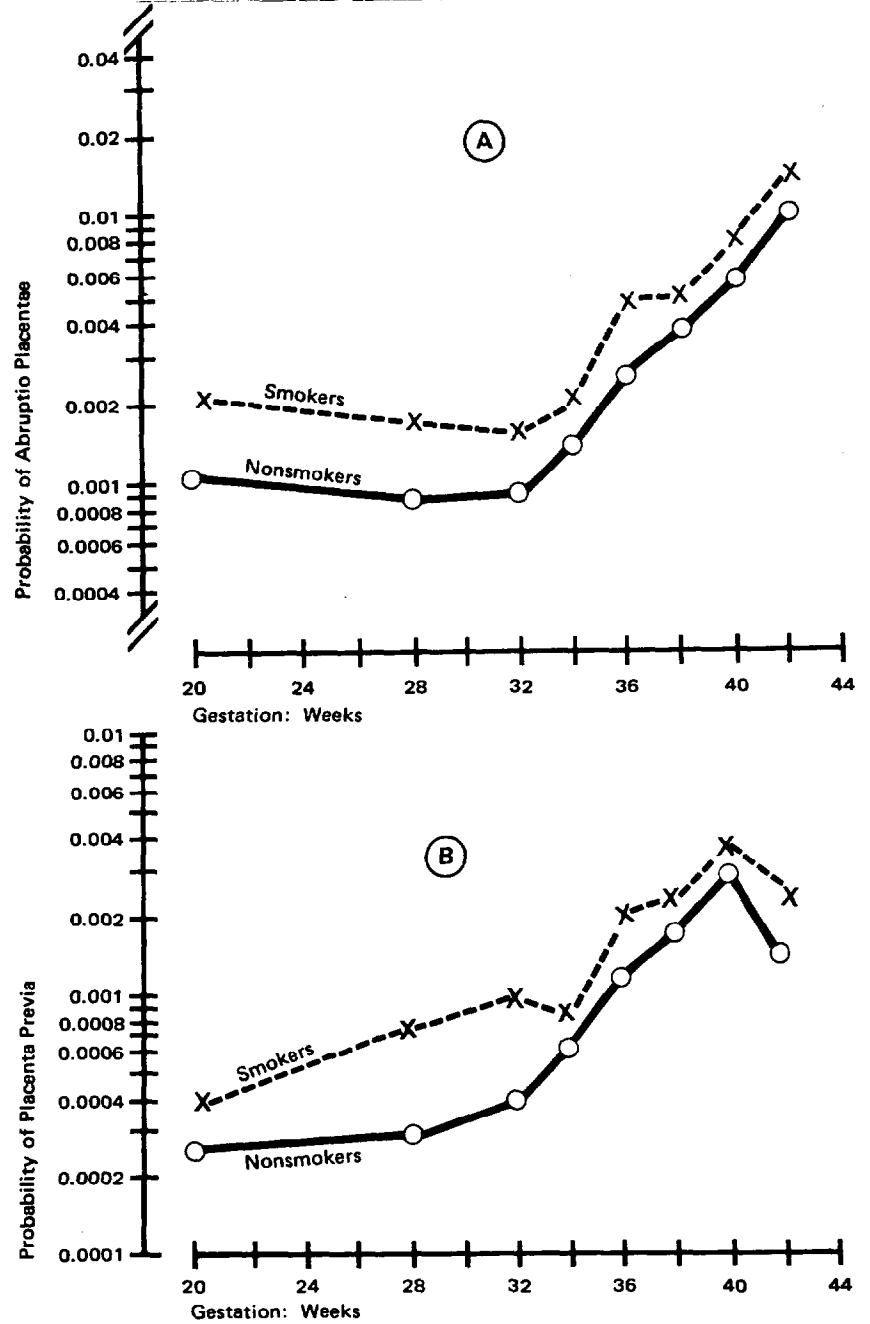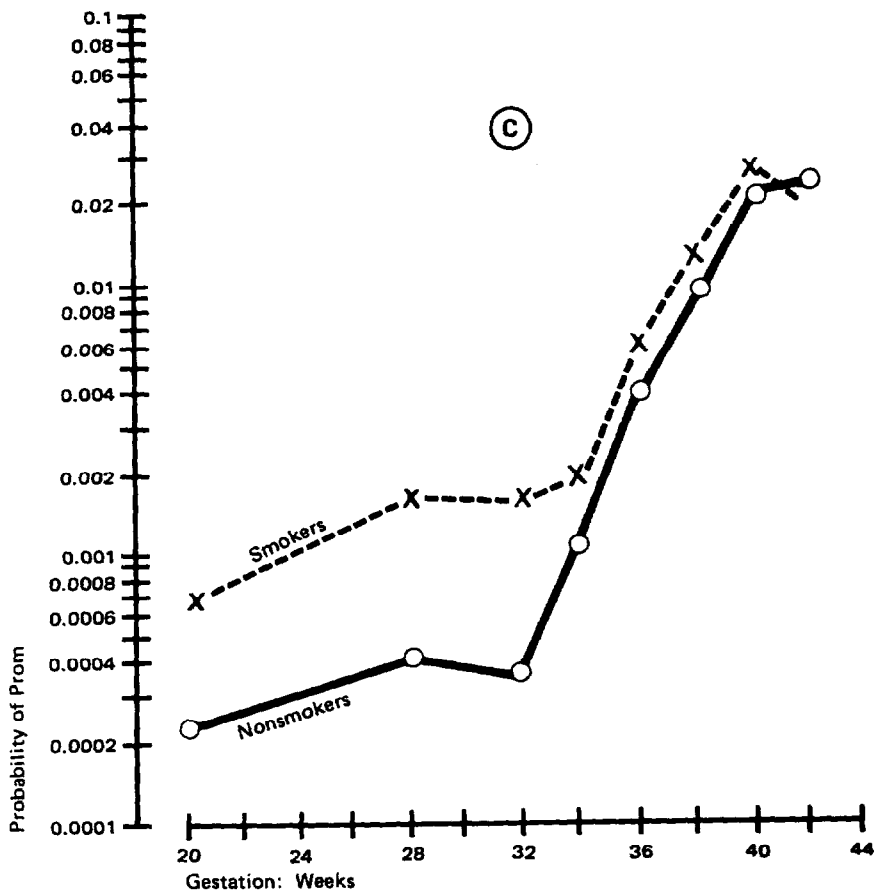
TABLE 2. Perinatal mortality and selected pregnancy complications, by maternal smoking levels

	Smoking level (packs per day) (rates per 1,000 total births)			
Outcome	0 23,358 Births)	< 1 (15,328 Births)	> 1 (6,581 Births)	X^{2*}
Perinatal Mortality	23.3	28.0	33.4	27.8‡
Abruptio Placentae	16.1	20.6	28.9	47.3‡
Placenta Previa	6.4	8.2	13.1	28.6‡
Bleeding During Pregnancy	116.5	141.6	180.1	201.9‡
Rupture of Membranes >48 Hours	15.8	23.3	35.8	109.9‡
Rupture of Membranes Only at Admission	30.3	39.3	45.0	45.7‡

*Cochran's chi square for trends.

‡p < 0.00001.

SOURCE: Meyer, M.B., et al. (27).

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relatively larger differences in the earlier weeks of pregnancy. The risk of premature rupture of membranes was more than three times greater for smokers than for nonsmokers among deliveries that occurred before 34 weeks gestation and remained higher than the risk for nonsmokers through term (Figure 1C).

A prospective investigation of 9,169 pregnant women was conducted by Goujard, et al. (15), and results showed a substantial increase in stillbirths among smokers. A large proportion of this increase was due to abruptio placentae. There were 100 stillbirths, classified into five categories of causes: vascular, abruptio placentae, mechanical, miscellaneous (syphilis, Rh, malformations, etc.), and unknown (Table 3). The abruptio placentae category exclusively represented cases without toxemia, the one toxemic case being classified with the vascular causes. The higher proportion of smokers is significant for only two of the categories: abruptio placentae (p = 0.005) and unknown causes (p = 0.0005). Although the numbers were small, the risk of stillbirths by abruptio placentae is six times higher among smokers.

TABLE 3. Stillbirths according to cause in relation to maternal smoking during pregnancy

Stillbirths	Number of Deliveries	Percent Smokers	Comparison With Live Births±
Cause of Death:			
Vascular	8 ·	25	
Abruptio Placentae	13	46	p = 0.005
Mechanical	13	15	
Miscellaneous (Syphilis, Rh, Malformations, . . .)	24	13	
Unknown	37	35	p = 0.0005
Detailed Records Not Available	5	—	
TOTAL	100	26	p = 0.0001
Livebirths	9069	12	

± When p is not given, the difference is not significant.

SOURCE: Goujard, J., et al. (15).

Long-Term Effects on Physical and Intellectual Development

Three studies (6, 16, 40) report on long-term effects of smoking in pregnancy. Data from two of the studies presented below demonstrate an association between smoking during pregnancy and impaired physical and intellectual development in the offspring. Additional reports further substantiate this association (10, 11).

Butler and Goldstein (6) analyzed the National Child Development Study, a longitudinal study of 17,000 children born in Britain from March 3 to 9, 1958. The test procedures included a reading

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test at the age of 7 years, and a mathematics test, a reading test, and a general ability test at the age of 11. At both ages the height of the child was also measured. Analyses at both ages were based on smoking habits of the mother after the fourth month of pregnancy

Statistically significant differences in height and reading ability between smoking categories (0, 1-9, or 10+ cigarettes daily) were found at both 7 and 11 years of age.

When account was taken for such factors as mother's height, age, social class as determined by father's occupation, number of older and younger children in the household, and the sex of the child, there was a deficit of height and reading ability in the offspring of mothers who smoked, the extent of which increased with the amount smoked.

These results establish an association of smoking in pregnancy with later intellectual development, although the gap between children of smokers (at all levels of smoking) and nonsmokers does not appear to change between the ages of 7 and 11 years. Smoking in pregnancy is associated with an impairment of both mental and physical growth, although compared with other social and biological factors, the effects are small.

In the study by Wingerd and Schoen (40), the net effects of various factors on length at birth and height at 5 years were determined in 3,707 single-born, white California children. Children of smoking mothers were found to be shorter (p< 0.001) at birth and at 5 years than children of nonsmoking mothers. (Intellectual development was not measured in this study.)

In contrast to these results, Hardy and Mellits (16) found very few significant differences in a number of body measurements and intellectual functions up to the age of 7 years between children of smokers and nonsmokers. A possible explanation for this discrepancy is that their sample was too small, and a weight-matched control group could add a bias. Whereas the British study by Butler and Goldstein involved a sample size of over 5,000 children, Hardy and Mellits based their findings on only 88 matched pairs of children. Calculations by the authors of the British study show that with the small sample used by Hardy and Mellits there was only about a 20 percent probability of detecting statistically significant differences in the heights of children born to smoking and nonsmoking mothers.

CARBON MONOXIDE AND CARBOXYHEMOGLOBIN LEVELS IN MATERNAL AND FETAL CIRCULATION AND THE POSSIBLE MECHANISMS OF SMOKING EFFECTS ON PREGNANCY

There is evidence to show that carboxyhemoglobin (COHb) levels are substantially elevated in pregnant women who smoke and may result in damage to placental and fetal blood vessels. Higher levels

of COHb in both fetal and maternal blood may also be a factor in the increased incidence of low birth weight of infants born to women who smoke.

