CHAPTER 3

CHANGES IN SMOKING-ATTRIBUTABLE MORTALITY

CONTENTS

Introduction

In 1938, Raymond Pearl reported elevated death rates among white males who smoked tobacco, especially those aged 30 to 60 years (Pearl 1938). Pearl's study of 6,800 subjects revealed the increase in mortality risk to be highest among heavy smokers. In 1954, Hammond and Horn reported on the 20-month followup of their prospective study of 188,000 white men, aged 50 to 69 years (Hammond and Horn 1954). Death rates were highest among men who smoked cigarettes but not other tobacco products, and increased with the amount of cigarette use. Overall, the number of deaths among cigarette smokers was 52 percent greater than would be expected from nonsmokers' mortality rates. Most of the increased mortality could be attributed to deaths from cancer and especially from coronary heart disease (CHD).

In 1964, the Advisory Committee to the Surgeon General reviewed seven prospective studies of smoking and mortality, encompassing over 1.7 million entrants. For the 1.1 million male enrollees, the overall mortality ratio, defined as the observed number of deaths in current cigarette smokers divided by the number expected from nonsmokers' rates, was 1.68. "For all seven studies," the Committee stated, "coronary artery disease is the chief contributor to the excess number of deaths of cigarette smokers over nonsmokers, with lung cancer uniformly in second place. For all seven studies combined, coronary artery disease (with a mortality ratio of 1.7) accounts for 45 percent of the excess deaths among cigarette smokers, whereas lung cancer (with a ratio of 10.8) accounts for 16 percent" (US PHS 1964, p. 30).

In 1979, the Surgeon General described cigarette smoking as "the single most important preventable environmental factor contributing to illness, disability and death in the United States" (US DHEW 1979, p. vii). The 1982 Surgeon General's Report, citing an analysis by Doll and Peto (1981), estimated that for the year 1978, tobacco use caused 122,000 cancer deaths in men and women (US DHHS 1982). For 1982, the estimate for smoking-caused cancers was 129,000 (US DHHS 1982). The 1983 Surgeon General's Report estimated that 170,000 Americans died annually from CHD caused by cigarette smoking (US DHHS 1983). "During 1965-1977," the Report noted, "there were an estimated 2.8 million premature deaths from heart disease, primarily CHD, in American men and women attributable to the use of tobacco" (US DHHS 1983, p. 66).

The 1984 Report estimated that 80 to 90 percent of the 62,000 deaths from chronic obstructive lung disease (COLD), referred to later in this discussion as chronic obstructive pulmonary disease (COPD), in the United States in 1983 were attributable to cigarette smoking (US DHHS 1984). "Over 50,000 of the COLD deaths can therefore be considered preventable and premature since these individuals would not have died of COLD if they had not smoked" (US DHHS 1984, p. ii). In 1987, the Economic Report of the President stated, "Smoking presents the largest single source of health risk in America" (U.S. President 1987, p. 184).

This Chapter further delineates the mortality consequences of cigarette smoking in the United States. Deaths attributable to cigarette smoking are reported for two benchmark years--1965 and 1985. The Chapter focuses on the health consequences of smoking for current and former cigarette smokers. Deaths of nonsmokers caused by environmental tobacco smoke (National Research Council 1986;US DHHS1988a) and

deaths from cigarette-related fires (Consumer Product Safety Commission 1987; Botkin 1988) are not discussed, nor are the morbidity consequences of cigarette smoking (US DHEW 1979; Rice et al. 1986).

A Twenty-Year Perspective: 1965-85

The two-decade interval, 1965-1985, was selected primarily for reasons of data availability. The year 1985 was the most recent one for which complete, nationwide, cause-specific mortality statistics were available from the National Center for Health Statistics (NCHS). Moreover, in both 1965 and 1985, questions on cigarette use were appended to the National Health Interview Survey (NHIS), a nationally representative, face-to-face interview survey that has been conducted annually by NCHS (Massey et al., 1987; NCHS 1986). In particular, 1985 was the most recent full year for which complete population-weighted data from the NHIS were available (see Chapter 5).

In addition, the years 1965 and 1985 represented the approximate midpoints of two large-scale prospective surveys of smoking and mortality among men and women in the United States, both sponsored by the American Cancer Society. In the first of these two prospective studies (Garfinkel 1980a,b, 1981; Hammond 1961, 1964a,b, 1966, 1968, 1969, 1972; Hammond and Garfinkel 1961, 1964, 1966, 1968, 1969, 1975; Hammond et al. 1976; Hammond and Seidman 1980; Lew and Garfinkel l984, 1988), about 1 million persons were followed from 1959 through 1972. In the second study (Garfinkel 1985; Stellman and Garfinkel 1986; Stellman, Boffetta, Garfinkel 1988), about 1.2 million participants were followed from 1982 through 1988. The two studies will be referred to, respectively, as "Cancer Prevention Study I (CPS-I)" and "Cancer Prevention Study II (CPS-II)." In particular, this Chapter will present unpublished, preliminary results from the 4-year followup (1982-86) of CPS-II.

The theory, mathematics, limitations, and other methodological issues concerning the calculations of smoking-attributable mortality are described in the next section. The results of the analysis follow thereafter. Readers interested primarily in those results may proceed directly to the Section entitled "Populations at Risk: 1965 and 1985."

The Concept of Attributable Risk

In 1953, Levin estimated that 62 to 92 percent of all male lung cancers were "attributable to cigarette smoking" (Levin 1953). Levin's computations addressed the general problem: How many cases of a disease in a given population can be explained by the presence of a particular hazardous agent or a particular personal trait? Put differently, how many cases would have been avoided but for the presence of the agent or the trait (Doll and Peto 1981)?

In principle, the answer requires an experiment whereby disease rates are measured before and after the complete elimination of the hazardous agent or particular trait from the population of interest. Since this type of experiment is usually impractical, the most widely used approach is to estimate disease rates in representative sample populations of exposed and unexposed persons. The results are then extrapolated to the population of interest.

The phrase "cases *attributable* to agent A" is often used interchangeably with "cases *caused* by agent A." The latter term is meaningful so long as it is recognized that "caused" refers to an entire population rather than to any single, predetermined member of the population. Thus, the scientific validity of an estimate that 1,000 lives would be saved by the removal of some hazardous agent does not hinge upon naming the names of the people to be saved.

The population-based notion of causation is especially important for chronic diseases with multiple causes. Agent A, for example, may promote or enhance the disease-causing effect of agent B. A case-by-case analysis of afflicted individuals may never identify agent A as the primary cause in a single instance. Yet its elimination might substantially reduce disease incidence in the population under study.

Moreover, the concept of attributable risk generally requires a timeframe. In an assessment of the effects of removing a hazardous agent, a researcher could ask how many cases of a specific disease could be avoided in a specified time period, such as 1 year. When the disease has multiple causes, this quantity may differ from the number of cases of the disease that may eventually be avoided. By specifying a timeframe, the researcher inquires not whether such cases could be completely prevented, but whether their premature occurrence could be avoided.

For many diseases, death rates are more accessible and reliable than disease rates. Accordingly, computations of "attributable deaths" from a disease have been used in place of "attributable cases" of the disease. Because death from one cause or another is inevitable, such computations necessarily refer to a specific time period during which premature mortality may have been prevented.

Mathematics of Attributable Risk

Let d_1 and d_0, respectively, denote the incidence rates (in terms of new cases per unit time) of a particular disease among two sample cohorts--one exposed to a hazardous agent, the other unexposed. The two samples are assumed not to differ materially in any other respect, so that both would experience disease incidence d_0 in the absence of exposure. Accordingly, the difference d_1-d_0 measures the increase in disease incidence, or absolute risk, due to the agent. Moreover, the unitless ratio $r = d_1/d_0$, termed the relative risk, measures the degree to which the hazardous exposure multiplies the baseline incidence rate. It is often employed as a measure of the epidemiologic and biological significance of an observed association between an agent and a particular disease (Lilienfeld and Lilienfeld 1980; US DHEW 1979).

In the exposed cohort, the proportion of disease cases attributable to the hazardous agent is thus equal to $s=(d_1-d_0)d_1$ (which equals $(r-1)/r$). This quantity has been variously termed the assigned share or probability of causation or attributable proportion of risk among the exposed (Bond 1981; Oftedal, Magnus, Hvinden 1968; Black and Lilienfeld 1984; National Research Council 1984; Cox 1987).

For some hazardous agents, such as cigarette smoke, the disease incidence rates d_1 and d_o and the relative risk r have been estimated directly from prospective longitudinal studies of exposed and unexposed cohorts. Alternatively, retrospective case-control studies do not provide estimates of d_1 and d_0 but yield a close approximation to the rela-

tive risk *r* when incidence of the disease is low (Cornfield 1951). Both types of studies provide estimates of the assigned share *s*.

The estimate of relative risk *r*, derived from epidemiologic studies, is then applied to the population of interest. Let *p* denote the proportion of exposed persons in the subject population, estimated independently from survey data. Then the quantity $f = pr/[p(r-1)+1]$ is the fraction of all cases of the disease (in a given time interval) that occurs among exposed persons in the subject population. This is sometimes called the "case fraction" (Miettinen 1974). Moreover, if fraction *f* of all cases occurs among exposed persons, and if fraction *s* of such exposed cases is attributable to the hazardous agent, then the fraction of all cases attributable to the agent is $a = fs$. From the definitions of *f* and s, the quantity *a* can be expressed as

$$a = \frac{p\,(r-1)}{p\,(r-1) + 1}$$

This is Levin's measure of attributable risk, also termed etiologic fraction (Miettinen 1974), attributable fraction (CDC 1987b), and population-attributable risk (MacMahon and Pugh 1970). When *a* is expressed in percentage terms, it is often termed percent attributable risk or population-attributable risk percentage.

Equation (1) shows how the attributable risk *a* depends upon both the relative risk *r* and the proportion exposed *p*. Thus, an agent may be significant in the causation of disease among exposed persons so that its relative risk *r* greatly exceeds 1. Yet that agent may cause a small proportion of all cases of the disease because exposure rates p are low. Conversely, an agent that is widely prevalent (with large *p)* may contribute substantially to the total number of cases, even when its relative risk *r* is close to unity.

As a consequence of equation (1) the logistic transformation of *a* is

$$\log \frac{a}{(1-a)} = log\ p + log\ (r-1) \qquad (2)$$

where log denotes the natural logarithm. Equation (2) provides a convenient method of decomposing the uncertainty in the attributable risk *a* into two components--uncertainty in the proportion exposed *p* and uncertainty in the relative risk *r*.

Levin's measure of attributable risk can be generalized to cases where there are multiple levels of exposure, multiple causative agents, or confounding or stratifying variables, or when an agent can prevent a disease (Walter 1976; Miettinen 1974). In the case of multiple levels of exposure, it is convenient to let *dk* denote the incidence rate and $r_k - 1 = d_k/d_0$ denote the relative risk for the k-th exposure level. Similarly, let p_k denote the proportion of the subject population exposed at the k-th level. Then $s_k = (r_k-1)/r_k$ is the assigned share among cases exposed at the k-th level. Likewise, the quantity $f_k = p_k r_k/[E_k\ (r_k-1) + 1]$, where E_k denotes summation over exposure levels, is the fraction of all cases occurring among persons exposed at the k-th level. The generalized formula for attributable risk becomes $E_k f_k s_k$, which can be expressed as

$$a = \frac{E_k p_k\,(r_k-1)}{E_k p_k (r_k - 1) + 1} \qquad (3)$$

Let *D* denote the total number of cases of disease in the population of interest in a given time interval. Then $A = aD$ is the estimated number of cases in the interval that

are attributable to the agent. The quantity A is sometimes called "attributable cases." When relative risks or exposure rates vary by age, sex, or other stratifying variables, then separate estimates of A can be made for each combination of variables.

When there are multiple causative agents, attributable risks can be computed for each agent separately and for combined exposures. Thus, if agents X and Y both have a causal role in the development of a particular disease, then the relative risk for agent X may depend upon the presence or absence of exposure to agent Y. When X and Y act synergistically, some portion of the total risk attributable to X will reflect the combined contribution of X and Y. For example, indoor exposure to radon has recently been estimated to account for about 13,300 lung cancer deaths annually in the United States (Lubin and Boice 1988). Radon exposure and cigarette smoking interact synergistically in causing lung cancer (National Research Council 1988). Of the estimated 13,000 deaths attributable to radon exposure, about 11,000 would be due to the combined effect of smoking and radon, while about 2,000 would reflect radon exposure in nonsmokers (Lubin and Boice 1988).

Illustrative Calculation: Smoking and Lung Cancer in Women

Table 1 provides a detailed illustrative application of Levin's method to female deaths from lung cancer in the United States during 1985. The population of female smokers has been divided into ten exposure levels: five categories of current cigarette smokers based on the number consumed per day; and five categories of former cigarette smokers based on the length of time since quitting. For each exposure category, the upper panel shows the estimated prevalence p_k, derived from the 1985 NHIS. Also given are estimates of relative risk r_k derived from the 4-year followup (1982-86) of the second American Cancer Society prospective study (Garfinkel and Stellman 1988). At each exposure level, the upper panel also shows the assigned share *sk* and the case fraction f_k.

The computations are summarized in the lower panel of Table 1. For both current and former smokers, as well as for all females at risk, the estimated prevalences p represent the corresponding sums $E_k p_k$ over the prevalence rates p_k in the individual subcategories. The case fractions f likewise represent sums of individual fractions f_k, while the attributable risks a are derived from the corresponding sums $E_k s_k f_k$. Attributable deaths A are derived from the products aD, where D = 38,687 lung cancer deaths among adult females in 1985.