Cole, Hawkins, and Roberts (7) studies the smoking habits of a group of pregnant women and related these to the level of COHb in the circulating blood. A group of 222 patients attending antenatal clinics at a London hospital were questioned about their smoking habits. Ninety-three (42 percent) were smokers, and 129 (58 percent) were nonsmokers. Simultaneous maternal and cord blood samples were taken at normal delivery and at Caesarean section from 28 patients, and the COHb and fetal hemoglobin levels of the samples were measured. Results showed that women who smoke during pregnancy have a significantly higher level of COHb in their blood than women who do not smoke ($p < 0.01$). The mean COHb levels were 1.2 percent (range 0 to 2.4 percent) for the non-smokers and 4.1 percent (range 0.5 to 14 percent) for the smokers. There was a positive correlation betwen the number of cigarettes smoked on the day of sampling and the COHb level (correlation coefficient 0.82) (Figure 2). With the exception of two patients,

FIGURE 2.—Number of cigarettes normally smoked per day compared with COHb level at time of sampling in 93 pregnant women. ♀ = Mean range of COHb levels for 129 nonsmokers
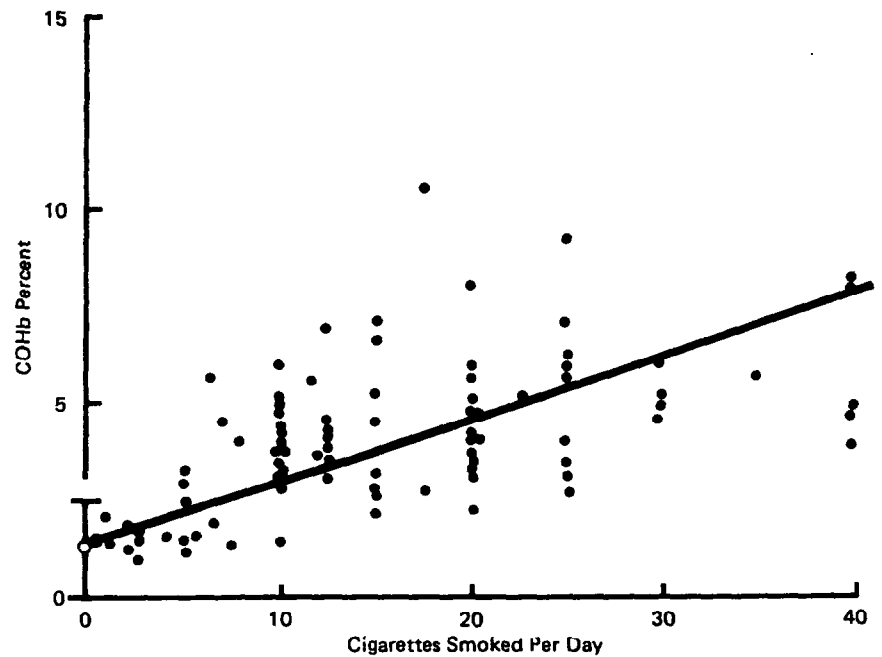
SOURCE: Cole, P.V., et al. (7)

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all the fetal COHb levels were demonstrably higher than the respective maternal ones. The mean fetal/maternal COHb ratio was 1.84 to 1 (standard deviation ∓ 0.85). Hemoglobin has a 210 times greater affinity for carbon monoxide (CO) than for oxygen. It is obvious, therefore, that cigarette smoking during pregnancy diminishes the oxygen carrying capacity of both fetal and maternal blood. This affects maternal oxygenation by increased pulmonary venous admixture and diminishes the oxygen available to the fetus at the tissue level by its effect on fetal oxyhemoglobin dissociation.

In a 1975 report by Dow, Rooney, and Spence (11), a significantly greater rise in COHb concentration in response to smoking a single cigarette was shown in pregnant women (3.9 percent increase) as opposed to nonpregnant women (2.1 percent increase). This was more pronounced when anemia was present (5.0 percent increase) and appeared to be inversely related to the hemoglobin concentration. Three groups of women, all smokers, were selected for this study. The first group consisted of 10 normal, pregnant women late in the second trimester of pregnancy, with hemoglobin levels of over 11 g per 100 milliliters (ml). The second group consisted of 10 women also late in the second trimester but whose hemoglobin levels were less than 10 g/100 ml. Apart from anemia at the time of admission to the study, these patients were normal. The third group consisted of 10 normal, nonpregnant women with normal hemoglobin levels (over 11 g/100 ml). The change in COHb was estimated spectrophotometrically in response to smoking the first cigarette of the morning, the women having rested for at least 30 minutes. A sample of venous blood was withdrawn before and 2 minutes after smoking the cigarette. The cigarettes were of a standard size and of a "non-mild" (i.e., not low "tar" and nicotine) variety. The women were instructed to take a puff every 40 seconds, inhaling as deeply as possible, to a total of 10 puffs.

In the nonpregnant group, the mean rise in COHb concentration ($\pm$standard error of mean) was 2.1 ± 0.2 percent. A significantly greater increase was found in the normal pregnant group (mean rise 3.9 ± 0.4 percent; $t = 3.91$; $p < 0.005$). The effect was more pronounced in the anemic pregnant women, who had a meaning rise of 5.0 ± 0.2 percent ($t = 9.9$; $p < 0.0005$).

Longo (21) studied the effects of CO on oxygenation of the fetus in utero. Results showed that the partial pressure of oxygen in fetal blood decreases in proportion to the COHb concentrations in fetal and maternal blood (Figure 3).

This decrease in oxygen tension may be a factor in the low birth weight of infants born to women who smoke or are exposed to severe air pollution. These results suggest that significant increases in maternal and fetal COHb concentrations can significantly reduce oxygen delivery to the fetus.

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FIGURE 3.—Oxyhemoglobin saturation curves of human maternal and fetal blood under control and steady-state conditions*
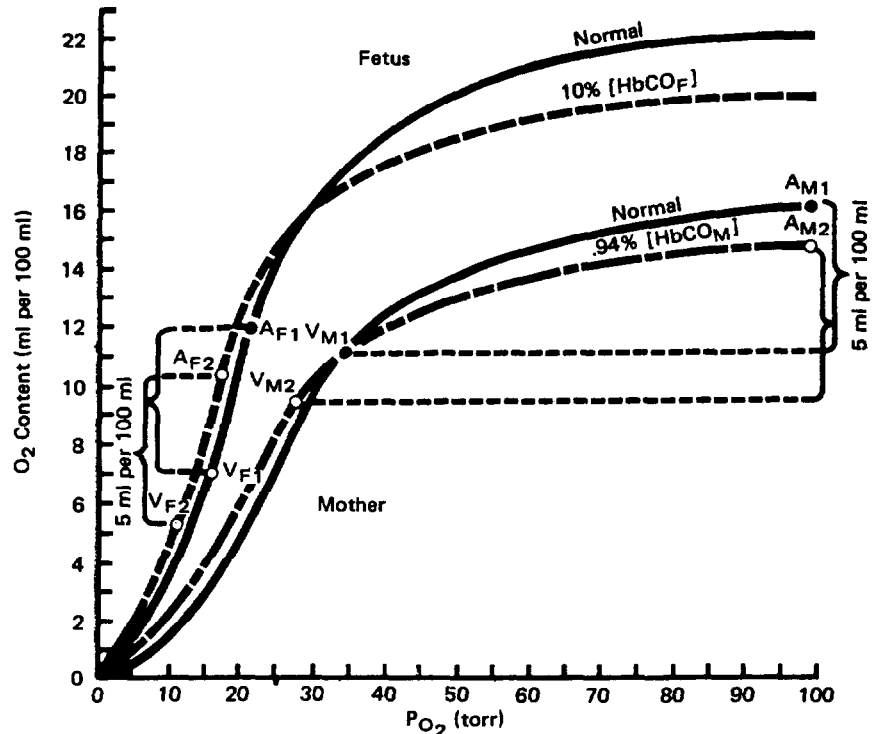
*With 10 percent fetal and 9.4 percent maternal HbCO concentrations. The maternal and fetal hemoglobin contents were assumed to equal 12 and 16.3 per 100 ml of blood, respectively. A normal O_2 consumption of 5 ml per 100 ml of blood was assumed for both the uterus and its contents and the fetus.

SOURCE: Longo, L., (21)

Astrup, et al. (3) carried out experimental studies on animals which may have a correlation with other data based on human studies in this report.

The investigation studied the effect of moderate CO exposure (180p.p.m. and 90p.p.m. CO in atmospheric air) on fetal development in rabbits. Exposure to 180p.p.m. CO (16-18 percent COHb) during pregnancy resulted in a 20 percent decrease in birth weight and a neonatal mortality rate of 35 percent as against 1 percent in the control group. Exposure to 90p.p.m. CO (8-9 percent COHb) had a less pronounced effect. There was a negative correlation between birth weight and maternal COHb concentration ($p < 0.05$). The authors conclude that these results indicate that CO in tobacco smoke might be responsible for the reduced birth weight of babies whose mothers smoke during pregnancy.

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A report from Denmark by Asmussen and Kjeldsen (2) studied the umbilical artery as a possible model for evaluating the vascular injury provoked by tobacco smoking in humans. Cords from newborn children delivered by 15 nonsmoking and 13 smoking mothers were studied in the transmission and the scanning electron microscope. The average weight of children born to smokers was 3,370 g and that of children born to nonsmokers was 3,695 g, a difference of 325 g. A difference of 123 g was found in the weights of the placentas.

Pronounced changes in the intima were found in the umbilical samples from smokers. The most important findings were degenerative changes in the endothelium, such as swelling, bleeding, contraction, and subsequent opening of the endothelial junctions, with formation of subendothelial edema. The basement membranes were considerably thickened. The smooth muscle cells in the ecematous subendothelial space often showed vacuolization. Since similar changes can be induced in arteries of animals by exposure to CO or perfusion with nicotine, the authors conclude that cigarette smoking is harmful to the vascular endothelium and may provide some rationale for the mechanism behind low birth weights and increased perinatal mortality.

SMOKING AND ITS EFFECTS ON CARDIOVASCULAR DISEASE AMONG WOMEN TAKING ORAL CONTRACEPTIVES

Smoking is a major cause of cardiovascular disease among women, and it has been found that the use of oral contraceptives potentiates its effect. Therefore, women who smoke and use oral contraceptives are at a much higher risk for cardiovascular disease and should be encouraged to stop smoking. In a review by Ory (30) of the original scientific data that exists on the association between oral contraceptives and myocardial infarction, cigarette smoking was found to be the most important factor in increasing the probability of women less than 50 years of age having myocardial infarction. Although this increased risk is independent of oral contraceptive use, oral contraceptive use appears to be an added risk factor. The use of these drugs in the absence of other predisposing factors appears to have only a small effect on increasing the risk of dying from myocardial infarction.

Jain (18) studied the risk of mortality associated with the use of oral contraceptives. For women 40-44 who neither use oral contraceptives nor smoke cigarettes, the overall mortality rate from myocardial infarction is 7.4 per 100,000 (Table 4). The comparable annual mortality rate among women of this age group who use oral contraceptives but do not smoke is 10.7 per 100,000. This compares to a rate of 62 per 100,000 for women who take oral contraceptives and smoke.

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TABLE 4. Estimated annual mortality rate per 100,000 women from mycardial infarction and thromboembolism, by use of oral contraceptives, smoking habits, and age (in years)

| | Myocardial Infarction | | | | Thromboembolism | | | |
| | Women Aged 30-39 | | Women Aged 40-44 | | Women Aged 20-34 | | Women Aged 35-44 | |
Smoking Habits	Users	Nonusers	Users	Nonusers	Users	Nonusers	Users	Nonusers
All Smokers	10.2	2.6	62.0	15.9	1.6	0.2	4.1	0.6
Heavy	13.0	5.1	78.7	31.3	4.4	0.2	11.4	0.6
Light	4.7	0.9	28.6	5.7	0.7	0.2	1.9	0.6
Nonsmokers	1.8	1.2	10.7	7.4	1.4	0.2	3.6	0.4
Smokers and Nonsmokers	5.4	1.9	32.8	11.7	1.5	0.2	3.9	0.5

*Estimated rates for smokers and nonsmokers were 0.24 and 0.16 respectively.
Rates appear the same because of rounding.

SOURCE: Jain, A.K. (18).

In a later study, Jain (17) analyzed the synergistic effect of smoking and the use of oral contraceptives on myocardial infarction. The relative risk of nonfatal myocardial infarction among those who use oral contraceptives and smoke is estimated to be 11.7 to 1 (Table 5). The authors suggest that smoking should be considered as another contraindication for the prescription of oral contraceptives.

TABLE 5. Estimated relative risks of nonfatal myocardial infarction, by use of oral contraceptives and cigarette smoking.

| | Current User of Oral Contraceptives | |
Smoking Data	Yes	No
Smokers		
Total	11.67	2.15
Heavy*	14.81	4.23
Light†	5.38	0.77
Nonsmokers	2.02	1.00

Based on data in Table VII by Mann and associates (25).

*Heavy smokers: at least 15 cigarettes per day.

†Light smokers: less than 15 cigarettes per day.

SOURCE: Jain, A.K. (17).

Results of a study by Beral (4) indicate that oral contraceptive users who smoke have a 10 times greater risk of dying from cardiovascular disease than women who neither smoke nor use the pill. Smoking by itself was responsible for a 4-fold increase in the risk of dying from cardiovascular diseases. Oral contraceptive use in the absence of smoking also appeared to increase one's risk, but the differences were not statistically significant.

Mann and his colleagues also studied tne relationships between smoking and myocardial infarction in women (23, 24). Their find-

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ings show an apparent but not a statistically significant increase in relative risk of nonfatal myocardial infarction for nonsmokers who use oral contraceptives (2.02, with a 95 percent confidence interval of 0.5 to 8.5). In contrast, for smokers who use oral contraceptives, the relative risk was estimated to be 11.67 compared to that of the nonsmoking, noncontraceptive user. In addition, these authors reported that the risk of nonfatal myocardial infarction was related to the amount smoked. It was found that in comparison with nonsmokers and ex-smokers, the relative risk of myocardial infarction increased significantly to 1.3 in women smoking fewer than 15 cigarettes a day, to 4.4 in women smoking 15 to 24 cigarettes a day, and to 11.9 in women smoking 25 or more cigarettes a day.

Among nonsmokers, oral contraceptive users have 2.0 (95 percent confidence interval, 0.5 to 8.5) times the risk of having a myocardial infarction. (Because the confidence interval includes 1.0, chance variation is a possible explanation for this finding.) Among smokers, if a woman uses oral contraceptives, she has 5.4 (95 percent confidence interval, 2.0 to 14.7) times the risk of having a myocardial infarction than if she is a nonuser. This result is highly statistically significant (p = 0.001).

EFFECTS OF CIGARETTE SMOKING ON LACTATION

Studies by Richer and Giudicelli (31), Rowan (32), and Vorherr (39), further document the effects of nicotine in breast milk on infants of smoking mothers. Since nicotine has been shown to cause nausea, vomiting, diarrhea, and tachycardia (38), it is recommended by the authors that lactating mothers refrain from smoking.

Bradt and Herrenkohl (5) studied the relationship between cigarette smoking and DDT in human milk. A total of 55 human milk samples from eastern Pennsylvania were studied. Ten of the donors were cigarette smokers, and they donated 13 of the milk samples. Results of the study showed that smoking was one of four variables which contributed to the increase in DDT. Mean total for the non-smoker was .101 units versus .146 units for smokers. Four factors were identified statistically as accounting for 54 percent of the variance on total DDT levels in human milk. These factors are: (1) number of children nursed; (2) number of cigarettes smoked daily; (3) use of nonpersistent pesticides; and (4) diet in calories. The relationship between the number of cigarettes smoked per day and the total amount of DDT in human milk suggests either that cigarette smoke may be a source of the human body burden of DDT or that cigarette smoke may cause more DDT to be excreted in the milk.

WHAT WOMEN KNOW ABOUT SMOKING AND PREGNANCY

There is much information circulating in the scientific community

regarding the effects of smoking on health in general and, specifically, on the outcome of pregnancy. In a survey conducted by the National Clearinghouse for Smoking and Health (37), an attempt was made to find out how successfully this information had been disseminated to the general population and particularly to women.

To what extent was the average woman informed about the consequences of her smoking on her own health and the health of her unborn child? The questions were designed to find out what women knew at the time of their last pregnancy (which in some cases was many years ago) and what they knew at the time of the survey.

At the time of their last pregnancy, 24 percent said they believed smoking was hazardous to the health of a pregnant woman, and 31 percent said they believed it harmed the developing fetus.

At the time of the survey in 1975, however, 53 percent reported that they knew smoking was harmful to a pregnant woman, and 60 percent believed it harmed the fetus.

It is clear that the level of knowledge among women about the effects of smoking on pregnancy is appreciably lower than that in the scientific community.

SUMMARY OF SMOKING-RELATED
PROBLEMS UNIQUE TO WOMEN

1. A strong, probably causal, association exists between cigarette smoking and higher late fetal and infant mortality among smokers' infants.

2. Perinatal mortality increases significantly with smoking as well as with other risk factors such as maternal age, parity, socioeconomic status, previous pregnancy history, and hemoglobin level.

3. A dose-response relationship exists between smoking and the incidence of low birth weight, preterm delivery, perinatal mortality, abruptio placentae, placenta previa, bleeding during pregnancy, and prolonged and premature rupture of the membranes.

4. In one study, the risk of premature rupture of membranes was more than three times greater for smokers than for nonsmokers among deliveries that occurred before 34 weeks gestation.

5. In another study, the risk of stillbirths by abruptio placentae was six times higher among smokers.

6. There is an association between smoking during pregnancy and impaired physical and intellectual development in the offspring.

7. COHb levels are substantially elevated in pregnant women who smoke and may result in damage to placental and fetal blood vessels.

8. Higher levels of COHb in both fetal and maternal blood may be a factor in the increased incidence of low-birth-weight babies among smokers.

9. The use of oral contraceptives potentiates the harmful effects of smoking on the cardiovascular system.

10. Results from one study showed that the relative risk of nonfatal myocardial infarction among women who use oral contraceptives and smoke is approximately 11.7 to 1.

11. Nicotine is present in the breast milk of lactating mothers who smoke and has been shown to cause nausea, vomiting, diarrhea, and tachycardia.

12. In one study, smoking was one of four variables which contributed to the increase of DDT in breast milk.

13. As recently as 1975, 40 percent of the women in the United States were not aware of the hazards to the developing fetus if they smoked during pregnancy.

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Chapter 2

Smoking and Overall Mortality

Contents

Chapter 2

Smoking and Overall Mortality

INTRODUCTION

In 1964, the subject of smoking and overall mortality was examined in the Report of the Advisory Committee to the Surgeon General of the Public Health Service (9). This subject was reviewed in 1967 and 1968 in *The Health Consequences of Smoking* (6,7). Since then, the updated results of three prospective, epidemiologic studies concerned with tobacco use and overall mortality have been published (1, 3, 5). The following is a review of work previously reported as well as an analysis of the three more recent studies.

Summary of the 1964 Report (9):
1. The death rate for male cigarette smokers was about 70 percent higher than that for nonsmokers.*
2. The death rates increased with the amount smoked.*
3. The ratio of the death rate of smokers to that of nonsmokers was highest at the earlier ages (40-50) and declined with increasing age.*
4. The mortality ratio was substantially higher for men who started smoking before the age of 20 than for men who started after 25.
5. The mortality ratio increased as the number of years of smoking increased.
6. In two studies which recorded the degree of inhalation, the mortality ratio for a given amount of cigarettes smoked was greater for inhalers than for noninhalers.
7. Cigarette smokers who had stopped smoking had mortality ratios of 1.4, compared to 1.7 for current cigarette smokers.
8. The mortality ratio declined as the number of years of cessation increased.
9. Death rates for men smoking less than five cigars daily were about the same as those of nonsmokers. For men smoking five or more cigars daily, death rates were slightly higher (9 to 27 percent) than those for nonsmokers. Death rates for ex-cigar smokers were higher than for current smokers in all four studies in which this comparison could be made. One possible explana-

*Data are derived from seven major prospective studies of male smokers and nonsmokers. The rate is for smokers of cigarettes only at the time of entry into the study. These are obtained by subtracting the yearly death rate for non-smokers from the death rate of a comparable group of smokers. This measure reflects the added probability of death in a 1-year period for the smoker over that for the nonsmoker.

tion may be that a substantial number of cigar smokers quit smoking due to illness.

10. Death rates for current pipe smokers were little if at all higher than for nonsmokers, even for those smoking 10 or more pipefuls per day and for those who had smoked for more than 30 years. Ex-pipe smokers, on the other hand, showed higher death rates than both nonsmokers and current smokers in four our of five studies. As similarly noted above, one possible explanation may be that a substantial number of cigar and pipe smokers quit smoking because of illness.

In the 1967 report of *The Health Consequences of Smoking*, additional conclusions were made relative to the effect of smoking on overall mortality (6). The highlights of that report are presented below:

1. The previous conclusions with respect to the association between smoking and mortality were both confirmed and strengthened.

2. With respect to effects of smoking on specific age groups, men 45 to 54 years of age were at greatest risk, both in terms of mortality ratios and excess deaths expressed as a percentage of total deaths. Nevertheless, although both of these measures declined with advancing age, the increment added to the death rate, which reflects one's personal chances of being affected, continued to increase with age.

3. Women who smoked cigarettes had significantly higher death rates than those who had never smoked regularly. The magnitude of the relationship varied with several measures of dosage. The same overall relationships between smoking and mortality were observed for women as for men, but at a lower level.

4. Previous findings on the lower death rates among those who had discontinued cigarette smoking were confirmed and strengthened by the additional data reviewed.

In 1968 report of *The Health Consequences of Smoking* (7) reported that the life expectancy for a two-pack-a-day or more smoker at age 25 is 8.3 years less than that for the corresponding nonsmoker. Even light smokers (those smoking less than 10 cigarettes per day) had 2.8 to 4.6 fewer years of life expectancy than corresponding nonsmokers.

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Overall mortality is a term familiar to epidemiologists and statisticians but one which is not commonly used or appreciated by many who are concerned with the health of the public. To many physicians, dentists, nurses, and other health professionals who have a primarily clinical orientation, the concept of overall mortality is often not clearly understood, since it has no immediate application to their practice. Individuals die of specific diseases. Disease-specific mortality rates are of more immediate interest to many in the health care field. Overall mortality rates are particularly useful in measuring the effect of agents which affect multiple organ systems and which are capable of causing or contributing to the cause of several diseases. In contrast, disease-specific mortality rates measure the effect of an agent on a specific cause of death but fail to measure the total impact of an agent on the public health. Overall mortality is, therefore, a good measure of the cumulative or total effect of an agent on health. The problem of how best to measure the relationship between smoking and mortality has been discussed in previous reports, as well as in some of the prospective study reports. A brief discussion of some of the measures of comparison available and their utility is presented below.

Mortality Ratios: These are obtained by dividing the death rate for a classification of smokers by the death rate of a comparable group of nonsmokers. A mortality ratio has been considered to reflect the degree to which a classification variable (e.g., smoking) identifies or may account for variations in death rates. As such, it is a measure of risk which indicates the relative effect of that variable on mortality, given that other important factors affecting mortality (e.g., age) are comparable in the numerator and denominator groups.

Differences In Mortality Rates: These are obtained by subtracting the yearly death rate for nonsmokers from the death rate of a comparable group of smokers. This measure reflects the added probability of death in a 1-year period for the smoker over that for the nonsmoker. As such, it is a measure of personal health significance, a means for the individual to estimate the added risk to which he is exposed.

Excess Deaths: These are obtained by subtracting from the number of deaths occurring in a group of smokers the number of deaths which would have occurred if that group of smokers had experienced the same mortality rates as a comparable group of nonsmokers. This measure is an indicator of the public health significance of the differences found, since it measures the number of people affected and therefore quantifies the magnitude of the

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problem for society as a whole.