Table 1 shows that almost two-thirds of all female lung cancer deaths occurred among women who currently smoke one pack or more daily or who have quit smoking within the last 5 years. Nine out of ten lung cancer deaths occurred in women with any history of regular cigarette use. Cigarette smoking accounted for an estimated 82 percent of lung cancer deaths in women, or 31,600 deaths in 1985. About 9,300 (or 29 percent) of the 31,600 female lung cancer deaths that were caused by smoking occurred among former smokers.

Both the prevalence rates and the relative risks in Table 1 are subject to sampling variability. By a formula analogous to equation (2), a standard error for the logistic transformation of a can be derived. Under the assumption that D has no sampling

TABLE 1.—Detailed computation of smoking-attributable lung cancer deaths among females, United States, 1985

Exposure category	Prevalence p (%)	Relative risk[a] r	Assigned share s (%)	Case fraction f (%)
Current smokers				
1–10 per day[b]	9.3	5.5	81.9	9.4
11–19 per day	3.3	11.3	91.1	6.7
20 per day	9.3	14.2	93.0	24.0
21–30 per day	3.2	20.4	95.1	11.8
≥31 per day	2.7	22.3	95.5	10.8
Former smokers				
0–2 years[c]	5.0	18.2	94.1	16.7
3–5 years	2.5	11.2	91.1	5.0
6–10 years	3.4	4.9	79.5	3.0
11–15 years	2.0	3.2	68.5	1.2
≥16 years	4.0	1.8	43.4	1.3

Exposure category	Prevalence p (%)	Case fractions f(%)	Attributable risk a (%)	Attributable deaths[d] A
Current smokers	27.8	62.7	57.7	22,300
Former smokers	16.9	27.2	24.1	9,300
Current and former smokers	44.7	89.9	81.8	31,600

[a]Ratio of age-adjusted death rates, where age adjustment was performed by direct standardization to the age distribution of woman-years of exposure among nonsmokers.

[b]Number of cigarettes smoked per day, as of the date of enrollment (September 1982).

[c]Number of years elapsed since last smoked regularly, as of the date of enrollment (September 1982).

[d]Attributable deaths A equal aD, where a is attributable risk and D equals 38,687 lung cancer deaths among adult females in 1985.

SOURCE: Garfinkel and Stellman (1988); NHIS 1985, unpublished tabulations; NCHS, Division of Vital Statistics, 1985, unpublished.

variability, statistical confidence bounds for A can also be calculated. For the calculation shown in Table 1, the estimated confidence interval on a for all smokers was 72.1 to 88.6 percent. The corresponding confidence interval for D was 27,900 to 34,300 deaths. Only 2.6 percent of the variance of the logistic transformation of a was due to sampling variability of prevalence rates.

Uncertainties in Attributable Risk

Aggregation Bias Versus Statistical Precision

Sampling variation is not the sole source of uncertainty in estimates of attributable risk. The computations of Table 1 entail the assumption that the relative risks *rk* depend only upon the specified indices of current and former smoke exposure.

Thus, for former cigarette smokers in Table 1, the degree of risk after cessation of smoking is shown as depending only upon the length of cessation. Yet the magnitude of the residual risk also depends upon the extent of prior cigarette smoke exposure (Hammond 1968; Lubin et al. 1984) and the reason for stopping (Kahn 1966). Also, some persons may have quit smoking after lung cancer had been diagnosed. As Table 1 shows, women who had stopped smoking for 16 or more years at the time of enrollment into CPS-II had a subsequent 4-year relative risk of lung cancer equal to 1.8. Within this group of long-term quitters, however, those women who had previously smoked 21 or more cigarettes daily had an estimated relative risk of 4.0 (Garfinkel and Stellman 1988).

Likewise, for current smokers in Table 1, the degree of lung cancer risk is shown as depending only upon the current number of cigarettes smoked per day. Yet the risk depends critically upon the lifetime dosage of cigarette smoking, especially the duration of cigarette use and the age of initiation of regular smoking (Brown and Kessler 1988; Doll and Peto 1978, 1981; Peto 1986; US DHHS 1982). While the relative risk *r* was 22.3 for all women currently smoking 31 or more cigarettes daily (Table 1), it was 18.9 for heavy smokers of 18 to 30 years' duration and 38.8 for heavy smokers of more than 40 years (Garfinkel and Stellman 1988).

A more detailed, multidimensional breakdown of exposure levels may minimize errors of classification, but such disaggregation also increases the sampling variability of the estimates. Conversely, increased aggregation of exposure levels will reduce sampling variability. Thus, if relative risk were assumed to depend only upon present smoking status (current versus former), then the estimated attributable risk for female lung cancer deaths in 1985 would be 80 percent, with a confidence range of 77 to 83 percent. The confidence range of attributable deaths *A* would be narrowed to 29,700 to 32,000.

Age-Standardization

The relative risks in Table 1 were estimated as a ratio of age-adjusted death rates, where the age adjustment was performed by direct standardization to the age distribution of nonsmokers' person-years at risk. In principle, if the relative risk is in fact age independent, then the estimate of relative risk in large samples should not be very sensitive to the choice of the standard population (Anderson et al. 1980). In practice, however, the estimates can depend strongly upon the standard population. For the illustrative calculation in Table 1, the use of the entire population of CPS-II woman-years at risk (rather than nonsmokers only) resulted in an attributable risk for lung cancer of 79 percent, with a confidence range of 75 to 82 percent (see Table 11).

Potential Biases in Applying the Results of Prospective Studies to the General Population

Subjects enrolled in the CPS-II prospective study constituted over 1.5 percent of all American adults age 45 and over (Stellman, Boffetta, Garfinkel 1988). Still, they differed from the U.S. population in a number of ways (Garfinkel 1985; Stellman and Garfinkel 1986). CPS-II entrants were more highly educated. The black and Hispanic populations were underrepresented, though less so than in CPS-I (Garfinkel 1985). As in CPS-I, institutionalized and seriously ill persons, as well as illiterate people who could not complete a questionnaire, were excluded (Lew and Garfinkel 1984). In both CPS-I and CPS-II, the overall mortality rates of the enrollees fell substantially below those of the general U.S. population (Hammond 1969; Lew and Garfinkel 1988).

These considerations do not by themselves invalidate the use of CPS-II to estimate smoking-attributable risks for the entire American population. The critical assumption in Table 1 above is whether the estimated relative risks *rk-not* the absolute death rates dk--are representative of the general population.

For CHD and for all-cause mortality, CPS-I subjects who were reportedly well at the time of enrollment showed higher estimated relative risks of cigarette smoking than those subjects who said they were sick or who gave a recent history of cancer, heart disease, or stroke (Hammond and Garfinkel 1969; Lew and Garfinkel 1988). A similar elevation of relative risk in well subjects has been found for lung cancer in CPS-II (Garfinkel and Stellman 1988). Since initially well persons had lower disease rates, the proportional effect of cigarette smoking appeared to be larger. While CPS-I and CPS-II excluded seriously ill and institutionalized persons, the magnitude of the resulting bias is unclear. In the 1980 U.S. Census, about 1.5 percent of the U.S. adult population was institutionalized. Among persons aged 65 years and over, the proportion was 5.3 percent (U.S. Bureau of the Census 1986).

Cigarette smoking has been found to act synergistically with certain workplace exposures (such as asbestos and ionizing radiation) in the development of lung cancer (US DHHS 1985; Saracci 1987; National Research Council 1988). Such interactions may also be present in the etiology of nonneoplastic lung disease. Alcohol and tobacco likewise interact synergistically in the etiology of oral and esophageal cancer (US DHEW 1979). Moreover, cigarette smoking has been found to interact synergistically with elevated serum cholesterol and elevated blood pressure in enhancing the risk of CHD (US DHHS 1983). Persons of lower socioeconomic status (SES) may be more likely to receive such workplace exposures, to consume alcohol heavily, or to have unfavorable CHD risk factors. However, if the effects of cigarette smoking are multiplicative, then exclusion of such persons from CPS-I and CPS-II would not bias the estimated relative risks of disease due to cigarette smoking. Conversely, if the effects of cigarette smoking are purely additive, rather than synergistic, then the exclusion of persons with elevated baseline disease rates would bias upward the estimated relative risks of disease due to smoking.

The estimated relative risks in Table 1 are specific to women and have been standardized for age. Standardization for other stratifying or confounding variables was not performed. In principle, failure to control for such variables could bias upward or

downward the estimated relative risks due to cigarette use. As discussed in Chapter 2, numerous attempts to control statistically for confounding and stratifying variables have not materially altered the estimated relative risks for cigarette-related diseases.

In the illustrative computation of Table 1, no distinction among the races has been drawn. For both sexes, the prevalence of current cigarette use is higher for blacks than for whites. Conversely, smaller fractions of black men and women are former cigarette smokers (US DHHS 1988b). Black persons were underrepresented in CPS-II, constituting only 4 percent of entrants (Stellman and Garfinkel 1986). Hence, the relative risks reported in Table 1 may not be accurate for black women. Among the 38,687 adult female lung cancer deaths in 1985, a total of 3,392 (8.8 percent) occurred in black women. Hypothetically, if the attributable risks *a* among black women had been only half those of whites, then the smoking-attributable lung cancer deaths in Table 1 would be reduced from 31,600 to 30,300.

In prospective cohort studies, mortality rates tend to be reduced in the initial year or two of followup. This phenomenon of lower initial mortality results from a tendency to exclude persons who are sick at the outset of the study. In particular, the relative risks in Table 1 were derived from the 4-year followup (1982-86) of CPS-II subjects. Accordingly, it is possible that the planned 6-year followup of CPS-II (1982-88) will reveal somewhat lower relative risks than those reported for the first 4 years.

Conversely, measurements of exposure and other personal characteristics, typically obtained at the start of a prospective study, become less accurate as the duration of followup increases. The relative risks reported in Table 1, for example, have been classified according to the subjects' cigarette smoking practices upon enrollment in 1982. If many women who were current smokers in 1982 had in fact quit smoking by 1986, then the reported relative risks for "current" smokers are actually those of a mixture of current and former smokers.

In the analysis reported below, the 4-year followup of CPS-II is to be compared with the 6-year followup of CPS-I. Such a comparison needs to be interpreted in light of potential biases arising from short- and long-duration followup in prospective studies.

Uncertainties in Exposure

Potential errors in estimated exposure rates *pk* are a further source of uncertainty in the computation of attributable risk *a*. In the illustrative calculation of Table 1, such exposure rates were derived from the 1985 NHIS, a large-scale, stratified, face-to-face household interview survey of the noninstitutionalized civilian population of the United States. Among the possible errors in NHIS estimates are: underreporting or misreporting of current cigarette use; inaccurate recall of past cigarette smoking; nonresponse biases due to exclusion of some persons not available for interview; and underrepresentation of certain population segments. These sources of uncertainty are discussed in Chapter 5. On the whole, NHIS-derived estimates of population smoking rates have been consistent with other face-to-face interview surveys (CDC 1987a).

The estimation of attributable deaths *A* requires information on total deaths *D*. For the computation in Table 1, the latter quantity was defined as deaths in 1985 whose underlying cause was primary lung cancer (International Classification of Diseases, Ninth Revision [ICD-9], Code 162). Deaths from the larger class of Respiratory Cancers (ICD-9 Codes 162-165) were not used because they include pleural mesotheliomas and secondary lung cancers. Still, the use of ICD-9 Code 162 alone may not eliminate all errors of death certification. In a review of over 1,300 thoracic cancer deaths in Minnesota between 1979 and 1981, Lilienfeld and Gunderson (1986) identified four cases of pleural malignant mesothelioma that had been classified as Code 162.9. Moreover, it is at least arguable that physicians in recent years have been reluctant to diagnose primary lung cancer in the absence of a history of cigarette smoking (McFarlane et al. 1986).

While errors in disease classification and death certification of lung cancer in 1985 may be relatively minor, the same cannot be said with assurance about other diseases caused by cigarette use. Thus, deaths certified as being caused by CHD (ICD-9 Codes 410-414) may not adequately reflect the lethal consequences of cigarette use on the cardiovascular system. Many deaths from Hypertensive Diseases (Codes 401-404, including Hypertensive Heart Disease, 402, and Hypertensive Disease, 404) may have been aggravated by cigarette use. Similarly, deaths certified as being caused by COPD (ICD-9 Codes 490-492 and 496) may incompletely reflect the numbers of deaths from nonneoplastic respiratory disease due to smoking. Many cases of Influenza and Pneumonia (ICD-9 Codes 480-487) may not have been lethal but for the coexistence of cigarette-induced lung damage.

The major prospective studies of cigarette smoking and mortality that were initiated in the 1950s relied upon the International Classification of Diseases, Seventh Revision (ICD-7) (Hammond 1966; Dorn 1959; Kahn 1966; Rogot 1974; Rogot and Murray 1980; Doll and Hill 1956, 1964, 1966; Doll et al. 1980; Doll and Peto 1976). Coding conventions have changed considerably since ICD-7 was adopted in 1955 (Klebba 1975, 1982; Klebba and Scott 1980). While ICD-7 Code 162 was reserved for lung cancer that was "specified as primary," a separate code 163 was allocated to lung cancers "not specified as primary or secondary." In practice, however, epidemiologists and vital statisticians recognized that the great fraction of lung cancer deaths certified under ICD-7 Code 163 were primary and that deaths certified under the two codes were in fact indistinguishable. Accordingly, it was standard procedure to report combined deaths for Codes 162 and 163--a practice adhered to in the analysis below. Still, the use of the combined category 162-163 in ICD-7 may have introduced greater diagnostic uncertainty than the current use of Code 162 in ICD-9.

Previous Estimates of Attributable Risk from Cigarette Smoking

Many authors have estimated the number or proportion of deaths attributable to cigarette use, either from a single cause, a group of causes, or all causes (Ravenholt 1964, 1984; Rice et al. 1986; McIntosh 1984; Whyte 1976; Hammond and Seidman

1980; Doll and Peto 1981; Garfinkel 1980a; U.S. Office of Technology Assessment (US OTA) 1985; Schultz 1986; Goldbaum et al. 1987; CDC 1987b). Doll and Peto (1981) estimated 83,000 smoking-attributable deaths from lung cancer in 1978. Rice and colleagues (1986, Table 5) estimated 270,000 smoking-attributable deaths among U.S. adults in 1980, including 86,000 from CHD, 75,000 from lung cancer, and 14,000 from "emphysema, chronic bronchitis." The Centers for Disease Control (1987b) estimated 315,000 smoking-attributable deaths for 1984, including 77,000 from CHD, 93,000 from lung cancer, and 51,000 from "chronic bronchitis, emphysema" combined with "chronic airways obstruction."

These studies differ with respect to specific causes of disease, the time period under consideration, the populations at risk, the sources of epidemiologic data, and the specific methodology for estimation of risk. Thus, some researchers have directly applied Levin's measure of attributable risk, as defined in equations (1) and (3) (Rice et al. 1986; McIntosh 1984; CDC 1987b; Goldbaum et al. 1987; Whyte 1976). In doing so, they assumed that estimates of relative risk *r*, derived from particular epidemiologic studies, could be extrapolated to the population under consideration. By contrast, Hammond and Seidman (1980) and Garfinkel(1980a) computed attributable risks directly for the CPS-I study population.

In an analysis of avoidable deaths from cancer, Doll and Peto (1981) employed a different model. Let *N* denote the size of the population at risk, while *D* denotes the total number of deaths from a specific cause. If *d0* denotes the cause-specific death rate among unexposed persons, then *D-d0N* is an estimate of the number of deaths attributable to the exposure. To estimate attributable cancer risks for the United States in 1978, Doll and Peto (1981) then assumed that the age- and sex-specific cancer mortality rates for nonsmokers do observed in CPS-I during 1959-72 could be applied to nonsmokers in the general population in 1978. In support of such an assumption, they note that for men, nonsmokers' cancer rates in other prospective studies (Kahn 1966; Doll and Peto 1976) closely matched those observed in CPS-I (Doll and Peto 1981). Moreover, CPS-I lung cancer rates of nonsmoking women were similar to those of U.S. women in 1950, before their lung cancer rates began to increase.

Doll and Peto's method was employed by OTA (1985) to estimate attributable deaths from CHD (US OTA 1985). For cancer, nonsmoker death rates in CPS-I may well approximate *d0* for the U.S. population. But the same conclusion does not appear to be warranted for CHD (Sterling and Weinkam 1987). In fact, the use of CPS-I nonsmoker death rates yielded an estimate of 142,000 smoking-attributable deaths from CHD in 1982. By contrast, application of the Levin method gave an estimate of 91,000 deaths (US OTA 1985).

Doll and Peto (1981) rejected the application of relative risks derived from CPS-I to the U.S. population in 1978. Their central concern was that such relative risks had increased in the two decades since the start of CPS-I in 1959. Among smokers aged 60 years or more in 1965, a much smaller fraction had smoked regularly during early life. For older women smokers, in particular, only one in eight had begun to smoke regularly as a teenager. This proportion increased markedly in subsequent decades (Chapter 5). In view of the importance of quantity and duration of smoking in determining lung cancer risk-and especially in view of the critical role of early-life smoking in the etiol-

ogy of smoking-induced cancers (Peto 1986)--it was highly likely that the relative risks for smoking-induced cancers would have increased since the early 1960s. (See also Doll et al. 1980.)

Accordingly, there may be serious biases in the application of relative risks from 1960s prospective epidemiologic studies to 1980s populations. Such potential biases constitute the most serious criticism of prior studies of smoking-attributable deaths. Updated epidemiologic evidence for the 1980s is needed to address this criticism.

Populations At Risk: 1965 and 1985

Table 2 and Figures 1 through 5 describe the populations at risk in 1965 and 1985. While Table 2 reports the percentages of smokers, the figures show the absolute numbers of U.S. resident adults in each smoking category for each year. Children and young adults under age 18, who may also suffer adverse effects from cigarette use, are excluded from Table 2 and the figures.

In both 1965 and 1985, respondents to the NHIS were asked, "Have you smoked at least 100 cigarettes in your entire life?" Those who answered affirmatively were then asked how much they smoked currently or, if they were not current smokers, when they

TABLE 2.--Prevalence of cigarette smoking, persons aged 18 years or more, United States, 1965 and 1985

	1965[a] (%)	1985[b] (%)
Males		
Current smokers[C]	53.4	32.7
Former smokers	20.8	29.1
Never smoked regularly[d]	25.8	32.8
Females		
Current smokers[C]	34.1	27.5
Former smokers	8.1	17.1
Never smoked regularly[d]	57.8	55.4

NOTE: Prevalence estimates for 1965 and 1985 have been directly standardized to the age distributions of the U.S. resident populations in each year, respectively (U.S. Bureau of the Census 1974, 1986).

[a]Based upon 52,873 self-responses to the Cigarette Smoking Supplement to the 1965 National Health Interview Survey. Standard errors 0.3 to 0.4 percent for males, 0.1 to 0.2 percent for females. Inclusion of 33,422 additional proxy responses resulted in the following estimates: male current smokers, 51.9 percent: male former smokers, 19.0 percent; female current smokers, 33.6 percent; and female former smokers, 7.7 percent.

[b]Based upon 32,859 self-responses to the Cigarette Smoking Supplement to the 1985 National Health Interview Survey. Standard errors 0.4 percent for males, 0.3 percent for females.

[c]In 1965, current smokers included all respondents who reported a current number smoked per day, including "less than 1 per day." In 1985, current smokers included all respondents who answered affirmatively to the question "Do you smoke now?"

[d]In both 1965 and 1985, the category "never smoked regularly" included two groups of respondents: (1) those who answered negatively to the question "Have you ever smoked at least 100 cigarettes in your life?"; and (2) those who answered affirmatively but denied ever smoking cigarettes regularly. In 1965 and 1985, respectively, group I accounted for 99 percent and 97 percent of all respondents in the category "never smoked regularly."

last smoked regularly. While the NHIS for 1965 permitted proxy respondents, the estimates in both years have been derived from self-respondents only (see Note b of Table 2).

Table 2 shows the percentage distribution among adult men and women in three categories: current smokers, former smokers, and those who never smoked regularly. Between 1965 and 1985, the proportions of current smokers declined and the proportions of former smokers increased. The most marked change was the decline in the prevalence of current cigarette use among adult men.

In Figure 1, the responses have been further divided into four categories: current smokers of fewer than 25 cigarettes daily; current smokers of 25 or more cigarettes daily; former smokers who quit within the last 5 years; and former smokers who stopped for more than 5 years. The weighted proportions in each category, tabulated by age and sex, were then multiplied by the corresponding estimates of the U.S. resident population (U.S. Bureau of the Census 1974, 1986).

In 1965, there were an estimated 53.7 million adult current cigarette smokers (standard error, 0.2 million), which represented about 43 percent of all U.S. residents aged 18 years or more. By 1985, there were an estimated 53.5 million adult current smokers, composing 30 percent of U.S. adults. While the total number of current smokers stayed about the same, there was a shift in their distribution by sex. The number of adult male current smokers declined from 31.7 million (53.4 percent) in 1965 to 28.2 million (32.7 percent) in 1985, while adult female smokers increased from 22.0 million (34.1 percent) to 25.3 million (27.5 percent) (Figure 1).

In 1965, about 28 percent of adult male smokers who were nonproxy respondents to the NHIS consumed 25 or more cigarettes per day (Figure 1). By 1985, this proportion had risen to 32 percent. For women, the proportions of heavier current smokers rose from 14 percent of nonproxy respondents in 1965 to 21 percent of smokers in 1985. The true population prevalence of smoking 25 or more cigarettes per day in 1965 is somewhat uncertain because the elimination of proxy respondents may make the sample nonrepresentative. As shown in Chapter 5, however, there was no significant change in the proportion of heavy smokers between 1974 and 1985.

By contrast, the numbers of former smokers increased substantially between 1965 and 1985. Thus, in 1965, there were about 17.6 million adult former smokers (12.4 million men and 5.2 million women). By 1985, this number had risen to 40.9 million (25.2 million men and 15.7 million women). There was an increase in the proportion of former smokers who had stopped for more than 5 years (from 49 to 63 percent of male former smokers, and from 41 to 57 percent of female former smokers) (Figure 1).

Cigarette Smoking and Other Forms of Tobacco Use

Figure 2 shows the 1965 and 1985 adult populations broken down according to the type of tobacco used. In 1965, the NHIS included questions on cigar and pipe smoking as well as cigarette use. The 1985 questionnaire inquired only about cigarette smoking. However, questions about all forms of tobacco use, including smokeless tobacco, were included on a supplement to the 1985 Current Population Survey, performed by the U.S. Bureau of the Census (see Chapter 5).

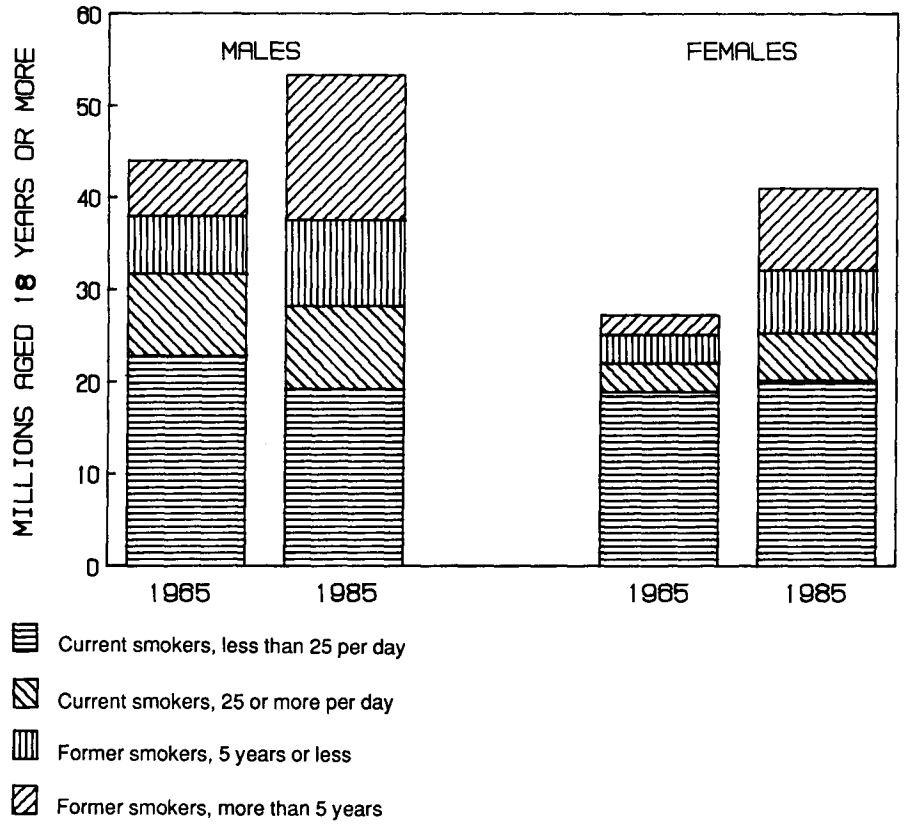

FIGURE 1.—Populations of current and former cigarette smokers, adult men and women, United States, 1965 and 1985

SOURCE: Estimated from unpublished tabulations, NHISs 1965 and 1985; and estimates of the resident populations of the United States by age and sex, 1965 and 1985 (US Bureau of the Census 1974, 1986).

Figure 2 shows a marked change over two decades in the forms of tobacco used by men. In 1965, 5.2 million men (9 percent) had a history of ever smoking pipes or cigars, but not cigarettes. In 1985, the number using noncigarette tobacco dropped to 2.7 million or 3 percent of the men. In 1965, 29 million men had a history of ever smoking cigarettes and other forms of tobacco, about two-thirds of all cigarette smokers. By 1985, the number had dropped to 5.6 million, only 1 in 10 of all cigarette smokers.