Life Expectancy: This is a concept which is easier to understand than it is to calculate. At a given age, it represents the average number of years one might be expected to live. It identifies the point in time at which half the population in question theoretically will be dead and the other half will be alive.

DESCRIPTION OF THE STUDIES

The following is a brief description of the design and methods used in each of the three studies which are reported in this chapter. Some comments are made concerning the relative strengths and weaknesses of each study.

The American Cancer Society

The largest of the three studies discussed here is the American Cancer Society (ACS) Study (4, 8). In late 1959 and early 1960, volunteer workers of the ACS enrolled 1,078,894 men and women in a prospective study. Information was solicited on age, sex, race, education, place of residence, family history, past diseases, present physical complaints, occupation, occupational exposures, various smoking habits, and other factors. Information concerning smoking habits included: type of tobacco used, number of cigarettes smoked per day, inhalation practices, age at initiation of smoking, and the brand of cigarettes smoked from which the "tar" and nicotine content of the cigarette could be calculated. All segments of the population were included except migrant workers and similar groups that could not have been traced easily. Also excluded were mental patients and those receiving long-term medical care in institutions. Enrollment was by households, with the specification that there be at least one person over age 45 in each household enrolled. The study area covered 25 states. At the time of enrollment, each person completed a lengthy questionnaire. At 2-year intervals, for a period of 6 years, brief repeat questionnaires were administered to each surviving subject. In the follow-up questionnaires, information was obtained concerning current cigarette usage, hospitalization, diseases acquired in the interval between questionnaires, and several other items. Almost 95 percent of survivors were successfully traced the first 6 years, (that is, through June of 1966). In October 1971 and September 1972, further follow-up questionnaires were distributed to the nearly 900,000 individuals who had been last contacted in September 1965. Nearly 93 percent of the survivors were successfully followed for the entire 12 years. The time period from July 1, 1960, to June 30, 1966, is referred to as Period 1 and that from July 1, 1966, to June 30, 1972, is referred to as Period 2.

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The positive features of this study include its prospective design, the unusually large population enrolled, which included all major segments of society, the frequency of the follow-up periods, the variety of the data collected, the thoroughness of follow-up with loss of but few enrollees, and the relatively long period of observation.

The U.S. Veterans Study

The U.S. Veterans Study (4,5) was initiated by Dorn in 1954 and continued by Kahn and later by Rogot. This study describes the overall mortality experience of about 250,000 U.S. veterans who held Government Life Insurance policies in December of 1953. Beginning in January 1954, questionnaires on smoking habits were mailed to these policy holders and nearly 175,000 (68 percent) responded. These individuals comprise what in this report is called the "1954 cohort." In January 1957, a second questionnaire was mailed to those not responding in 1954, and an additional 50,000 replies were obtained, raising the response rate to 85 percent. These are referred to as the "1957 cohort." The annual probability of dying for the 1957 cohort was somewhat greater than that of the 1954 cohort. Because of this, the mortality experience of these two cohorts was examined separately. Only the data from the 1954 cohort will be considered here, as a separate analysis of both cohorts is beyond the scope of this paper. The study population was quite select; almost all policy holders were white males. Most were white-collar, skilled workers who were veterans of World War I. This group was questioned as to smoking habits, etc., and followed for 16 years. Since significant changes have occurred in the smoking practices of white males in the United States over the past 20 years, it is likely that similar changes also occurred in the smoking habits of the subjects of this particular study in the study period. It is unfortunate, therefore, that the recent mortality experience of this population has to be correlated with smoking practices of many years ago.

The strengths of this study include its large population, its prospective design, and its long period of follow-up. Its weaknesses include its narrow population, which limits the applicability of the results to the general population, and the lack of information about more recent changes in smoking habits among members of the study population which would affect the mortality experience of the group.

The British Doctors Study

In 1951, a total of 34,440 male British doctors responded to a questionnaire distributed by the British Medical Association relative to smoking habits (1). Nearly all of those enrolled were followed

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for a period of 20 years. Updated information concerning smoking practices was obtained in 1957, 1966, and 1972. More than 10,000 deaths occurred in this population in the period of observation. Information was obtained on the type of tobacco used, inhalation practice, the use of filter cigarettes, and the number of cigarettes smoked per day. The usual demographic data concerning the background of the individual were also obtained.

The strengths of this study include its large size, prospective design, the usually long period of follow-up, the frequent determination of smoking habits of the subjects enrolled in the study, and the thoroughness of follow-up. Perhaps the only significant drawback is that the study population was so narrow.

The most recent analysis has been limited to overall mortality, since death certificates were not obtained for those who died in the last half of the study period. Smoking classifications used in the latest paper are somewhat different from those used in previous reports. The occasional smoker was grouped with the nonsmoker, since their mortality experience was essentially similar. As a result, occasional smokers who had quit smoking were grouped with those who had never smoked, and regular smokers who became occasional smokers were grouped with ex-smokers.

OVERALL MORTALITY AND CIGARETTE SMOKING

Cigarette smoking as related to overall mortality was examined in these three studies using several different measures of dosage.

Number of Cigarettes Smoked

In the study of U.S. veterans, mortality increased with the number of cigarettes smoked per day. The mortality ratio was 1.25 for smokers of less than 10 cigarettes per day and increased to 1.89 for men smoking two packs (40 cigarettes) or more per day (Table 1). In the study of British doctors, the mortality ratio was 1.41 for smokers of 1-14 cigarettes per day and increased to 2.16 for smokers of 25 or more cigarettes per day. The mortality ratio for all

TABLE 1. Age-adjusted mortality ratios for male cigarette smokers, by amount smoked. U.S. Veterans Study, 1954 cohort, 16-year follow-up

Number of Cigarettes Smoked Per Day	Mortality Ratio
< 10	1.25
10-20	1.51
21-39	1.69
> 40	1.89
Nonsmokers	1.00
Total	1.55

cigarette smokers compared to nonsmokers was 1.63 (Table 2). The mortality experience of U.S. veterans by age and the number of cigarettes smoked per day are presented in Table 3. Cigarette smoking appears to have a stronger effect on the mortality of younger smokers than on older smokers. The death rate for smokers increases with age, but since the risk of dying in general increases more rapidly with advancing age than the risk associated with smoking, the relative contribution of cigarette smoking to overall mortality decreases with time. This relationship is imperfectly demonstrated when mortality ratios are used.

TABLE 2. Mortality ratios for cigarette smokers, by number of cigarettes smoked per day, British Doctors Study

Number of Cigarettes Smoked Per Day	Mortality Ratio
Mixed (Cigarette / other)	1.21
1-14	1.41
15-24	1.57
>25	2.16
Nonsmokers	1.00
Total	1.63

TABLE 3. Mortality ratios for male cigarette smokers, by age and number of cigarettes smoked per day, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Number of Cigarettes Smoked Per Day	Age				
	30-34	35-44	45-54	55-64	65-74
None	1.00	1.00	1.00	1.00	1.00
< 10	1.94*	1.44	1.44	1.20	1.15
10-20	1.27	1.79	1.64	1.49	1.30
21-39	1.76	2.23	2.10	1.67	1.42
> 40	2.33**	2.72	2.13	1.86	1.65
Total	1.52	1.95	1.83	1.53	1.32

*This figure is calculated on the basis of 140 individuals and nine deaths, which is why it may appear to be somewhat unstable.

**This figure is calculated from 68 individuals and five deaths.

Age Began Smoking

The earlier one begins smoking, the more exposure that individual will have had to cigarette smoke at any subsequent age. In the U.S. Veterans Study, the overall mortality ratio for those men who began smoking before the age of 15 was 1.86. This decreased to 1.32 for those who did not start smoking until after the age of 25 (Table 4). Table 5 presents the mortality ratios for males by number of cigarettes smoked per day and age began smoking. The lowest mortality ratio (1.36) was experienced by those men who smoked fewer

than 21 cigarettes per day and who were more than 20 years old when they began smoking. The highest mortality ratio (1.82) occurred among those who smoked more than 21 cigarettes per day and began smoking before the age of 20.

TABLE 4. Age adjusted mortality ratios for male cigarette smokers, by age began smoking, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Age Began Smoking (Years)	Mortality Ratio
< 15	1.86
15-19	1.64
20-24	1.51
> 25	1.32
Nonsmokers	1.00
Total	1.55

TABLE 5. Age-adjusted mortality ratios for male cigarette smokers, by number of cigarettes smoked per day and age began smoking, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Number of Cigarettes Smoked Per Day	Age Began Smoking (Years)	Mortality Ratio
< 21	> 20	1.36
< 21	< 20	1.56
> 21	> 20	1.59
> 21	< 20	1.82
Nonsmokers	—	1.00

Inhalation Practice

Death rates by inhalation practice were examined in the study of British doctors (Table 6). The mortality ratio for those who did not inhale was 1.28. This increased to 1.43 for those who did inhale.

TABLE 6. Mortality ratios for cigarette smokers, by inhalation practice, British Doctors Study

Inhalation Practice	Mortality Ratio
Smokers Who Inhaled	1.43
Smokers Who Did Not Inhale	1.28
Nonsmokers	1.00

"Tar" and Nicotine

The "tar" and nicotine content of cigarette smoke in relation to overall mortality was examined by Hammond, et al. (3) using the ACS data. Several important issues relative to the concept of less hazardous smoking were settled in this study. It has been generally accepted that the harmful effects of cigarette smoking

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are proportional to the "tar" and nicotine levels delivered by the cigarette. For several years, the "tar" and nicotine levels of all the popular brands of cigarettes have been checked periodically by the Federal Trade Commission. This information has been made available to the public through various public and private agencies and is included in cigarette advertisements. Those who have decided not to quit or who have not been able to quit have been encouraged to switch to brands of cigarettes which deliver less "tar" and nicotine. This pattern of smoking is thought to be one way of partially reducing the risks associated with smoking. Some persons in the scientific community have questioned whether or not there would be any substantial reduction in risk of mortality associated with such a switch. Smokers might increase the number of cigarettes smoked per day, thus keeping their intake of "tar" and nicotine relatively constant. Smokers switching to low "tar" and nicotine cigarettes may inhale the smoke more deeply into the lungs, thus tending to maintain a similar exposure to the toxic elements in the smoke.

In the study by Hammond, et al. (3), "tar" and nicotine (T/N) levels were defined as follows: High T/N: 25.8-35.7 milligrams (mg.) "tar" and 2.0-2.7 mg. nicotine; Medium T/N: 17.6-25.7 mg. "tar" and 1.2-1.9 mg. nicotine; Low T/N: less than 17.6 mg. "tar"

TABLE 7. Mortality ratios for all cigarette smokers in two time periods, by sex and "tar" and nicotine (T/N) content of cigarettes smoked*

| Sex | Period | Mortality Ratio | | |
		High T/N	Medium T/N	Low T/N
Male	1	1.00	0.90	0.88
Male	2	1.00	0.98	0.81
Female	1	1.00	0.89	0.84
Female	2	1.00	0.87	0.82
Total		1.00	0.91	0.84

SOURCE: Hammond E.C., et al. (3).

*A matched-group analysis adjusted for several factors. See text.

and less than 1.2 mg. nicotine. A matched group analysis was utilized. Subjects within each group were matched for: (1) age, (2) race, (3) number of cigarettes smoked per day, (4) age began smoking, (5) place of residence (urban or rural), (6) history of hazardous occupational exposure, (7) education, (8) history of lung cancer, and (9) history of heart disease. Matching was done separately for men and women in both time periods of the study. Within each matched group, the subjects were divided into three subgroups according to "tar" and nicotine (high, medium, or low). The entire group was discarded if it did not contain at least one subject in each "tar" and nicotine category. The adjusted number of subjects in Period 1

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was 14,688 men and 30,176 women. In Period 2, there were 6,475 men and 15,342 women. The mean age of subjects in Period 1 was 53.6 years for men and 51.6 years for women; in Period 2, the mean age was 58.4 years for men and 56.7 years for women.

Table 7 shows mortality ratios by sex and "tar" and nicotine content of the cigarettes smoked. In this instance, the mortality ratio for the high T/N smokers is represented as 1.00. There is a small but significant ($p< 0.0005$) reduction in the risk of dying with the use of lower T/N cigarettes. The mortality ratio was reduced to 0.91 for the medium T/N smokers and was further reduced to 0.84 for the low T/N smokers. A comparison was also made between the mortality experience of low T/N smokers and nonsmokers. Subjects were matched according to the same factors as the previous analysis with the exception of the number of cigarettes smoked per day. The adjusted number of subjects for Period 1 was 15,346 men and 32,702 women. For Period 2, adjusted numbers were 6,822 and 16,803 for men and women, respectively. The mean age of subjects in Period 1 was 53.8 years for men and 52.3 years for women. In Period 2, the mean ages for men and women were 58.7 and 57.3 years, respectively. The mortality ratios for these matched groups are presented in Table 8. The death ratio for the low T/N group is 1.00, and that for nonsmokers is 0.66. The mortality ratio for the low T/N group is, therefore, approximately 50 percent higher than that for the nonsmokers.

TABLE 8. Mortality ratios for smokers of low "tar" and nicotine (T/N) cigarettes and nonsmokers in two time periods, by sex

Sex	Period	Mortality Ratio	
		Low T/N	Nonsmokers
Male	1	1.00	0.57
Male	2	1.00	0.64
Female	1	1.00	0.76
Female	2	1.00	0.71
Total		1.00	0.66

SOURCE: Hammond E.C., et al. (3).

*A matched-group analysis adjusted for several factors. See text.

Assuming that the composition of the two low T/N groups was quite similar in these separate analyses, whese two sets of data can be combined to compare mortality rates of smokers of various levels of "tar" and nicotine with those of nonsmokers (Table 9). These results are approximate, however, and are subject to some error.

Another matched group analysis was done comparing mortality ratios of smokers of relatively few (1-19) high T/N cigar-

TABLE 9. Mortality ratios for all cigarette smokers and nonsmokers in two time periods, by sex and "tar" and nicotine (T/N) content of cigarettes smoked

Sex	Period	Nonsmokers	Mortality Ratio		
			Low T/N	Medium T/N	High T/N
Male	1	1.00	1.75	1.80	2.00
Male	2	1.00	1.56	1.89	1.92
Female	1	1.00	1.32	1.40	1.57
Female	2	1.00	1.41	1.49	1.73
Total		1.00	1.52	1.64	1.80

SOURCE: Hammond E.C., et al. (3).

ettes with those smokers of relatively large numbers (20-39) of low T/N cigarettes. The mortality ratios of these two groups were very similar, and the difference between them was not statistically significant.

EX-SMOKERS

The mortality experience of ex-smokers is a subject in which there has been increasing interest in the past several years. When the harmful effects of smoking were initially suspected and examined, the question at first was one of the magnitude of the problem. More recently, there has been a nationwide recognition of the adverse morbidity and mortality which results from smoking. As a result, more than 30 million Americans have quit smoking, and millions more anticipate quitting within the next several years. One of the questions of greatest concern to the smoker at this time is not, "How bad is my smoking for my health?" but rather, "After all these years of smoking will it make any difference if I quit?" The benefits of stopping smoking are more clearly understood as a result of the studies reviewed here.

The relationship between cessation of smoking and overall mortality was examined in considerable detail in the study of U.S. veterans. A differentiation was made between ex-smokers who stopped smoking on the recommendation of a doctor and those who quit for other reasons (Tables 10, 11, 12). In each cohort, about 10 percent of the ex-smokers had stopped on doctor's orders, and this group had much higher mortality levels than those who stopped for other reasons. There was a direct relationship between mortality levels and the maximum amount previously smoked, an inverse relationship between mortality and years since stopping smoking, and an inverse relationship between mortality and age when smoking began.

The combined effects of these three factors on mortality are presented in Table 13. The lowest mortality ratio (1.03) was experienced by ex-smokers who began smoking after the age of 20,

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smoked fewer than 21 cigarettes per day, and had stopped smoking for more than 10 years at the time of enrollment in the study. Conversely, the highest mortality ratio (1.45) was experiened by ex-smokers who began smoking before the age of 20, smoked more than 21 cigarettes per day, and had stopped smoking for less than 10 years at the time of enrollment in the study.

TABLE 10. Mortality ratios for ex-smokers who quit smoking on doctor's orders and for other reasons, by years since stopping, U.S. Veterans Study, 1954 cohort, 16-year follow-up

| Years Since Stopping | Mortality Ratio | |
	Quit on Doctor's Orders	Quit for Other Reasons
< 5	1.55	1.23
5–9	1.43	1.23
10–14	1.77	1.14
15–19	1.35	1.04
> 20	1.16	1.06
Total	1.52	1.18

TABLE 11. Mortality ratios for ex-smokers who quit smoking on doctor's orders and for other reasons, by number of cigarettes smoked per day, U.S. Veterans Study, 1954 cohort, 16-year follow-up

| Number of Cigarettes Smoked Per Day | Mortality Ratio | |
	Quit on Doctor's Orders	Quit for Other Reasons
< 10	1.42	1.00
10–20	1.48	1.17
21–29	1.53	1.30
> 40	1.60	1.32
Total	1.52	1.18

TABLE 12. Mortality ratios for ex-smokers who quit smoking on doctor's orders and for other reasons, by age began smoking, U.S. Veterans Study, 1954 cohort, 16-year follow-up

| Age Began Smoking (Years) | Mortality Ratio | |
	Quit on Doctor's Orders	Quit for Other Reasons
< 15	1.59	1.36
15–19	1.55	1.20
20–24	1.49	1.12
> 25	1.34	1.15
Total	1.52	1.18

A detailed study of the mortality experience of ex-smokers who stopped smoking for various reasons other than a doctor's order

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TABLE 13. Mortality ratios for ex-smokers of cigarettes only, by years since Stopping, number of cigarettes smoked per day, and age began smoking, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Years Since Stopping	Number of Cigarettes Smoked Per Day	Age Began Smoking (Years)	Mortality Ratio
< 10	> 21	< 20	1.45
< 10	> 21	> 20	1.27
< 10	< 21	< 20	1.21
< 10	< 21	> 20	1.12
> 10	> 21	< 20	1.19
> 10	> 21	> 20	1.07
> 10	< 21	< 20	1.08
> 10	< 21	> 20	1.03
Nonsmokers			1.00
Total			1.18

is given in Figures 1-4. This information is derived from the U.S. Veterans Study for men aged 55-64 who used to smoke from

FIGURE 1.—Annual probability of dying for current cigarette smokers, ex-smokers who quit less than 5 years ago, and never smokers, ages 55–64*

*U.S. Veterans Study, 1954 cohort, 16-year follow-up.

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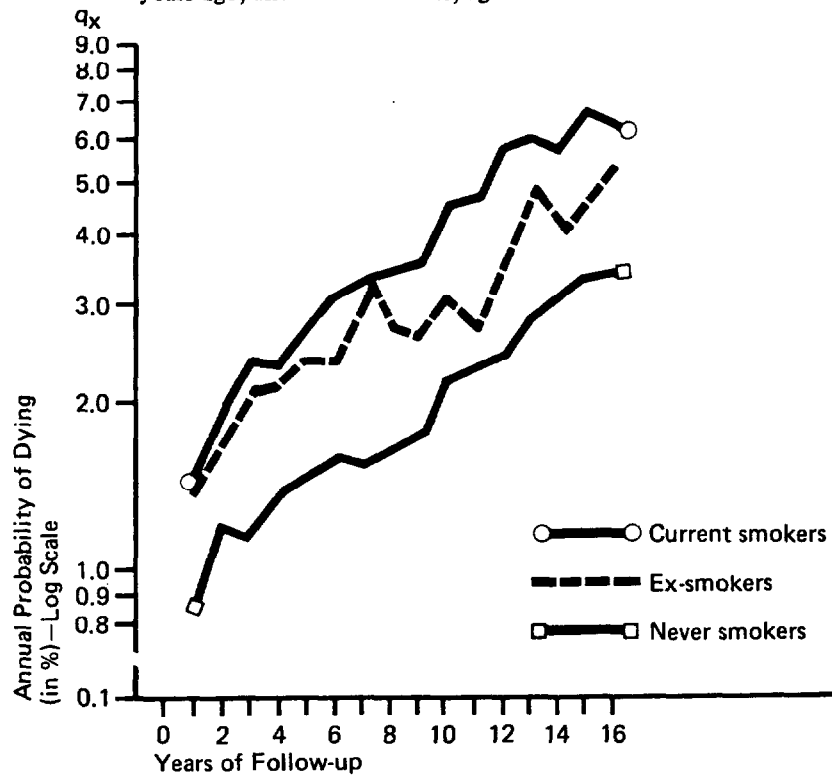
FIGURE 2.—Annual probability of dying for current cigarette smokers, ex-smokers who quit 5-9 years ago, and never smokers, ages 55-64*
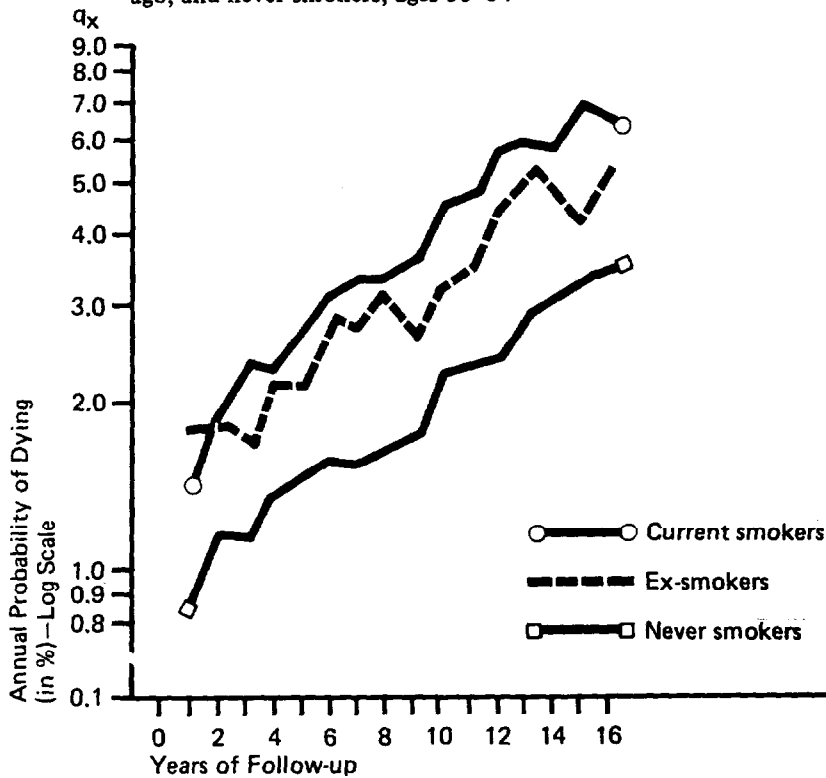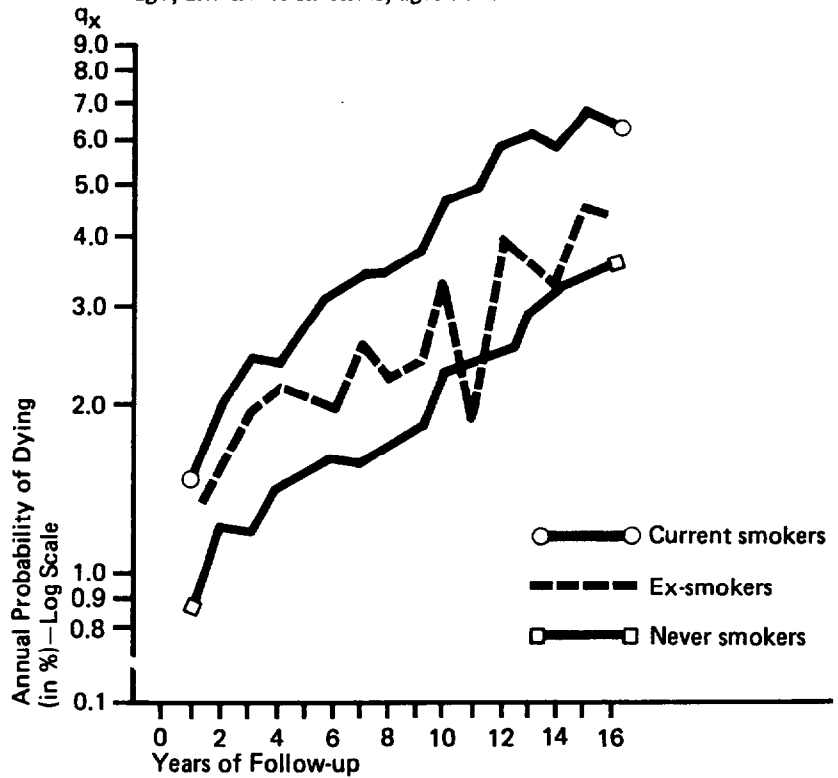

*U.S. Veterans Study, 1954 cohort, 16-year follow-up.