Older Cohorts of Cigarette Smokers

Figures 3 and 4 focus on persons aged 60 years and over, who suffer the highest incidence rates of smoking-related diseases. For 1965 and 1985, respectively, these groups of older persons were born before 1906 and before 1926. Among older men, as shown in Figure 3, the two-decade interval witnessed a increase in the number of former cigarette smokers. Among older women, the number of current smokers

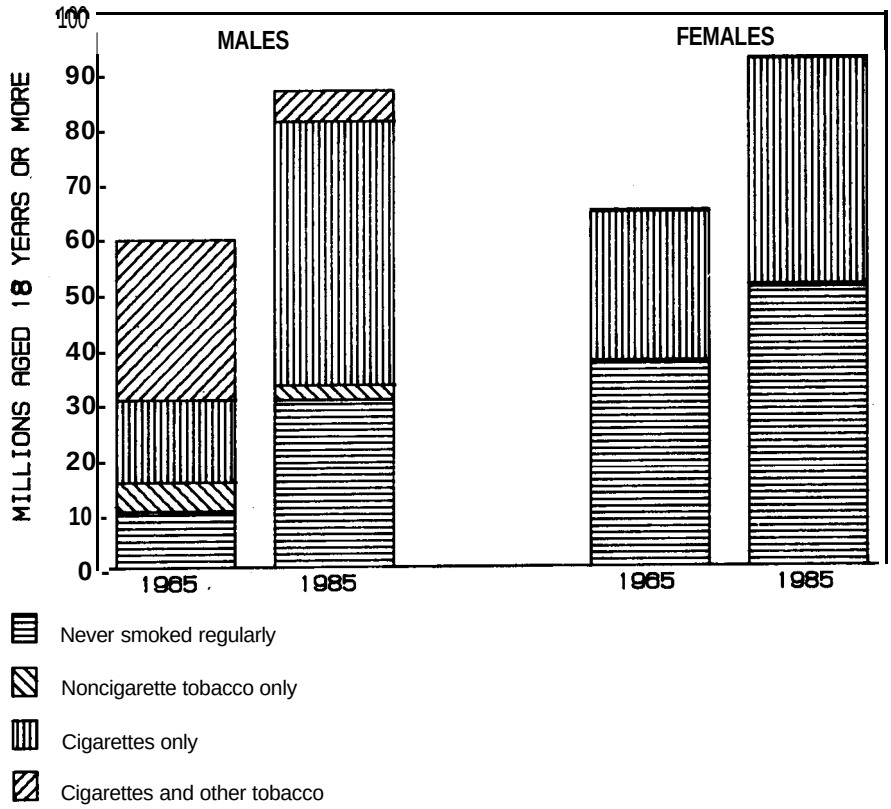

FIGURE 2.--Populations of adult men and women classified by history of tobacco use, United States, 1965 and 1985

SOURCE: Estimated from unpublished tabulations, NHISs 1965 and 1985; unpublished tabulations. CPS 1985; and estimates of the resident populations of the United States by age and sex, 1965 and 1985 (US Bureau of the Census 1974, 1986).

doubled, while the number of former smokers increased sixfold. Between 1965 and 1985, the population of older women with a history of regular cigarette use, past or present, increased over threefold.

The NHISs for 1965 and 1985 did not ask about the age of initiation of cigarette use. However, this information is available from other sources. For 1985, tabulations of the age of onset of regular cigarette use were made from the Current Population Survey. About 69 percent of older men with a history of cigarette use, past or present, began to smoke before age 20 (Figure 4). Among older women, the proportion was 39 percent.

For 1965, three sources of information provide the age of smoking initiation among cohorts born before 1906: the NHISs of 1978-80 (Harris 1983), the Current Population Survey of 1955 (Haenszel et al. 1956), and the initial 1959 questionnaire to CPS-I (Hammond 1966, Appendix tables). For older men with a history of cigarette use, about 60 percent started smoking before age 20 (range, 56 to 62 percent). For older women smokers, about 12 percent started in their teenage years (range, 9 to 15 percent).

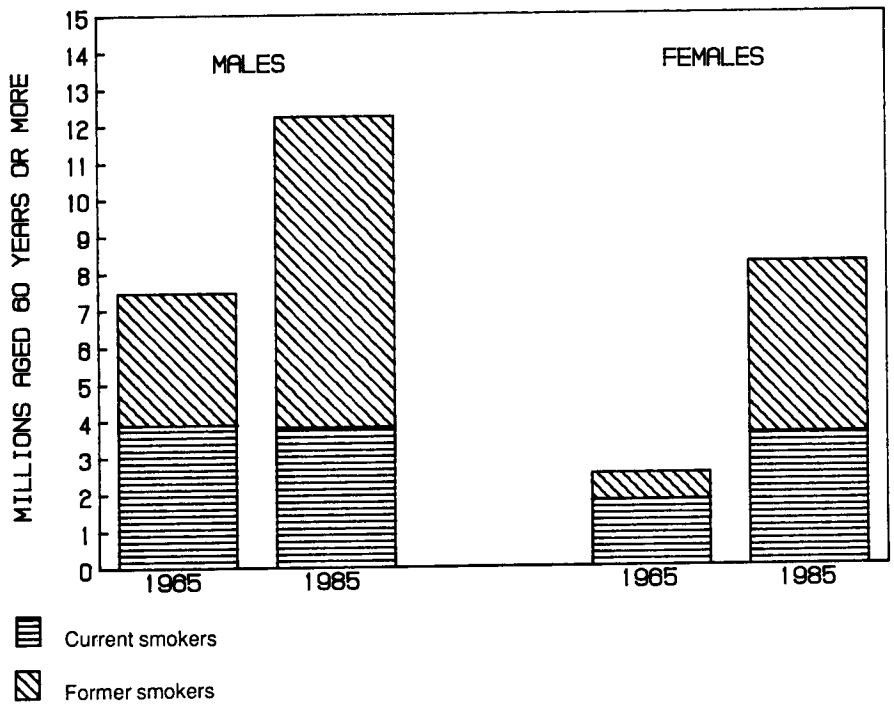

FIGURE 3.—Populations of current and former cigarette smokers, men and women aged 60 years or more, United States, 1965 and 1985

SOURCE: Estimated from unpublished tabulations, NHISs 1965 and 1985; and estimates of the resident populations of the United States by age and sex, 1965 and 1985 (US Bureau of the Census 1974, 1986).

Accordingly, the period between 1965 and 1985 saw a marked increase in the number of women smokers who reached the age of 60 years (Figures 3 and 4). Moreover, the number of such women who started smoking in their teens increased by about tenfold (Figure 4). Additional data on age of initiation are presented in Chapter 5.

Overlapping Populations at Risk

In 1965, a total of 71.3 million adults had a history of regular cigarette smoking, past or present. By 1985, this count had increased to 94.4 million. These two populations overlapped. Among the adult population at risk in 1985, about 54.8 million were born before 1948, and therefore they were also aged 18 years or more in 1965. About 95 percent of the latter group began to smoke during 1965 or earlier (Harris 1983; unpublished tabulations from the Current Population Survey 1985). This means that about 51.8 million adults, who had ever smoked in 1985, had also been at risk in 1965.

The overlap is depicted graphically in Figure 5, where the diagonal lines show the populations common to both years. Among 44.1 million adult men with a history of

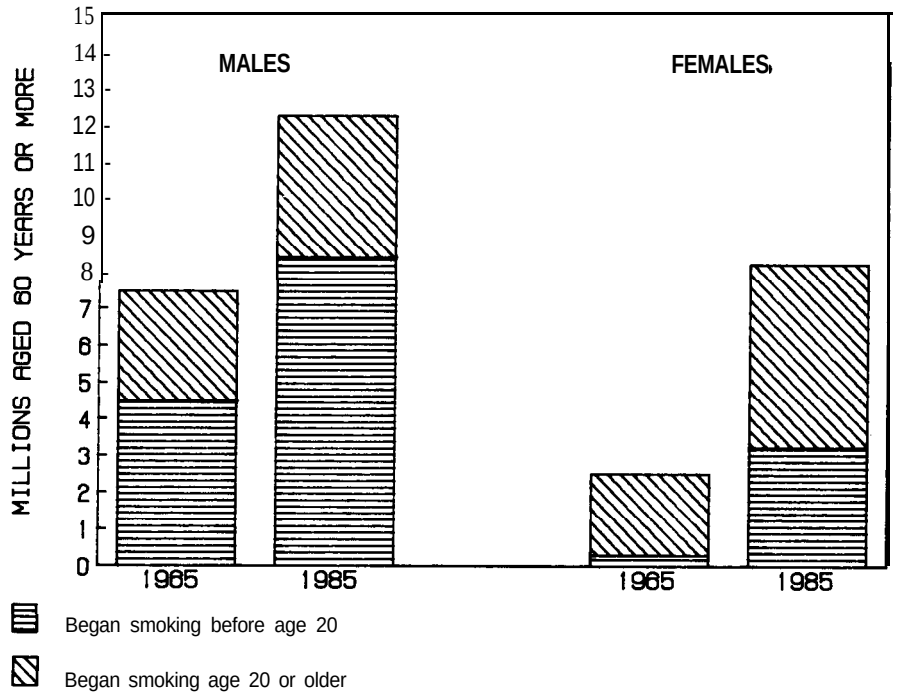

FIGURE 4.--Populations of men and women aged 60 years or more with a history of regular cigarette smoking, classified by age started to smoke regularly, United States, 1965 and 1985

SOURCE: Estimated from Harris (1983); Haenszel et al. (1956); Hammond (1966); unpublished tabulations. NHISs 1965 and 1985; unpublished tabulations, CPS 1985; and estimates of the resident populations of the United States by age and sex, 1965 and 1985 (US Bureau of the Census 1974, 1986).

cigarette smoking in 1965, about 30.8 million survived to 1985. The vertical lines show the remaining 13.3 million men who died before 1985 (standard error, 0.4 million). Likewise, among 27.2 million adult women with a smoking history in 1965 (diagonal lines and vertical lines combined), about 6.2 million died before 1985 (vertical lines). Not all of the decedents, however, died as a consequence of their cigarette use.

The horizontal lines in Figure 5 show the populations of adults at risk in 1985 who were not also at risk in 1965. The estimates are 22.6 million men and 20.0 million women. These counts do not include persons who may have taken up smoking after 1965 but died before 1985. Nor do they include smokers under age 18 in 1965 and 1985. Still, it appears that in the two-decade period following the 1964 Surgeon General's Report and the 1965 Federal Cigarette Labeling and Advertising Act, some 43 million Americans took up regular cigarette smoking, either temporarily or permanently. About two-thirds of them began to smoke by age 18.

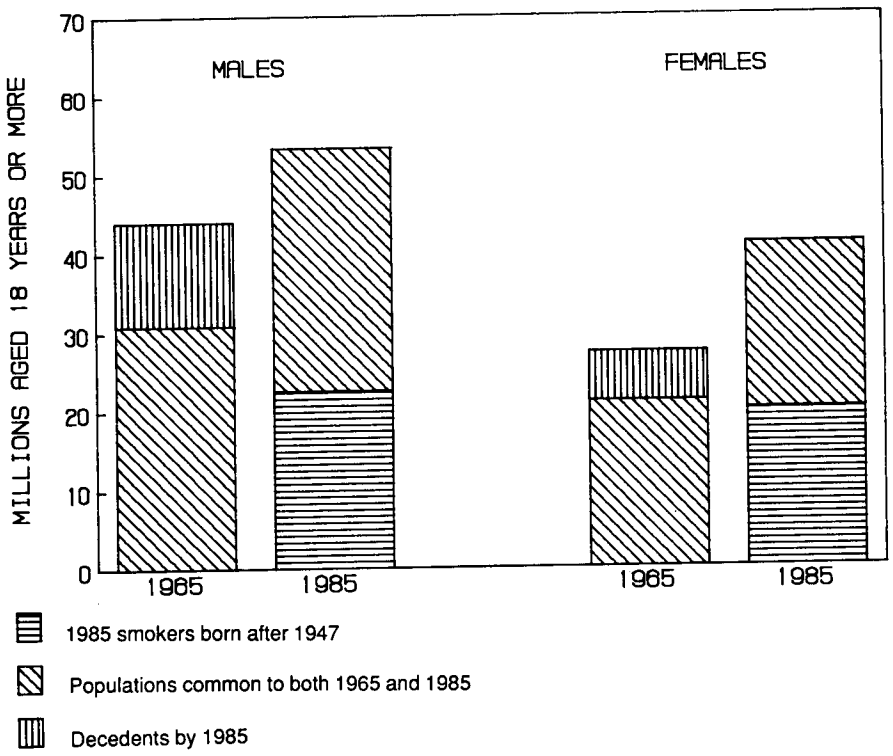

FIGURE 5.—Populations of adult men and women with a history of regular cigarette smoking, United States, 1965 and 1985

SOURCE: Estimated from Harris (1983); unpublished tabulations, NHISs 1965 and 1985; unpublished tabulations, CPS 1985; and estimates of the resident populations of the United States by age and sex, 1965 and 1985 (US Bureau of the Census 1974, 1986).

Changes in the Cigarette Product

The 1965 and 1985 population surveys did not elicit information on the type of cigarette smoked. However, there was a decline in the average tar and nicotine yield of cigarettes, at least as measured by the U.S. Federal Trade Commission (FTC) using smoking machines under standardized conditions (Chapters 2 and 5). Data on aggregate cigarette sales and other population surveys (US DHEW 1979; US DHHS 1980, 1981; Chapter 5) also show that the proportion of persons smoking filter-tipped cigarettes increased substantially. Among entrants into CPS-II in 1982, more than 90 percent were filter-tipped-cigarette smokers. In this group, was an average of 18 years of filter-tipped-cigarette smoking prior to enrollment (Stellman and Garfinkel 1986). The majority of these persons had smoked nonfilter cigarettes earlier in life.

It remains problematic whether such changes in cigarette manufacture and patterns of cigarette smoking have substantially reduced risks to cigarette smokers. There is considerable evidence that the actual reduction in the dangerous chemicals in cigarette smoke is much smaller than implied by the FTC machine measurements (US DHHS 1988a). While there is evidence that the long-term use of filter cigarettes and low-tar cigarettes may somewhat reduce the risk of lung cancers, there are considerably fewer data on a protective effect for other smoking-induced diseases (Alderson et al. 1985; Castelli et al. 1981; Hawthorne and Fry 1978; Kaufman et al. 1983; Lee and Garfinkel 1981; Lubin et al. 1984; Hammond et al. 1976; Wynder and Stellman 1979; US DHHS 1981; Wilcox et al. 1988; Stellman 1986a,b).

During the 1965-85 period, numerous chemical treatments and additives have been applied to cigarettes during tobacco curing and storage, sheet reconstitution, puffing, casing, and cigarette assembly. The chemicals include humectants, pesticides, flavorings, plasticizers, ash adhesives, and other agents. Cigarette filters, plug wraps, and tipping papers have evolved. The mix of domestic tobaccos has also changed, and oriental varieties have been added increasingly to American cigarette blends. The details of these product changes remain proprietary (US DHHS 1981).