21-39 cigarettes per day. The years since stopping smoking is considered as a variable, and the mortality rates are compared with those of current cigarette smokers and nonsmokers. Annual probabilities of dying are plotted on a logarithmic scale. This results in a fairly smooth, linear pattern for both smokers and nonsmokers. The positive slope indicates increasing mortality with the passing of time of both smokers and nonsmokers. These lines also appear to run parallel or perhaps diverge slightly. This indicates an approximately constant or slightly increasing excess probability of dying between cigarette smokers and nonsmokers over the 16-year period. For ex-smokers who quit less than 5 years prior to the beginning of the study, the probability of dying is at first nearly identical to that of smokers (Figure 1). Over the years, the probability gradually falls to a position approximately halfway between that of smokers and nonsmokers. Figures 2 and 3 show that with longer

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periods of cessation the probability of dying more nearly approaches that of nonsmokers. The probability of dying for ex-smokers who had stopped smoking for 15 or more years is virtually the same as that for nonsmokers for the entire 16-year period (Figure 4).

The mortality experience of British doctors who quit smoking indicates that there are benefits to quitting no matter how long one has smoked (Table 14). After 10-15 years of not smoking, the risk of dying for ex-smokers is similar to that of those who have never smoked (1.1 compared to 1.0). It should be remembered that overall mortality examines the probability of dying from all causes. This masks the relative benefits of quitting for specific diseases. It is known that the risk of dying from ischemic heart disease is reduced almost immediately after cessation of smoking,

FIGURE 3.—Annual probability of dying for current cigarette smokers, ex-smokers who quit 10-14 years ago, and never smokers, ages 55-64*

*U.S. Veterans Study, 1954 cohort, 16-year follow-up.

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FIGURE 4.—Annual probability of dying for current cigarette smokers, ex-smokers who quit more than 15 years ago, and never smokers, ages 55-64*
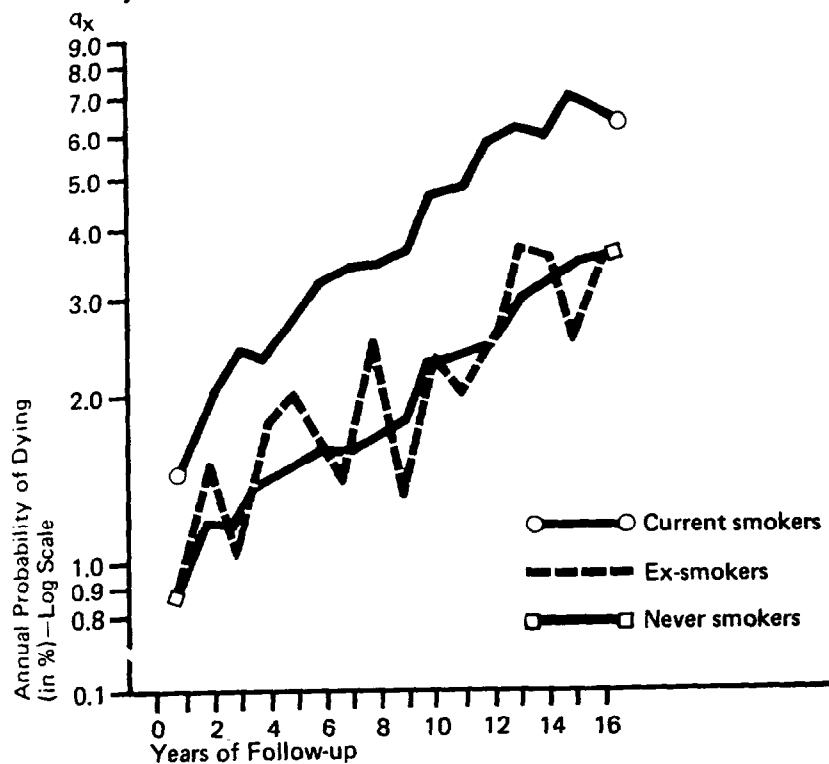
*U.S. Veterans Study, 1954 cohort, 16-year follow-up.

while the risk of dying from lung cancer decreases more slowly. Only the net or total effect is demonstrated in overall mortality figures.

TABLE 14. Mortality ratios for ex-smokers compared to nonsmokers, by number of years since stopping and age, British Doctors Study

Years Since Stopping	Mortality Ratio		
	Age 30-64	Age >65	All Ages
0*	2.0	1.6	1.8
1-4	1.7	1.4	1.5
5-9	1.6	1.4	1.5
10-14	1.4	1.2	1.3
>15	1.1	1.1	1.1
Nonsmokers	1.0	1.0	1.0

*Current Smokers

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Pipe and cigar smoking as related to overall and specific causes of mortality was last reviewed in the 1973 report of *The Health Consequences of Smoking* (8). The combustion products of pipe and cigar smoke contain many of the same chemical compounds found in cigarette smoke condensate. Since pipe and cigar smokers are less likely to inhale than cigarette smokers, they experience much lower mortality from certain diseases strongly associated with cigarette smoking. These include lung cancer, ischemic heart disease, and chronic obstructive lung disease. They do have death rates that are virtually similar to those for cigarette smokers, however, for cancers of the oral cavity, pharynx, larynx, and esophagus.

It should not be inferred from the above that switching to a pipe or cigar will necessarily reduce the mortality risks experienced by a current cigarette smoker, particularly one who inhales. The reason for this is that a cigarette smoker who inhales would probably continue to inhale after switching (8). Lower risks for pipe and cigar smokers may be associated with the lower prevalence of inhalation among these smokers and not with less hazardous tobacco products.

TABLE 15. Age-adjusted mortality ratios for pipe-only, cigar-only, and cigarette–only smokers, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Type of Tobacco	Mortality Ratio
Pipe Only	1.07
Cigar Only	1.16
Cigarettes Only	1.55
Nonsmokers	1.00

The U.S. Veterans Study contains the most detailed information concerning the mortality experience of pipe and cigar smokers. The mortality ratios for both pipe and cigar smokers are predictably greater than those for nonsmokers, and they are less than the mortality ratios of cigarette smokers (Table 15). Significant dose-response relationships were demonstrated for both pipe and cigar smokers by amount smoked and age began smoking.

The mortality ratio for cigar smokers increased from 1.11 for those smoking 1-2 cigars per day to 1.39 for those smoking nine or more cigars per day (Table 16). The mortality ratio was 1.13 for

TABLE 16. Age-adjusted mortality ratios for current cigar smokers, by number of cigars smoked per day, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Number of Cigars Smoked Per Day	Mortality Ratio
1-2	1.11
3-4	1.13
5-8	1.22
>9	1.39
Nonsmokers	1.00
Total	1.16

those who began smoking after the age of 25 and 1.22 for those who began smoking before the age of 15 (Table 17). Table 18 combines these variables and shows that the lowest mortality ratio for cigar-only smokers is 1.07 for those who smoked less than five cigars per day and began smoking after the age of 25. The highest mortality ratio of 1.28 was experienced by those who smoked more than five cigars per day and began smoking before the age of 25.

Somewhat similar dose-response relationships were demonstrated for pipe-only smokers; however, the risk associated with pipe smoking is slightly less than that with cigar smoking (Tables 19, 20, and 21).

TABLE 17. Age adjusted mortality ratios for current cigar smokers, by age began smoking, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Age Began Smoking (Years)	Mortality Ratio
< 15	1.22
15-19	1.23
20-24	1.16
> 25	1.13
Nonsmokers	1.00
Total	1.16

TABLE 18. Age-adjusted mortality ratios for current cigar smokers, by number of cigars smoked per day and age began smoking, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Number of Cigars Smoked Per Day	Age Began Smoking (Years)	Mortality Ratio
< 5	> 25	1.07
< 5	< 25	1.16
> 5	> 25	1.28
> 5	< 25	1.23
Nonsmokers		1.00
Total		1.16

TABLE 19. Age-adjusted mortality ratios for current pipe smokers, by number of pipefuls smoked per day, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Number of Pipefuls Smoked Per Day	Mortality Ratio
< 5	0.93
5-9	1.12
10-19	1.08
> 20	1.21
Nonsmokers	1.00
Total	1.07

TABLE 20. Age-adjusted mortality ratios for current pipe smokers, by age began smoking, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Age Began Smoking (Years)	Mortality Ratio
< 15	1.04
15-19	1.12
20-24	1.06
> 25	1.06
Nonsmokers	1.00
Total	1.07

TABLE 21. Age-adjusted mortality ratios for current pipe smokers, by number of pipefuls smoked per day and age began smoking, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Number of Pipefuls Smoked Per Day	Age Began Smoking (Years)	Mortality Ratio
< 10	> 25	1.03
< 10	< 25	1.05
>10	> 25	1.12
> 10	>25	1.12
Total		1.07

The above discussion relates to those who have limited their lifetime smoking to cigars only or pipes only. Frequently, however, a smoker will have used tobacco in several different forms. For instance, a cigar smoker may be a former cigarette smoker and may occasionally smoke pipes. The U.S. Veterans Study contains data on the mortality ratios of individuals who use tobacco in various forms. These data have been arranged so that the various patterns of smoking are arranged by increasing risk of mortality. Table 22 shows the age-adjusted mortality ratios of current cigar smokers who have or are using pipes and/or cigarettes. Smoking cigarettes and cigars is more risky, and smoking pipes and cigars is less risky than smoking cigars alone.

TABLE 22. Age-adjusted mortality ratios for current cigar smokers, by use of other types of tobacco, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Type of Tobacco Used		Mortality Ratio
Cigarettes	Pipes	
Never	Never	1.16
Never	Current	1.10
Never	Former	1.10
Former	Former	1.10
Former	Current	1.13
Former	Never	1.23
Current	Current	1.21
Current	Never	1.30
Current	Former	1.33

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TABLE 23. Age-adjusted mortality ratios for current pipe smokers, by use of other types of tobacco, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Type of Tobacco Used		
Cigarettes	Cigars	Mortality Ratio
Never	Never	1.07
Never	Current	1.10
Never	Former	1.11
Former	Former	1.14
Former	Current	1.14
Former	Never	1.10
Current	Current	1.21
Current	Never	1.28
Current	Former	1.36

The mortality experience of pipe smokers is shown in Table 23. Pipe smoking alone is the least hazardous form of smoking. The combination of pipes and cigars is a less risky combination than the combination of pipes and cigarettes. It is interesting to note that when the pipe smoker divides his smoking three ways and uses both cigarettes and cigars in addition to pipe smoking, the mortality ratio is less than if the time devoted to smoking is split two ways between pipes and cigarettes. Evidently to the extent that cigarettes are replaced there is a reduction in risk. The mortality ratios of current cigarette smokers who have or are using pipes or cigars is shown in Table 24.