Other Changes in the Cigarette Smoking Population

The present comparison of populations at risk in 1965 and 1985 has been confined to sex, age, and history of tobacco use. Still, there may have been other changes in the characteristics of persons who smoke cigarettes.

Surveys such as the NHIS have consistently shown a socioeconomic gradient in current cigarette use, as measured by education, occupation, and other characteristics (US DHEW 1979; US DHHS 1980; Novotny et al. 1988; US DHHS 1988a; Brackbill, Frazier, Shilling 1988; Chapter 5). There is some evidence that socioeconomic differentials in smoking rates have widened. The proportionate decline in adult smoking rates between 1965 and 1985 was highest for people who had graduated from college and lowest for those who had not completed high school (Chapter 5). Between 1970 and 1980, white-collar men and women showed proportionately greater declines in smoking rates than their blue-collar counterparts (US DHHS 1985).

Among the factors that may influence the risks of cigarette smoking are: the coexistence of untreated hypertension; elevated serum cholesterol; consumption of oral contraceptives; alcohol use; diabetes mellitus; and workplace exposure to other toxic and carcinogenic agents such as asbestos and radon daughters. With respect to these factors, it needs to be determined whether the typical cigarette user of the 1980s differs from his or her counterpart of the 1960s.

Cigarette smokers have higher rates of alcohol use, are more sedentary, and are less likely to wear seat belts (Schoenborn and Benson 1988; Williamson et al. 1986). It is unknown whether these relationships have strengthened or weakened over the years. There is evidence in the American population of declines in dietary cholesterol, in dietary saturated fat as a percentage of total calories, and in serum cholesterol levels (Havlik and Feinbeib 1979). The prevalence of untreated and inadequately treated hypertension has also declined (Havlik and Feinleib 1979). However, detailed studies of

the clustering of cigarette smoking with other risk factors for CHD are unavailable. It remains unclear whether the observed long-term declines in hypercholesterolemia and hypertension have been more or less pronounced in cigarette smokers than in nonsmokers. There is some evidence that cigarette smoking reduces therapeutic effectiveness of new pharmacologic and invasive treatments of CHD (Deanfield et al. 1984; Galan et al. 1988). Finally, in 1965, oral contraceptives were just coming into widespread use. By 1985, oral contraceptive use was prevalent among both smokers and nonsmokers (Goldbaum et al. 1987).

Those Smokers Most at Risk in 1985 Were Also Smokers in 1965

In sum, between 1965 and 1985, there have been major changes in the populations of smokers at risk for cigarette-related injury. In 1965, most men who smoked cigarettes had also used cigars and pipes. However, by 1985 the great majority smoked cigarettes exclusively. In 1965, about 40 percent of current smokers were women. By 1985, women numbered almost half of current smokers.

Moreover, the numbers of former smokers increased substantially in both sexes--especially in men. In 1965, about one-quarter of all living men (self-respondents to NHIS, age 18 or older) with a history of regular cigarette use were former smokers. By 1985, former smokers made up almost half of all living men age 18 or older who ever smoked. Finally, the two-decade interval witnessed a substantial increase in the number of women smokers reaching the age of 60 years, with a tenfold rise in the population of older women who had begun to smoke as teenagers.

These changes in the population at risk have also been observed in other, nonrandom samples of the U.S. smoking population, including a recent comparison of the 1959 entrants into CPS-I with the 1982 entrants into CPS-II (Stellman and Garfinkel 1986). The percentage of male smokers who smoked 20 or more cigarettes per day in CPS II (76 percent) was higher than in CPS-I (69 percent); the percentage of female smokers who smoked 20 or more cigarettes per day increased even more from CPS-I to CPS-II (43 percent to 61 percent).

Among the 94.4 million adults in 1985 with a history of cigarette use, about 51.8 million smoked cigarettes as adults before 1966. The youngest of these persons is now in his or her late thirties. This group represents the vast majority of persons who are now at risk for the fatal and nonfatal consequences of cigarette smoking.

Cancer Prevention Study I and Cancer Prevention Study II

CPS-I, formerly termed the American Cancer Society 25-State study, began in October 1959 and ended in October 1972. Over 1 million men and women, representing 3 percent of the population over the age of 45 years, were recruited in 1,121 counties (Hammond 1964a,b, 1966; Garfinkel 1985). Illiterate persons, institutionalized populations, itinerant workers, and illegal aliens were not recruited. More than 97 percent of enrollees were white. Enrollment was by family; an eligible family had to have one member over age 45. Once a family was eligible, every family member over the age of 35 was asked to participate. As a result of family-based recruitment, more than

three-quarters of CPS-I subjects were married. As a consequence of the eligibility rules, the age distribution of entrants peaked at 45-49 years. More than one-third of participants had at least some college education.

CPS-II was instituted in September 1982. The study, conducted in all 50 States, had the same enrollment plan and organizational structure as CPS-I. Over 1.2 million persons were enrolled. As in CPS-I, subjects were predominantly white and more educated than the general population. While 2 percent of CPS-I participants were black, the proportion increased to 4 percent in CPS-II. Still, black persons were underrepresented. Like CPS-I participants, CPS-II enrollees were predominantly over 40 years of age. Unlike CPS-I, the mode of their age distribution was 50 to 59 years (Garfinkel 1985; Stellman and Garfinkel 1986).

CPS-II is planned to continue through 1988. Preliminary results of the first 4 years of followup (1982-86) are available. For these 4 years, ascertainment of the fact of death among enrollees is thought to be virtually complete. However, as of July 1988, the cause of death had not been ascertained for about 9 percent of male deaths and 13 percent of female deaths.

Comparison of the 6-year followup (1959-65) of CPS-I and the 4-year followup of CPS-II is reported below. For computation of relative risks, cause-specific death rates for CPS-I males and females have been standardized to the age distributions of man-years and woman-years of exposure during 1965-69. Relative risks in CPS-II were likewise computed as the ratios of age-adjusted death rates, where standardization was performed with respect to the age distributions of man- and woman-years of exposure during 1982-86.

For comparison of absolute death rates (as opposed to relative risks), the age-specific rates in both studies were standardized to the age distribution of U.S. resident white males and females in 1965. For CPS-II, absolute death rates have been corrected for underascertainment of causes of death. No such correction was made for CPS-I, where death certificate retrieval is virtually complete.

No attempt has been made to correct for possible noncomparability between ICD-7 (CPS-I) and ICD-9 (CPS-II). Studies of the transition between the Seventh and Eighth Revisions of the International Classification of Diseases have shown significant noncomparability (Klebba 1975, 1982). Similar results have been reported for the transition between the Eighth and Ninth Revisions (Klebba and Scott 1980). Comparison of the Seventh and Ninth Revisions, however, suggests that the combined changes have been self-cancelling (Personal communication, J. Klebba to J. Harris, June 1988).

Both CPS-I and CPS-II are more representative of middle-class white Americans than the U.S. population as a whole. Still, the two cohorts were derived from virtually identical sampling schemes, and analysis of the entrants has shown similar demographic characteristics (Stellman and Garfinkel 1986). These considerations enhance the validity of comparisons between the American Cancer Society studies.

Nonsmokers' Death Rates

Table 3 reports a comparison of the age-adjusted death rates for the three leading causes of death from cigarette smoking: CHD; chronic obstructive pulmonary disease

(COPD); and lung cancer. For COPD and lung cancer, in particular, there has been no discernible change in nonsmokers' death rates. The relatively small changes--less than 15 percent up or down--are all statistically insignificant. The absence of significant change in nonsmokers' lung cancer rates confirms and extends the findings of Doll and Peto (1981) and Garfinkel (1981). For COPD, the table presents the first information on trends in nonsmokers' death rates.

It needs to be emphasized, however, that the statistical test for a change in lung cancer or COPD rates is of relatively low power. For COPD, there are sufficient data to have detected an increase of 53 percent or more in males and an increase of 42 percent or more in females at the 0.05 level of significance. For lung cancer, increases of more than 37 and 24 percent for males and females, respectively, were detectable as statistically significant.

In contrast to lung cancer and COPD, Table 3 shows a very marked decline in CHD death rates in nonsmokers. Over an approximate 20-year period, nonsmokers' age-adjusted death rates dropped by 64 percent in men and 69 percent in women. The observed decline in nonsmokers' CHD death rates is in keeping with the CHD decline in the general population. However, the magnitude of the decline is larger in the American Cancer Society subjects. Among U.S. white males, the age-adjusted death rate from CHD (standardized to the 1965 population distribution) declined by 41 percent during 1965-85. For U.S. white females, the decline was 40 percent (NCHS 1967 and unpublished; U.S. Bureau of the Census 1974, 1986).

TABLE 3.--Age-adjusted annual death rates per 100,000 for CHD, COPD, and lung cancer among males and females, aged 35 years or more, who never smoked regularly, 6-year followup (1959-65) of CPS-I compared with 4-year followup (1982-86) of CPS-II

Disease	Males		Females	
	CPS-I	CPS-II[ab]	CPS-I	CPS-II[ab]
CHD 420[c];410-414[e]	745 (726-775)[d]	270 (256-284)	479 (467-491)	153 (146-159)
COPD 500-502,527.1[c]; 490-492,496[e]	9.5 (7.0-12.9)	8.7 (6.5-11.7)	4.0 (3.1-5.3)	5.6 (4.5-7.0)
Lung cancer 162-163[c];162[e]	15.5 (12.5-19.3)	13.6 (10.8-17.0)	10.3 (8.9-11.9)	11.4 (9.8-13.3)

[a]For both CPS-I and CPS-II, age adjustment of rates was performed by direct standardization to the age distributions of U.S. resident white males and females, respectively, in 1965 (U.S. Bureau of the Census 1974).
[b]For CPS-II, death rates were corrected for delayed ascertainment of causes of death. Among 4,959 known deaths during 1982-86 in male nonsmokers, death certificates had not been received for 439 by June 1988. Among 10,161 known deaths in female nonsmokers. 1,411 had not been received.
[c]CPS-I coding, International Classification of Diseases, Seventh Revision.
[d]Numbers in parentheses are 95-percent confidence intervals.
[e]CPS-II coding, International Classification of Diseases, Ninth Revision.
SOURCE: Unpublished tabulations, American Cancer Society.

Current Cigarette Smokers' Death Rates: Lung Cancer

Figures 6 and 7, respectively, show changes in the age-specific lung cancer death rates of men and women who described themselves as regular cigarette smokers on the original questionnaire for each prospective study. The death rates, depicted in each figure on a logarithmic scale, apply to all such current smokers. No adjustment has been made for differences in the number of cigarettes smoked or duration of cigarette use.

The age-incidence curves in both figures show a striking crossover effect. Among older male smokers, especially those aged 70 years or more, lung cancer death rates in CPS-II exceed those in CPS-I twofold to fourfold. By contrast, among younger male smokers, especially those less than 50 years old, CPS-II death rates are about 30 to 40 percent lower. The observed crossover phenomenon appears to be consistent with long-term changes in cigarette smoke exposure among successive cohorts. The increase in lung cancer among older male smokers reflects their increased frequency of cigarette use and increased cigarette smoking in early life. The decline in lung cancer among

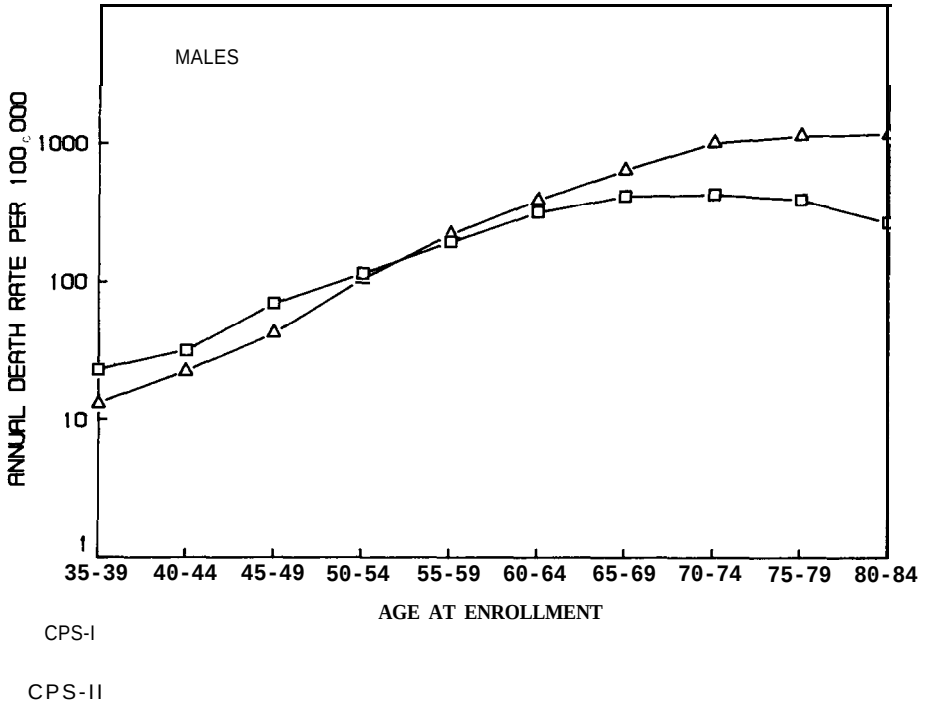

FIGURE 6.--Age-specific death rates (log scale) for lung cancer, male current cigarette smokers aged 35-84 years; 6-year followup of CPS-I (1959-65), compared with 4-year followup of CPS-II (1982-86)

SOURCE: Unpublished tabulations, American Cancer Society. Estimates for CPS-II are preliminary.

younger men may reflect their increased use of filter-tipped and low-tar cigarettes. Most currently smoking men aged 35 to 39 years in CPS-II, for example, were likely to have been lifelong filter-tipped cigarette smokers.