TABLE 24. Age-adjusted mortality ratios for current cigarette smokers, by use of other types of tobacco, U.S. Veterans Study, 1954 cohort, 16-year follow-up

Type of Tobacco Used		
Cigarettes	Cigars	Mortality Ratio
Never	Never	1.55
Never	Current	1.28
Never	Former	1.47
Former	Former	1.48
Former	Current	1.36
Former	Never	1.53
Current	Current	1.21
Current	Never	1.30
Current	Former	1.33

In the study of British doctors, Doll and Peto (1) reported that those who smoked only pipes or cigars experienced mortality rates which were similar to, or only slightly above, those of men who did not smoke at all. Pipe and cigar smokers who also used cigarettes had mortality ratios which were intermediate between those who only smoked pipes and cigars and those who smoked cigarettes. These figures are presented in Table 25.

TABLE 25. Age-adjusted mortality ratios for all smokers, by type of tobacco used, British Doctors Study

Type of Tobacco Used	Mortality Ratio
Pipe or Cigar	
Never Cigarettes	1.09
Pipe or Cigar	
and Cigarettes	1.31
Cigarettes Only	1.73
Nonsmokers	1.00

1. Overall mortality rates for cigarette smokers are about 70 percent higher than those for nonsmokers.

2. Overall mortality risk increases with the amount smoked. For the two-pack-a-day cigarette smoker, the risk of premature death is approximately twice that of the nonsmoker.

3. Overall mortality ratios of smokers compared to nonsmokers are highest at earlier ages and decline with increasing age. For cigarette smokers, the risk of premature death is twice that of nonsmokers at age 40.

4. Overall mortality ratios are higher for those who begin smoking at a young age compared to those who begin later. For those who begin smoking before the age of 15, the risk of premature death is about 86 percent higher than that for nonsmokers.

5. Overall mortality ratios are higher for those smokers who inhale than for those who do not.

6. There is about a 15 percent reduction in overall mortality risk for smokers of low "tar" and nicotine cigarettes (less than 17.6 mg. "tar" and less than 1.2 mg. nicotine) compared to those who smoke high "tar" and nicotine cigarettes (25.8-35.7 mg. "tar" and 2.0-2.7 mg. nicotine).

7. Overall mortality rates of low "tar" and nicotine cigarette smokers are about 50 percent higher than for nonsmokers.

8. Overall mortality rates of former smokers decline as the number of years of cessation increase. After 15 years off cigarettes, death rates for former smokers are nearly identical to those of nonsmokers.

9. Overall mortality rates of former smokers are directly proportional to the number of cigarettes the person used to smoke.

10. Overall mortality rates of former smokers are inversely proportional to the age at which the person began smoking.

11. Regardless of length of time smoked or number of cigarettes smoked, former smokers have lower mortality rates than continuing smokers, provided they are not ill at the time of cessation.

12. Overall mortality ratios for cigar smokers are somewhat higher than for nonsmokers. The U.S. Veterans Study showed a mortality ratio of 1.16, compared to 1.0 for nonsmokers. The overall mortality ratio was 39 percent higher than the ratio in nonsmokers for men smoking nine or more cigars a day. A positive dose-response relationship exists between cigar smoking and mortality.

13. Overall mortality ratios for male cigar smokers are inversely proportional to the age at which the individual began smoking.

14. Overall mortality ratios for pipe smokers are only slightly higher than for nonsmokers. The mortality ratio in the U.S. Veterans Study was 1.07. Overall mortality ratios were 21 percent

higher than nonsmokers for men who smoked 20 or more pipefuls a day than for nonsmokers. A positive dose-response relationship exists between pipe smoking and mortality.

15. Overall mortality ratios of men who smoke cigarettes in combination with pipes and/or cigars are intermediate between those who smoke pipes or cigars only and those who smoke cigarettes only. Cigarette smokers who also smoke cigars or pipes have overall mortality rates approximately 30 percent higher than nonsmokers.

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Appendix

Smoking and Disease
— What Must Be Done

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HOW SMOKING CAUSES DISEASE

Since the early 1950s, when cigarette smoking was first implicated as a major cause of lung cancer in men, further research into the relationship between smoking and ill health has provided substantial additional data that support various theories about the mechanisms caused or enhanced by smoking, with regard to both mortality and morbidity. The following five mechanisms have been proposed:

(1) Cigarette smoking starts a disease process that progressively produces irreversible damage, the end-effect of which is more or less proportional to the total dosage accumulated during the years of smoking. Cessation of smoking leaves the individual with functional impairments that neither improve appreciably nor, of themselves, continue to deteriorate—except perhaps as a result of aging or exposure to other harmful agents. Owing to the interference with normal mechanisms for clearing the respiratory tract and the destruction of peripheral airways, this kind of process probably accounts for the high rates of chronic obstructive lung disease in cigarette smokers. A similar process seems to explain the high levels of atherosclerosis found in cigarette smokers; the almost continuously elevated level of carboxyhaemoglobin found in the blood of moderate to heavy smokers interacts with high levels of cholesterol to produce increased formation of atherosclerotic plaques.

(2) Cigarette smoking starts a disease process characterized by continual repair and recovery until a critical point is reached when the process is no longer reversible. The total effect is related to cumulative exposure over the years, so that several short periods of heavy smoking could lead to the point of irreversibility. Unless this point has been reached, cessation of smoking results in a rapid decrease in risk. A mechanism of this kind probably accounts for both the high dose-response relationship in lung cancer and the rapid relative reduction in risk of lung cancer among populations of ex-smokers. Other sites of cancer related to cigarette smoking probably also react in this way, which would correlate with the evidence that tobacco smoke contains both cancer-initiating and cancer-promoting substances.

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(3) Cigarette smoking promotes rather than starts the disease process, either by directly supporting a developing pathological condition or by diminshing the body's normal capacity to defend itself against disease. By this mechanism, cigarette smoking could promote a subclinical disease to a clinically recognizable state or a mild disease to a more severe form or even increase the fatality rates of certain diseases. This mechanism might account for the slightly increased mortality rates for influenza or tuberculosis among smokers, although cigarette smoking itself is not the cause of these diseases. Furthermore, unless severe chronic obstructive lung disease or high levels of atherosclerosis have already developed, stopping smoking both lessens the severity of heart attacks and improves the recovery rates from them.

(4) Cigarette smoking induces temporary conditions favouring a critical combination of events, which leads to disease, disability, and possible fatal consequences. For example, there is substantial evidence to support a theory that each cigarette can increase the probability of myocardial damage. This comes about through an increased demand for oxygen in response to the nicotine in cigarette smoke, at the same time that the carbon monoxide in the smoke has decreased the supply of oxygen by raising the carboxy-haemoglobin levels in the blood. Once this imbalance of supply and demand for oxygen is alleviated, the probability of myocardial damage would presumably revert to normal levels; in this instance, stopping smoking should bring about an almost immediate and sharp decline both in associated morbidity and mortality.

(5) Cigarette smoking may be artificially related to excess disability or death because of a close association with some other condition, which occurs at a high level in smokers and is itself responsible for the disease. The generally accepted example of this mechanism at work is cirrhosis of the liver. Because many heavy drinkers of alcohol are also heavy smokers, the high rate of cirrhosis among cigarette smokers has sometimes been attributed to smoking. Some evidence does suggest that high levels of exposure to both cigarette smoking and alcohol produce an effect greater than that for alcohol exposure alone.

Implications for action

The different mechanisms described above are important for the evaluation of potential public health benefits that could result from programmes aimed at (i) inducing smokers to stop smoking, (ii) dissuading young nonsmokers from starting to smoke, or (iii) changing the ingredients of cigarettes to make their smoke less harmful. For some types of associated morbidity and mortality there would be no benefits from any of these actions; for others, rather small benefits or substantial benefits taking place rather

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rapidly or substantial benefits taking place slowly over a long period of time. For example, the greatest long-term benefits would result from dissuading youngsters from taking up smoking, but more immediate, albeit smaller, benefits could be derived from persuading adults to stop smoking, provided the programme reaches many of the individuals at greatest risk.

In addition to taking these mechanisms into account in designing control efforts, there are certain epidemiological findings of special importance in this respect. First and foremost is the evidence that cigarette smoking seems to act in concert with many other risk factors so that the combined risk for almost any disease, on which cigarette smoking by itself has an effect, is sharply increased. For example, the radioactivity to which uranium miners under current mining conditions are exposed appears to have relatively little effect on lung cancer rates among nonsmokers; for smokers, however, it appears to produce far higher lung cancer rates than those for smokers who are not exposed to radioactivity.

Similarly, certain forms of chronic obstructive lung disease caused by sustained inhaling of particles and fibres are more common and severe in those who smoke. This applies equally to byssinosis (caused by inhaling cotton fibres) and the fungus-produced respiratory problems experienced by pigeon breeders. Both smokers and nonsmokers exposed to asbestos fibres show elevated rates of asbestosis, but only the smokers manifest extremely high rates of lung cancer. With ischaemic heart disease also, cigarette smoking appears to combine with other generally accepted risk factors, i.e., hypertension and hypercholesterolaemia, to produce a multiplicative, rather than simply an additive effect. On the other hand, there is evidence to suggest that certain endogenous factors, such as inherited susceptibility, can have an effect opposite to that of the exogenous factors just noted. Among women, for example, whose rates of ischaemic heart disease and chronic obstructive lung disease appear to be lower than those of men in most national and ethnic groups, even among nonsmokers, the effect of smoking at apparently equivalent dosage levels seems to be less.

In sum, then, the design of specific control programmes needs to take into account the effect of smoking as an interaction of three influences:

–dosage, i.e., the effective level of exposure to noxious substances in cigarette smoke, both accumulated and current

–exposure to other elements that contribute to or produce the same disease process

–susceptibility to the disease in the host population, presumably determined by genetic factors.

As smoking habits may be more amenable to control than other important risk factors or high levels of host susceptibility, measures

aimed at reducing exposure to cigarettes can be expected to pro-
duce substantial benefits. But whether to concentrate on short-
term programmes for helping adults whose accumulated exposure
may be approaching the critical point or longer-term efforts aimed
at the youth or some combination of both requires careful identifi-
cation of the groups that will benefit most from the planned con-
trol measures.

SMOKING BEHAVIOUR IN THE INDIVIDUAL

Cigarette smoking represents a category of health problems that can
be called *personal choice health behaviour*. This class of behaviour
includes many normal ways of increasing the enjoyment of life or
coping with its problems; it includes useful, frequently necessary,
forms of behaviour that have varying degrees of social acceptability.
For a more complete understanding of this sort of behaviour as it
applies to smoking, it is helpful to look at its four stages: *initiation,
establishment, maintenance,* and *cessation or other modification.*

The *initiation* of smoking usually occurs with young people,
frequently rather young children, and depends on how available
cigarettes are to them, the degree of their curiosity about what
smoking is like, and their need to conform with the behaviour of
others—whether parents, older siblings, or peers—or to rebel against
what seems like unreasonable proscriptions against smoking. Ac-
cordingly, smoking is much more common among children of par-
ents who are themselves regular smokers. As cigarette smoking be-
comes widespread in a society, it tends to be taken up with increas-
ing frequency by successive generations of young people. Many
older people may also turn to it, especially if it serves as a substi-
tute for a previously well-established behaviour pattern as was the
case when many males switched to cigarettes from cigars and pipes
in the years between 1920 and 1950.