An even more striking crossover is shown for female current cigarette smokers in Figure 7. In particular, the age of crossover comes somewhat earlier. Among women smokers aged 45 years or more, lung cancer death rates have increased fourfold to sevenfold. (There were no deaths and a small number of person-years of exposure at ages 7.5 or more in CPS-I.) By contrast, lung cancer death rates in the very youngest cohorts, aged 35 to 44 years, have declined by 35 to 55 percent. As in the case of men, the crossover appears to reflect differential trends in cigarette smoking among successive cohorts of women.

Current Cigarette Smokers' Death Rates: Coronary Heart Disease

Figure 8 shows the proportional decline from CPS-I to CPS-II in the age-adjusted CHD death rates of current smokers and nonsmokers. The relative declines are depicted

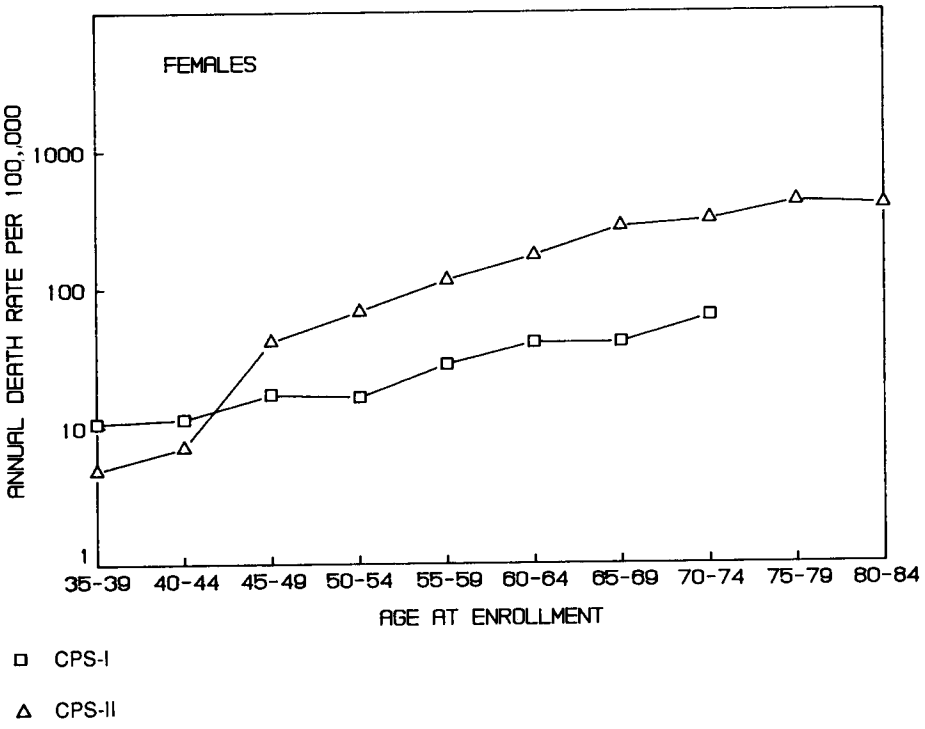

FIGURE 7.—Age-specific death rates (log scale) for lung cancer, female current cigarette smokers aged 35–84 years; 6-year followup of CPS-I (1959–65), compared with 4-year followup of CPS-II (1982–86)

SOURCE: Unpublished tabulations, American Cancer Society. Estimates for CPS-II are preliminary.

separately for men and women, and for persons younger than 65, and 65 and older. CHD death rates have declined in both cigarette smokers and nonsmokers. For the predominantly white, middle-class populations under study in CPS-I and CPS-II, the overall decline among smokers and nonsmokers was greater than observed for the U.S. white population.

Still, the declines in CHD mortality rates among nonsmokers were notably greater than among current cigarette smokers. The disparity is seen at all ages, but appears somewhat greater among younger persons. In contrast to lung cancer (Figures 6 and 7), no crossover in age-incidence curves is observed. The increasing smoker-non-smoker disparity at younger ages argues against a significant salutary effect of lifelong filter-tipped cigarette use. The possibility that changes in other coronary risk factors among cigarette smokers may explain their reduced decline in CHD rates needs further investigation.

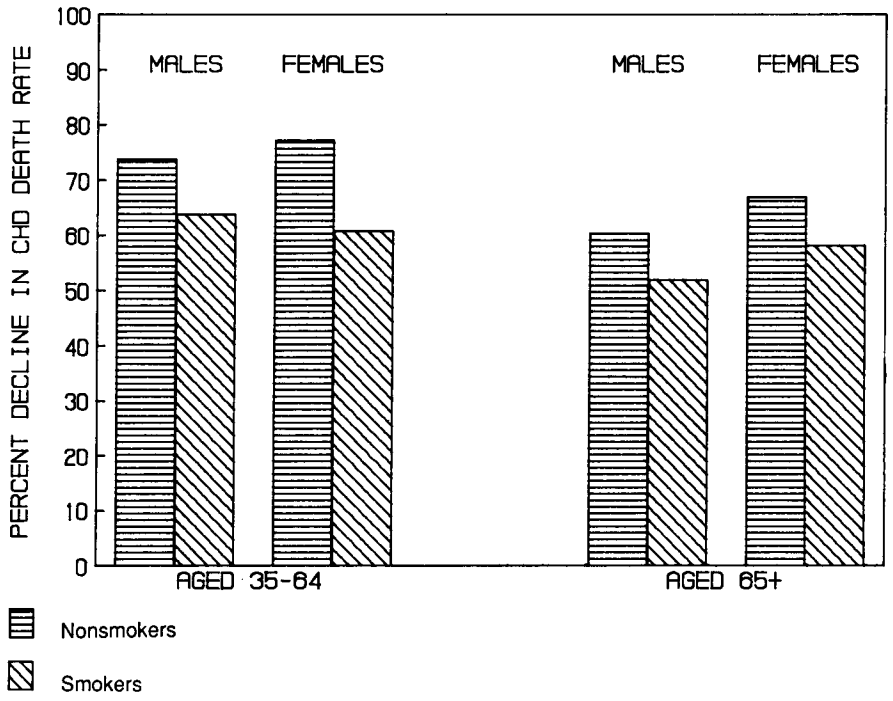

FIGURE 8.—Percentage decline in age-adjusted death rates for CHD; 6-year followup of CPS-I (1959–65), compared with 4-year followup of CPS-II (1982–86)

SOURCE: Unpublished tabulations, American Cancer Society. Estimates for CPS-II are preliminary.

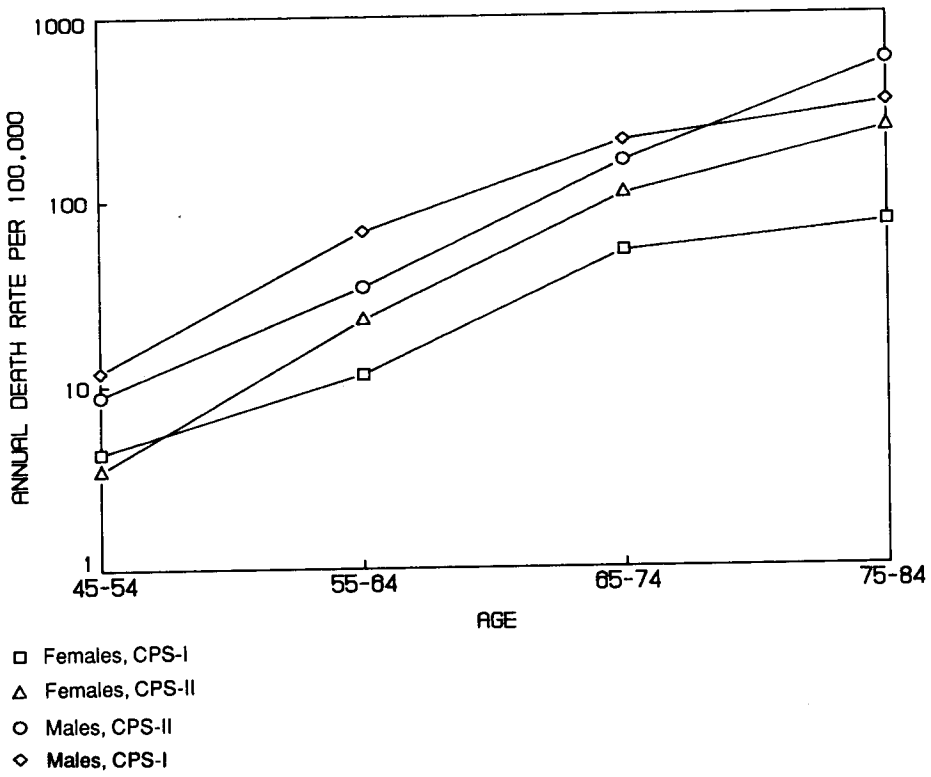

FIGURE 9.—Age-specific death rates for COPD, male and female current cigarette smokers aged 45–84 years; 6-year followup of CPS-I (1959–65), compared with 4-year followup of CPS-II (1982–86)

SOURCE: Unpublished tabulations, American Cancer Society. Estimates for CPS-II are preliminary.

Current Cigarette Smokers' Death Rates: Chronic Obstructive Pulmonary Disease

Figure 9 gives corresponding changes in age-specific death rates for COPD. In this figure, the ages are grouped into 10-year rather than 5-year age ranges as in Figures 6 and 7. For male smokers, there has been a reduction in COPD death rates for ages 45 to 74 years. For female smokers over 55 years old, there has been about a twofold to threefold increase in COPD rates.

Estimated Relative Risks from CPS-I and CPS-II

For men and women, respectively, Tables 4 and 5 depict estimated relative risks in the 6-year followup of CPS-I for all-cause mortality and for 14 specific causes of death (15 causes for women, including cervical cancer). For men in Table 4, the estimated

relative risks for current and former cigarette smokers are given separately. For women in Table 5, the numbers of deaths and person-years of exposure among former smokers were too small to give reliable death rates for many causes. Accordingly, in conformity with earlier reports of CPS-I mortality, the death rates for current smokers are compared with those of women with any history of regular cigarette use, past or present.

For both men and women, the estimates in Tables 4 and 5 are in accord with earlier reports on CPS-I mortality (Garfinkel 1980b; Hammond 1964a,b, 1966, 1972; Hammond and Garfinkel 1969; Hammond and Seidman 1980). Among men, former smokers have lower mortality ratios. In both sexes, relative risks for CHD are higher at younger ages. Both sexes, but to a greater extent, men, show elevated risks of other cardiovascular diseases including stroke, hypertensive heart disease, and aortic aneurysm. In both sexes, smokers' death rates are higher for bronchitis and emphysema and for seven cancers including lung cancer. The relative risk of lung cancer among current smokers in CPS-I is about 11.3 for men and 2.7 for women.

The results for CPS-II, given in Tables 6 and 7, show substantial changes in the mortality risk of cigarette smoking over two decades. The all-cause relative risk for men has increased from 1.8 in CPS-I to 2.3 in CPS-II. For women, it has risen from 1.2 to 1.9. These increases in overall mortality are not an artifact of the method of age adjustment, because CPS-II contained proportionately fewer person-years of exposure at the youngest ages than CPS-I.

As reflected in Table 6 and Table 7, the relative risks for CHD death have increased for both men and women. The relative risks for men, in particular, are consistent with those reported from recent case-control studies (Kaufman et al. 1983; Rosenberg et al. 1985) and from the followup of the Multiple Risk Factor Intervention Trial (MRFIT) cohort, as described in Chapter 2. The markedly elevated relative risks for younger women in Table 7 are consistent with those reported in a recent case-control study (Slone et al. 1978) and in a prospective study of 120,000 female nurses (Willett et al. 1987). Such consistencies across epidemiologic studies--especially cohort and case-control studies reported during the 1980s--argue against any appreciable bias in the 4-year preliminary results of CPS-II given in Tables 6 and 7.

Tables 6 and 7 show consistently increased relative risks for cerebrovascular lesions among both men and women, particularly in the younger age groups. Among women under 65 years old, the estimated relative risk of death from stroke is 4.8, with a 95-percent confidence range of 3.5 to 6.5. The observed increases in risk for current smokers are reduced in former smokers.

The finding of an elevated risk of cerebrovascular disease among cigarette smokers is not new. Elevated death rates from stroke were reported in CPS-I (Hammond 1966; Hammond and Garfinkel 1969) and are reproduced in Tables 4 and 5. The 1983 Surgeon General's Report noted the association between stroke and cigarette use; no data on the effect of smoking cessation were available (US DHHS 1983). A recent prospective study of 8,000 men of Japanese origin (Abbott et al. 1986) showed an elevated risk of thromboembolic and hemorrhagic strokes among cigarette smokers. While there was no clear trend of increasing risk with higher daily smoking rates, subjects who quit smoking had reduced risks compared with continuing smokers. In the prospective study of 120,000 female nurses, Colditz et al. (1988) found a dose-response relationship be-

TABLE 4.—Estimated relative risks for current and former smokers of cigarettes, males aged 35 years or more, 6-year (1959–65) followup of American Cancer Society 25-State study (CPS-I)

Underlying cause of death	Current smokers[a]	Former smokers[a]
All causes	1.80 (1.75–1.85)[b]	1.38 (1.33–1.42)[b]
CHD, age ≥35 (420)[c]	1.83 (1.76–1.91)	1.42 (1.34–1.49)
CHD, age 35–64[d] (420)	2.25 (2.13–2.39)	1.56 (1.45–1.68)
CHD, age ≥65 (420)	1.39 (1.30–1.48)	1.27 (1.17–1.37)
Hypertensive Heart Disease (440–443)	1.63 (1.36–1.96)	1.19 (0.94–1.51)
Cerebrovascular Lesions, age ≥35 (330–334)	1.37 (1.25–1.49)	0.96 (0.85–1.08)
Cerebrovascular Lesions, age 35–64 (330–334)	1.79 (1.55–2.08)	1.02 (0.83–1.25)
Cerebrovascular Lesions, age ≥65 (330–334)	1.15 (1.02–1.30)	0.93 (0.80–1.08)
Aortic Aneurysm, Non-Syphilitic (451)	4.11 (3.13–5.40)	2.40 (1.73–3.34)
Ulcer, Duodenal, Gastric, and Jejunal (540–542)	3.06 (2.24–4.18)	1.49 (0.98–2.27)
Influenza and Pneumonia (480–481, 490–493)	1.82 (1.45–2.27)	1.62 (1.24–2.12)
Bronchitis and Emphysema (500–502, 527.1)	8.81 (6.40–12.13)	10.20 (7.34–14.17)
Cancer, Lip, Oral Cavity, and Pharynx (140–148)	6.33 (3.60–11.13)	2.73 (1.36–5.49)
Cancer, Esophagus (150)	3.62 (2.02–6.48)	1.28 (0.53–3.08)
Cancer, Pancreas (157)	2.34 (1.81–3.02)	1.30 (0.92–1.84)
Cancer, Larynx (161)	10.00 (3.51–28.51)	8.60 (2.87–25.74)
Cancer, Lung (162–163)	11.35 (9.10–14.15)	4.96 (3.86–6.38)
Cancer, Kidney (180)	1.84 (1.23–2.76)	1.79 (1.11–2.87)
Cancer, Bladder, Other Urinary Organs (181)	2.90 (2.01–4.18)	1.75 (1.07–2.87)

NOTE: Based upon 1,692,652 man-years of exposure among male subjects who never smoked regularly, or who smoked only cigarettes, present or past. Relative risks, estimated with respect to men who never smoked regularly, have been directly standardized to the age distribution of all man-years of exposure.
[a]Refers to cigarette smoking status at enrollment (October 1959–March 1960).
[b]Numbers in parentheses are 95-percent confidence intervals, computed on the assumption that the logarithm of relative risk was normally distributed.
[c]All disease codes refer to International Classification of Diseases, Seventh Revision.
[d]When an age range is given, it refers to the age at enrollment in 1959.
SOURCE: Unpublished tabulations, American Cancer Society.

TABLE 5.—Estimated relative risks for current cigarette smokers and for all subjects with a history of regular cigarette smoking, females aged 35 years or more, 6-year (1959–65) followup of American Cancer Society 25-State study (CPS-I)

Underlying cause of death	Current smokers[a]	Current and former smokers[a]
All causes	1.23 (1.18–1.28)[b]	1.24 (1.20–1.28)[b]
CHD, age ≥35 (420)[c]	1.40 (1.29–1.51)	1.38 (1.29–1.74)
CHD, age 35–64[d] (420)	1.81 (1.67–1.97)	1.74 (1.61–1.89)
CHD, age ≥65 (420)	1.24 (1.11–1.39)	1.25 (1.14–1.37)
Hypertensive Heart Disease (440–443)	1.31 (1.04–1.66)	1.27 (1.04–1.55)
Cerebrovascular Lesions, age ≥35 (330–334)	1.19 (1.06–1.35)	1.26 (1.13–1.80)
Cerebrovascular Lesions, age 35–64 (330–334)	1.92 (1.69–2.18)	1.80 (1.59–2.03)
Cerebrovascular Lesions, age ≥65 (330–334)	0.97 (0.81–1.16)	1.09 (0.95–1.26)
Aortic Aneurysm, Non-Syphilitic (451)	4.64 (3.00–7.20)	3.67 (2.46–5.48)
Ulcer, Duodenal, Gastric, and Jejunal (540–542)	1.37 (0.81–2.31)	1.52 (0.96–2.41)
Influenza and Pneumonia (480–481, 490–493)	0.91 (0.59–1.41)	0.96 (0.69–1.33)
Bronchitis and Emphysema (500–502, 527.1)	5.89 (3.97–8.76)	5.85 (4.02–8.53)
Cancer, Lip, Oral Cavity, and Pharynx (140–148)	1.96 (1.14–3.39)	1.89 (1.16–3.08)
Cancer, Esophagus (150)	1.94 (1.02–3.69)	2.15 (1.09–4.23)
Cancer, Pancreas (157)	1.39 (1.04–1.86)	1.38 (1.07–1.78)
Cancer, Larynx (161)	3.81 (0.78–18.52)	3.10 (0.65–14.99)
Cancer, Lung (162–163)	2.69 (2.14–3.37)	2.59 (2.04–3.30)
Cancer, Cervix Uteri (171)	1.10 (0.83–1.47)	1.32 (1.02–1.71)
Cancer, Kidney (180)	1.43 (0.89–2.31)	1.47 (0.97–2.23)
Cancer, Bladder, Other Urinary Organs (181)	2.87 (1.74–4.74)	2.31 (1.45–3.67)

NOTE: Based upon 3,325,989 woman-years of exposure among subjects who never smoked regularly, or who smoked only cigarettes, present or past. Relative risks, estimated with respect to women who never smoked regularly, have been directly standardized to the age distribution of all woman-years of exposure.
[a]Refers to cigarette smoking status at enrollment (October 1959–March 1960).
[b]Numbers in parentheses are 95-percent confidence intervals, computed on the assumption that the logarithm of relative risk was normally distributed.
[c]All disease codes refer to International Classification of Diseases, Seventh Revision.
[d]When an age range is given, it refers to the age at enrollment in 1959.
SOURCE: Unpublished tabulations, American Cancer Society.

TABLE 6.—Estimated relative risks for current and former smokers of cigarettes, males aged 35 years or more, 4-year (1982–86) followup of American Cancer Society 50-State study (CPS-II)

Underlying cause of death	Current smokers[a]	Former smokers[a]
All causes	2.34 (2.26–2.43)[b]	1.58 (1.53–1.64)[b]
CHD, age ≥35 (410–414)[c]	1.94 (1.80–2.08)	1.41 (1.33–1.50)
CHD, age 35–64[d] (410–414)	2.81 (2.49–3.18)	1.75 (1.55–1.99)
CHD, age ≥65 (410–414)	1.62 (1.48–1.77)	1.29 (1.20–1.38)
Other Heart Disease[e] (390–398, 401–405, 415–417, 420–429)	1.85 (1.63–2.10)	1.32 (1.18–1.48)
Cerebrovascular Lesions, age ≥35 (430–438)	2.24 (1.88–2.67)	1.29 (1.10–1.51)
Cerebrovascular Lesions, age 35–64 (430–438)	3.67 (2.51–5.36)	1.38 (0.91–2.07)
Cerebrovascular Lesions, age ≥65 (430–438)	1.94 (1.58–2.38)	1.27 (1.07–1.50)
Other Circulatory Disease[f] (440–448)	4.06 (3.08–5.35)	2.33 (1.81–3.01)
COPD (490–492,496)	9.65 (7.00–13.30)	8.75 (6.48–11.80)
Other Respiratory Disease[g] (010–012, 480–489,493)	1.99 (1.52–2.61)	1.56 (1.25–1.95)
Cancer, Lip, Oral Cavity, Pharynx (140–149)	27.48 (9.96–75.83)	8.80 (3.15–24.59)
Cancer, Esophagus (150)	7.60 (3.81–15.17)	5.83 (3.02–11.25)
Cancer, Pancreas (157)	2.14 (1.62–2.82)	1.12 (0.86–1.45)
Cancer, Larynx (161)	10.48 (3.61–30.43)	5.24 (1.83–14.99)
Cancer, Lung (162)	22.36 (17.77–28.13)	9.36 (7.43–11.77)
Cancer, Kidney (189)	2.95 (1.92–4.54)	1.95 (1.31–2.90)
Cancer, Bladder, Other Urinary Organs (188)	2.86 (1.85–4.44)	1.90 (1.28–2.82)

NOTE: Preliminary estimates, based upon 1,491,791 man-years of exposure among male subjects who never smoked regularly, or who smoked only cigarettes, present or past. Relative risks, estimated with respect to men who never smoked regularly, have been directly standardized to the age distribution of all man-years of exposure.
[a]Refers to cigarette smoking status at enrollment (September 1982).
[b]Numbers in parentheses are 95-percent confidence intervals, computed on the assumption that the logarithm of relative risk was normally distributed.
[c]All disease codes refer to International Classification of Diseases, Ninth Revision.
[d]When an age range is given, it refers to the age at enrollment in 1982.
[e]Includes Hypertensive Heart Disease (401–404).
[f]Includes Aortic Aneurysm, Non-Syphilitic, and General Arteriosclerosis (440–441).
[g]Includes Influenza and Pneumonia (480–487).
SOURCE: Unpublished tabulations, American Cancer Society.

TABLE 7.—Estimated relative risks for current and former cigarette smokers, females aged 35 years or more, 4-year (1982–86) followup of American Cancer Society 50-State study (CPS-II)

Underlying cause of death	Current smokers[a]	Former smokers[a]
All causes	1.90 (1.82–1.98)[b]	1.32 (1.27–1.37)[b]
CHD, age ≥35 (410–414)[c]	1.78 (1.62–1.97)	1.31 (1.19–1.44)
CHD, age 35–64[d] (410–414)	3.00 (2.50–3.59)	1.43 (1.15–1.77)
CHD, age ≥65 (410–414)	1.60 (1.42–1.80)	1.29 (1.16–1.43)
Other Heart Disease[e] (390–398, 401–405, 415–417, 420–429)	1.69 (1.44–1.99)	1.16 (1.00–1.34)
Cerebrosvascular Lesions, age ≥35 (430–438)	1.84 (1.56–2.16)	1.06 (0.88–1.27)
Cerebrovascular Lesions, age 35–64 (430–438)	4.80 (3.52–6.54)	1.41 (0.94–2.13)
Cerebrovascular Lesions, age ≥65 (430–438)	1.47 (1.19–1.81)	1.01 (0.83–1.24)
Other Circulatory Disease[f] (440–448)	3.00 (2.20–4.08)	1.34 (0.95–1.90)
COPD (490–492,496)	10.47 (7.78–14.09)	7.04 (5.33–9.30)
Other Respiratory Disease[g] (010–012,480–489,493)	2.18 (1.60–2.97)	1.38 (1.04–1.84)
Cancer, Lip, Oral Cavity, Pharynx (140–149)	5.59 (3.15–9.91)	2.88 (1.57–5.26)
Cancer, Esophagus (150)	10.25 (4.94–21.27)	3.16 (1.45–6.85)
Cancer, Pancreas (157)	2.33 (1.77–3.08)	1.78 (1.37–2.30)
Cancer, Larynx (161)	17.78 (3.45–91.74)	11.88 (2.46–57.34)
Cancer, Lung (162)	11.94 (9.99–14.26)	4.69 (3.86–5.70)
Cancer, Cervix Uteri (180)	2.14 (1.06–4.30)	1.94 (0.97–3.87)
Cancer, Kidney (189)	1.41 (0.86–2.30)	1.16 (0.72–1.87)
Cancer, Bladder, Other Urinary Organs (188)	2.58 (1.31–5.08)	1.85 (1.00–3.42)

NOTE: Preliminary estimates, based upon 2,418,909 woman-years of exposure among female subjects who never smoked regularly, or who smoked only cigarettes, present or past. Relative risks, estimated with respect to women who never smoked regularly, have been directly standardized to the age distribution of all woman-years of exposure.
[a]Refers to cigarette smoking status at enrollment (September 1982).
[b]Numbers in parentheses are 95-percent confidence intervals, computed on the assumption that the logarithm of relative risk was normally distributed.
[c]All disease codes refer to International Classification of Diseases, Ninth Revision.
[d]When an age range is given, it refers to the age at enrollment in 1982.
[e]Includes Hypertensive Heart Disease (401–404).
[f]Includes Aortic Aneurysm, Non-Syphilitic, and General Arteriosclerosis (440–441).
[g]Includes Influenza and Pneumonia (480–487).
SOURCE: Unpublished tabulations, American Cancer Society.

tween cigarette use and risk of stroke. They also noted a slight increase in risk among former cigarette smokers, especially for the first 2 years after cessation. The preliminary results from CPS-II, reported in Tables 6 and 7, further support a causal role for cigarette smoking in stroke.

The preliminary results of CPS-II also show significantly higher relative risks for cancers of the lip, oral cavity and pharynx, esophagus, and lung, as compared with CPS-I. The computed relative risk for lung cancer death has increased to 22 in men and 12 in women. While the relative risks for COPD death have not changed significantly among men, there is a trend toward increasing risk among women. The available data from CPS-II do not permit identification of specific mortality risks for hypertensive heart disease, aortic aneurysm, and influenza and pneumonia, as in CPS-I. However, among broader categories of cardiovascular and nonneoplastic respiratory disease, increased risks are likewise found in CPS-II.

Endocrine and Sex-Related Cancers in Women

A protective effect of smoking on cancer of the endometrium has been suggested in a recent case-control study (Lesko et al. 1985). For CPS-I, the relative risk for cancers of the uterine corpus (ICD-7 Codes 172-174) among current smokers was 0.94 (95-percent confidence interval, 0.57 to 1.53). Preliminary results for CPS-II suggest a reduced relative risk for endometrial cancer (ICD-9 Code 182).

Recent data on a possible protective effect of smoking for breast cancer have been contradictory (See Chapter 2; Rosenberg et al. 1984). For CPS-I, the relative risk for breast cancer (ICD-7 Code 170) among current smokers was 0.88 (95-percent confidence interval, 0.77 to 1.01), while the relative risk among former smokers was 1.20 (95-percent confidence interval, 1.15 to 1.35). Preliminary data from CPS-II have likewise been contradictory.