The *establishment* of smoking as a continuing habit in adoles-
cents can be influenced by three groups of factors: a cost-benefit
balance, common perceptual stereotypes, and psychological per-
sonal structure and integration. The *costs* may be either those to
the individual or to society and may reflect health concerns or eco-
nomic or aesthetic values. The *benefits* are similarly varied, ranging
from easing social contacts and reducing tension to enhancing the
sense of pleasure. The *perceptual stereotypes* have to do with the
mythology of what smoking is like, what smokers are like, and why
people smoke, and are derived from both the brand-name advertis-
ing of cigarettes and the counter-publicity of antismoking groups.
No one pattern of *psychological forces* dominates the reasons
either for or against smoking. Either smoking or *not* smoking can be
a way of expressing the conflict between satisfying one's own de-
sires and conforming to the demands and expectations of society

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and its leading figures. Similarly, these opposite kinds of behaviour may reflect the individual's relative need to maintain control over his own behaviour and destiny as opposed to being subject to the control of others or the vagaries of chance.

The *maintenance* of smoking behaviour is usually supported by the development of habituation or dependence—*habituation* tending to reflect simply repetitive behaviour and *dependence* an increasing desire or need for the effects produced by the behaviour. In either case, prior to 1950, confirmed smokers tended to continue smoking unless they became too ill or had their supply of cigarettes interrupted by wartime shortages or economic deprivation. Since then, the threat to health posed by cigarette smoking has become sufficiently well known to millions of smokers to influence many of them towards trying to give up cigarettes or modifying their smoking in some way that would minimize its potential hazards.

Whether a smoker considers the idea of *cessation or other modification* of his smoking habits and how successful he will be in this effort depends largely on a number of factors, such as his perception of the threat posed by his continued smoking, how psychologically useful his smoking is to him, and the environmental forces that either encourage or interfere with his efforts at behavioural change. Not only must the smoker be aware of a threat to his health, but he must perceive this as important and personally relevant, as well as feel able to alter his behaviour and accept as valuable the results of such alterations. Psychologically, the smoker in the process of quitting must be able to deal both with the absence of the stimulation provided by cigarettes and with the sense of craving (for tobacco) and other withdrawal symptoms. In this, he can be aided by social forces, interpersonal influences and mass communications, plus influences generated by health workers and other key groups in encouraging behavioural change. When these influences become significant to enough people, action will be taken through legislation, changes in regulations, and changes in customs. A good example is the growing movement to "protect the rights of the non-smoker" by reducing his exposure to tobacco smoke produced by others.

SMOKING BEHAVIOUR IN A SOCIETY

New fashions in smoking tend typically to appear first among the younger, more affluent members of the adult population who form part of the upper classes of the society. In most developed countries cigarette smoking increased sharply during the years between 1910 and 1920 because of the switch to this form of tobacco use by young males taking up the habit for the first time and by older men who had hitherto smoked pipes and cigars. The increase in cigarette smoking was temporarily halted in many countries during

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the Second World War because of tobacco shortages but soon resumed its upward course after the war and quickly made up for lost time. Also contributing to this increase were the larger numbers of women who began smoking cigarettes during and immediately after the 1940s. As with men thirty years earlier, cigarette smoking among women was at first confined mainly to the upper and upper-middle classes, but rapidly moved down the social scale to include a substantial number of middle and lower-class women.

By the mid-1950s, when new information about the harmful effects of cigarette smoking began to receive widespread publicity, the growth of cigarette smoking began to be curtailed, first among men in the upper classes, then among males in the other social classes, and finally among adult women by the late 1960s. Of particular interest is the phenomenon observed in the USA where the percentage of adolescent boys taking up cigarette smoking has gradually dropped, apparently in response to the intensive educational efforts begun in the late 1950s, while the percentage of adolescent girls taking it up has gradually risen. By 1974 the rates for boys and girls were almost identical and it seems likely that the continuous increase in the proportion of young girls taking up smoking has probably reached its limit, perhaps because "equality" in smoking with boys seems to have been achieved. However, despite the substantial numbers of adults who no longer smoke cigarettes, this increase in smoking by younger women in developed countries—at rates far higher than for their mothers and grandmothers—has caused the *per capita* consumption of cigarettes to continue to rise in these countries.

The growth of cigarette smoking in developing countries is a subject on which there is as yet little information. We do know that there have been large relative increases in smoking in some countries with low pre-1950 rates, but there are few reliable statistics to indicate the exact rate of growth. From anecdotal material, however, it is clear that cigarette smoking tneds to be taken up first by persons having the closest first-hand association with people from the developed countries, i.e., the professional, political, and business leaders of those countries. Although the relatively high cost of cigarettes and the low standards of living in many developing countries may have prevented cigarette smoking from increasing as rapidly as it might otherwise have done, recent improvements in the economic conditions of some of these countries have provided a strong incentive for cigarette makers to launch aggressive marketing campaigns aimed at offsetting the more static markets for cigarettes in developed countries, brought on in part by changed attitudes toward smoking. Although there is no scientifically acceptable evidence to prove that advertising has contributed to the growth of cigarette use, and although cigarette smoking has, in fact, grown sharply in

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some countries without the help of advertising, the overwhelming aura of respectability and social acceptance conveyed by widespread advertising has almost certainly been an important factor in stimulating increased smoking.

BARRIERS TO SUCCESSFUL CONTROL

The main barriers to the successful design and implementation of control programmes are the following:

(1) *Gaps in medical and epidemiological knowledge.* At present, the knowledge of the effect of smoking on health and of how this manifests itself is sufficiently complete to convince the overwhelming majority of medical scientists and nearly as much of the general public in developed countries of the hazards involved. However, to direct control actions toward persons and groups that would benefit most from them, we need better means of identifying those at greater risk. At the moment our means of doing this are limited to identifying persons at the highest level of dosage exposure, those with concurrent exposure to other risk factors that increase the likelihood of lung cancer, ischaemic heart disease, or chronic obstructive lung disease, and those from ethnic groups with a high prevalence of these smoking-related diseases. Thus, earlier and more precise measurement of the effect of cigarette smoking on an individual or a group of individuals would provide an important basis for sharpening the direction of control action.

(2) *Economic and political conflicts.* In the early years after cigarette smoking was identified as a serious health problem, the economic and political influence of tobacco farmers and the importance of receipts from the sales and taxation of cigarettes undoubtedly impeded the development of political support in this area from governments. However, as the medical and epidemiologcal case against smoking became clearer and the costs to society in death and early disability were better identified, these economic and political barriers to control action have begun to be lowered, albeit rather slowly. Similarly, the importance of individual rights has grown, at first in preventing control actions that would infringe on the rights of smokers and, more recently, in restricting smoking in public places so as to protect the rights of nonsmokers.

On another front, efforts to develop less harmful substitutes for tobacco have recently resulted in the marketing of cigarettes partially made from such substitute materials. With these cigarettes the problem in health terms is the difficult one of ensuring that they are at least no more harmful than cigarettes made wholly from tobacco. In economic and political terms, they pose a significant threat to tobacco as an agricultural product because if they became

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widely accepted they could cause the world demand for tobacco to shrink by as much as fifty per cent.

(3) *Lack of knowledge on smoking behaviour.* Because of gaps in our knowledge of smoking behaviour, we are unable to be more helpful in assisting individuals who wish to quit smoking. In the USA, for example, during the decade from the early 1960s to the early 1970s, when the climate of social support for giving up smoking improved sharply, the spontaneous success rates for those who tried to give up smoking more or less permanently nearly trebled to about 40%. However, even in this favourable climate, only about one person in three who tried to stop smoking was successful.

These figures do not include the many persons who did not even try to give up smoking; although they accepted the fact that they would be better off if they did so, they were unwilling to expose themselves to the risk of failure. Despite the numerous attempts that have been made to develop systematic therapeutic programmes for helping people to quit smoking, on either an individual or a group basis, these have had no better results than the spontaneous success rates just mentioned, a record probably influenced by the tendency of these programmes to attract smokers with a history of previous failure, with whom the prospects of success are quite low. Much the same can be said of attempts to develop low-cost self-aids for individuals, as a substitute for costly professional therapy, and of pharmacological aids; although these aids have had some success, their effects have been limited and they are in need of further improvement.

Another area suffering from a lack of knowledge is our ignorance of the full potential of the effect of various regulatory and legislative aids such as increasing the price of cigarettes, differential taxation to promote one type of cigarette as against another, restrictions on places where smoking is permitted, restrictions on the ease of purchase and general availability of cigarettes, and the effect of reducing or banning cigarette advertising and promotional activities. Although there is some evidence to suggest that these actions are useful, the benefits achieved have not been adequately evaluated under controlled conditions.

A third area affected by the lack of knowledge is health education of the young and the development of more successful programmes to dissuade young people from taking up smoking. Although much is known about some of the principles contributing to effective health education of the young, these have not yet been incorporated into programmes, which could provide convincing evidence of their ability to reduce smoking. Without such evidence, it is difficult to gain support for making widespread and expensive changes in educational programmes.

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(4) *Lack of communication and coordination.* Occasional international conferences and sporadic reviews of the smoking problem, no matter how well thought out, are no substitute for a continuing system of communication and information exchange. The three international conferences on smoking that were held in 1967, 1971 and 1975 and the resolutions on the subject adopted by the World Health Assembly in 1970, 1971, 1975 and 1976 represent efforts to support and encourage international action, but they have not resulted in any system for ensuring that this takes place. National programmes that have attempted to deal with the total problem, such as the National Clearinghouse for Smoking and Health in the USA, have provided systematic annual bibliographical publications and literature reviews, which have made this task easier, but such programmes must be augmented by systematic attempts to facilitate communication and enhance cooperative efforts.

WHAT IS TO BE DONE?

In the face of these obstacles and the indifferent success of control efforts to date, it is clear that a much more systematic and multifaceted international attack will have to be mounted on the hazards of smoking if this public health problem is to be reduced; equally, this attack must be a coordinated one so that maximum benefit is derived from separate national efforts against different aspects of the problem. In its coordinating role on international health matters, WHO is ideally situated to undertake this task. Some of the key areas in need of immediate attention are:

● *Expert advice.* Because of the multidisciplinary nature of the problem, current control efforts are too often fragmented and piecemeal. What is needed is a small panel of international experts, operating under the aegis of WHO and comprised of behavioural scientists, economists, sociologists, toxicologists, pharmacologists, etc., in addition to public health workers, to provide national, regional and international programmes with ready access to the best available advice on the probable effectiveness of various measures.

● *Monitoring the problem.* Far too little is known about the various factors that affect smoking and smoking behaviour in different countries; the available information is often collected in sufficiently different ways to make comparisons and extrapolations from national experience impossible. There is thus the need not only for greatly expanded collection activities but also for countries to be informed about what sort of information to collect and in what way so that national efforts will reinforce one another and provide the basis for global analyses.

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- *Information exchange.* In addition to the dearth of information, there is not enough cross-dissemination of available data to permit making the best use of it. This gap could be filled by periodic newsletters, compiled by WHO from national contributions, and by occasional regional and international conferences.

- *Economic implications.* If cigarette smoking is reduced by a substantial amount or the use of tobacco substitutes in cigarettes is found to be safe and is widely adopted, then the economies of tobacco-producing countries will be adversely affected. Although we are far from this point at present, it is none too early to begin considering alternative employment of tobacco lands and farmers and the other economic effects of this process.

- *Research.* Virtually all aspects of the programme to control smoking can benefit from more systematic research—ranging from the epidemiological aspects that would identify better predictive measures of dosage, through research into why people smoke and what educational and other methods are best calculated to limit smoking, to the development of various aids and methods to help people stop smoking. In the latter instance, to cite just one example, there has been no reliable or systematic testing of the usefulness of the various commercial products that claim to help a person stop smoking.

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