An increased risk of cervical cancer among cigarette smokers has been reported in case-control studies (LaVecchia et al. 1986; Nischan, Ebeling, Schindler 1988). For CPS-I, the relative risk for cervical cancer (ICD-7 Code 171) was 1.10 (95-percent confidence interval, 0.83 to 1.47). Data from CPS-II show a twofold increase in cervical cancer mortality among current smokers (relative risk 2.14, 95-percent confidence interval 1.06 to 4.30).

Summary

The relative risks for current smokers for selected comparable disease categories causally related to smoking in CPS-I and CPS-II are summarized and listed side by side in Table 8. These comparisons show substantial increases in the risk of death due to smoking for most of the disease categories listed between the years 1959 and 1965 and 1982 and 1986. Statistically significant increases in relative risks occurred in those disease categories for which 95-percent confidence limits around the estimated relative risks do not overlap between CPS-I and CPS-II. Compared with men during this period, women experienced greater increases in the relative risks of cerebrovascular lesions (ages 35 to 64 years), COPD, laryngeal cancer, and lung cancer.

TABLE 8.—Summary of estimated relative risks for current cigarette smokers, major disease categories causally related to cigarettes, males and females aged 35 years and older, CPS-I (1959–65) and CPS-II (1982–86)

Underlying cause of death[a]	Males		Females	
	CPS-I	CPS-II	CPS-I	CPS-II
CHD, age ≥35	1.83	1.94	1.40	1.78[b]
CHD, age 35–64	2.25	2.81[b]	1.81	3.00[b]
Cerebrovascular Lesions, age ≥35	1.37	2.24[b]	1.19	1.84[b]
Cerebrovascular Lesions, age 35–64	1.79	3.67[b]	1.92	4.80[b]
COPD	8.81	9.65	5.89	10.47
Cancer, Lip, Oral Cavity, and Pharynx	6.33	27.48	1.96	5.59
Cancer, Esophagus	3.62	7.60	1.94	10.25[b]
Cancer, Pancreas	2.34	2.14	1.39	2.33
Cancer, Larynx	10.00	10.48	3.81	17.78
Cancer, Lung	11.35	22.36[b]	2.69	11.94[b]

[a]See Tables 4–7 for International Classification of Disease codes.
[b]95-percent confidence intervals do not overlap between CPS-I and CPS-II.
SOURCE: Tables 4–7.

Smoking-Attributable Mortality in the United States, 1965 and 1985

Table 9 reports the attributable risks a from cigarette smoking during the year 1965. Ten causes of death are considered: CHD, COPD, cerebrovascular disease, and cancers of seven sites. The computations are based upon the age-adjusted relative risks reported in CPS-I and the prevalence rates reported in the 1965 NHIS. For men, the age-adjusted relative risks among present and past cigarette smokers with a history of pipe or cigar use were slightly lower than those for present and past smokers of cigarettes exclusively. While the latter are reported for comparison in Table 4, the former were used in the attributable risk computations. In 1965, as shown in Figure 2, about two-thirds of men with a history of regular cigarette smoking were also exposed to pipe or cigar smoke. (As noted in Note b of Table 10 below, the use of relative risks derived from the death rates of men who smoked cigarettes exclusively resulted in about a 5-percent increase in attributable deaths for 1965.) For women, the computation of attributable risks in 1965 did not distinguish between current and former smokers.

TABLE 9.—Estimated attributable risks for 10 selected causes of death from cigarette smoking, males and females, United States, 1965

Cause of death	Males[a] (%)	Females[b] (%)
CHD, age 35–64	42 (40–45)[c]	26 (23–30)
CHD, age ≥65	11 (9–14)	3.3 (2.1–5.1)
COPD	84 (79–88)	67 (57–76)
Cancer of lip, oral cavity, and pharynx	74 (59–85)	27 (12–51)
Cancer of larynx	84 (61–94)	47 (8–90)
Cancer of esophagus	57 (36–76)	14 (6–29)
Cancer of lung	86 (82–88)	40 (31–50)
Cancer of pancreas	41 (30–53)	14 (6–30)
Cancer of bladder	53 (39–66)	36 (20–56)
Cancer of kidney	36 (19–56)	17 (5–42)
Cerebrovascular disease, age 35–64	28 (21–36)	28 (22–33)
Cerebrovascular disease, age ≥65	2.0 (0.6–6.6)	1.3 (0.2–6.5)

[a]For males, computations based on prevalence rates in Table 2 and relative risks for male current and former cigarette smokers, with or without a history of pipe and cigar smoking, derived from CPS-I.

[b]For females, attributable risks computed from prevalence rates in Table 2 and relative risks for all female smokers, past and present, in Table 5.

[c]Numbers in parentheses are 95-percent confidence intervals.

In 1965, as Table 9 reveals, cigarette smoking was responsible for 42 percent of CHD deaths among younger men and 26 percent of deaths among younger women. For COPD deaths at all ages, the smoking-attributable risks were 84 percent for men and 67 percent for women. For lung cancer, the respective attributable risks were 86 percent and 40 percent for men and women. With the exception of deaths from stroke among younger persons, attributable risks were markedly higher for men.

Table 10 reports the corresponding smoking-attributable deaths, *A*, during the year 1965. Attributable deaths were computed by multiplying the attributable risk percentages in Table 9 by the corresponding cause-specific death rates among persons aged 20

TABLE 10.—Estimated deaths (in thousands) attributable to cigarette smoking, 10 selected causes, males and females, United States, 1965

Cause of death	Males	Females
CHD, age <65	51 (48–54)[a]	9.5 (8.2–10.8)
CHD, age ≥65	25 (20–30)	6.0 (3.9–9.4)
COPD	16 (15–17)	2.3 (2.0–2.7)
Cancer of lip, oral cavity, and pharynx	3.6 (2.9–4.2)	0.4 (0.2–0.8)
Cancer of larynx	1.9 (1.4–2.2)	0.1 (0.02–0.3)
Cancer of esophagus	2.4 (1.5–3.2)	0.1 (0.2–0.8)
Cancer of lung	35 (34–36)	3.1 (2.4–3.8)
Cancer of pancreas	3.8 (2.8–4.9)	0.9 (0.4–2.0)
Cancer of bladder	3.0 (2.2–3.7)	1.0 (0.5–1.5)
Cancer of kidney	1.2 (0.7–1.9)	0.3 (0.1–1.8)
Cerebrovascular disease, age <65	5.5 (4.2–7.2)	4.7 (3.8–5.6)
Cerebrovascular disease, age ≥65	1.5 (0.4–4.8)	1.0 (0.2–5.9)
Ten causes	150[b] (143–157)	30 (26–34)

NOTE: Computed from Table 9 and tabulations of deaths at ages 20 years or more by cause for 1965 (NCHS 1967). Sums may not equal totals because of rounding.

[a]Numbers in parentheses are 95-percent confidence intervals.

[b]When the attributable risk estimates given in Note a of Table 9 were used, the total attributable deaths for males were 158,000 (95-percent confidence interval, 151,000 to 166,000). Approximately two-thirds of the 8,000 additional deaths were from CHD.

years or more. For the 10 causes combined, cigarette smoking was responsible for 150,000 deaths among men and 30,000 deaths among women in 1965.

Among men, CHD deaths made up 51 percent of smoking-attributable mortality for the 10 causes combined. This proportion is consistent with the estimate of 45 percent reported by the 1964 Advisory Committee to the Surgeon General for excess mortality from all causes (US PHS 1964). Similarly, lung cancer accounted for 23 percent of the smoking-attributable mortality for the 10 causes combined-again consistent with the

1964 Report's estimate of 16 percent of deaths from all causes. Among women, CHD deaths made up 52 percent and lung cancer 10 percent of the smoking-attributable mortality from the 10 causes combined.

Table 11 shows the estimated attributable risks *a* from cigarette smoking for the year 1985. For comparability with the 1965 calculations, the same 10 causes of death are considered. The computations are based upon the relative risks reported in CPS-II and the prevalence rates reported in the 1985 NHIS. For men, the computations employed the relative risks for past and present smokers of cigarettes exclusively, as shown in Table 6. As Figure 2 indicates, the proportion of male smokers who used other forms

TABLE 11.—Estimated attributable risks for 10 selected causes of death from cigarette smoking, males and females, United States, 1985

Cause of death	Males (%)	Females (%)
CHD, age <65	45 (40–50)[a]	41 (34–48)
CHD, age ≥65	21 (17–26)	12 (9–15)
COPD	84 (78–88)	79 (73–83)
Cancer of lip, oral cavity, and pharynx	92 (79–97)	61 (45–76)
Cancer of larynx	81 (57–93)	87 (56–97)
Cancer of esophagus	78 (62–89)	75 (57–87)
Cancer of lung	90 (88–92)	79 (75–82)
Cancer of pancreas	29 (18–43)	34 (25–44)
Cancer of bladder	47 (31–63)	37 (18–61)
Cancer of kidney	48 (32–64)	12 (3–43)
Cerebrovascular disease, age <65	51 (36–65)	55 (45–65)
Cerebrovascular disease, age ≥65	24 (16–35)	6 (2–14)

NOTE: Computed from Tables 2, 6, and 7. For adult men under 65, the proportions of current and former cigarette smokers in 1985 were, respectively, 34.7 and 25.8 percent. For men 65 or older, the prevalences of current and former cigarette smoking were, respectively, 19.4 and 51.1 percent. For adult women under 65, the corresponding proportions were 30.1 and 16.5 percent; for adult women 65 or older, 12.6 and 19.6 percent.

[a]Numbers in parentheses are 95-percent confidence intervals.

of tobacco was too small to affect significantly the results for 1985. For women, relative risks for current and former cigarette smokers were employed (Table 7).

Comparison of Tables 9 and 11 reveals significant increases in attributable risk from 1965-85. In 1985, smoking accounted for 21 percent of CHD deaths in older men, compared with 11 percent in 1965. The attributable risks for cancers of the lip, oral cavity and pharynx, esophagus, and lung increased significantly.

Changes in the attributable risk estimates for women are even more striking. Among younger women, smoking now accounts for an estimated 41 percent of CHD deaths and an estimated 55 percent of lethal strokes, compared with 26 and 28 percent, respectively, in 1965. Among women of all ages, 79 percent of lung cancers are attributable to cigarette use (see Table 11).

Overall, smoking accounted for 86.7 percent of all lung cancer deaths (95-percent confidence interval 84.9 to 88.4), 81.8 percent of all COPD deaths (95-percent confidence interval 78.3 to 85.3), and 21.5 percent of all CHD deaths (95-percent confidence interval 19.4 to 23.4). In addition, smoking accounted for 18.0 percent of all stroke deaths (95-percent confidence interval 14.2 to 22.9).

Table 12 reports estimated smoking-attributable deaths for the 10 causes during 1985. Total deaths have increased to 231,000 for men and 106,000 for women. As opposed to 1965, CHD in men now accounts for only one-third of the smoking-attributable mortality from the 10 causes combined. The proportion of these attributable deaths due to lung cancer has increased to one-third. Likewise, among women, smoking-attributable CHD fatalities now account for one-third of the 10-cause total; the relative importance of smoking-induced cancer fatalities has also increased.

The total 10-cause smoking-attributable mortality for 1985 was 337,000 deaths, compared with 183,000 in 1965. A portion of the observed 1965-85 increase, however, was the result of population growth. In addition, there were increases in the proportion of elderly persons who would be more at risk for smoking-induced death. For men and women, respectively, Figures 10 and 11 show the results of a correction for population increase and population aging. In each figure, three quantities are shown for each of four categories of smoking-attributable mortality: CHD deaths under age 65; CHD deaths age 65 years or more; COPD deaths; and lung cancer deaths. The first quantity is the estimated smoking-attributable deaths for 1965. The second bar shows smoking-attributable deaths for 1985. The third bar shows the estimated 1985 smoking-attributable deaths if the U.S. populations at each age had remained at 1965 levels. The latter quantities were computed as aD^*, where a is the attributable risk given in Table 11 and D^* is a population-corrected estimate of 1985 U.S. deaths. The latter quantity was computed by multiplying 1985 age-specific death rates by the populations at risk in 1965.

Figures 10 and 11 show that population growth and aging cannot explain the changes in smoking-attributable mortality between 1965 and 1985. In particular, the marked increases in smoking-attributable deaths from lung cancer and COPD in women are systematic consequences of the American woman's adoption of lifelong cigarette smoking, from teenage years onward.

For men, population-corrected deaths due to smoking in 1985 were 165,000, compared with 150,000 in 1965. For women, population-corrected deaths due to smoking

TABLE 12.—Estimated deaths (in thousands) attributable to cigarette smoking, 10 selected causes, males and females, United States, 1985

Cause of death	Males	Females
CHD, age <65	34 (30–38)[a]	11 (9–12)
CHD, age ≥65	44 (36–54)	26 (20–34)
COPD	37 (35–39)	20 (18–21)
Cancer of lip, oral cavity, and pharynx	5.1 (4.4–5.4)	1.6 (1.2–2.0)
Cancer of larynx	2.3 (1.6–2.7)	0.6 (0.4–0.7)
Cancer of esophagus	5.0 (4.0–5.7)	1.6 (1.3–1.9)
Cancer of lung	76 (74–77)	30 (29–32)
Cancer of pancreas	3.3 (2.1–5.0)	3.4 (2.8–5.1)
Cancer of bladder	3.1 (2.1–4.2)	1.1 (0.6–1.9)
Cancer of kidney	2.6 (1.8–3.5)	0.4 (0.1–1.5)
Cerebrovascular disease, age <65	5.5 (3.9–7.0)	5.2 (4.3–6.2)
Cerebrovascular disease, age ≥65	12 (8–17)	4.8 (1.9–11.4)
Ten causes	231 (220–242)	106 (98–115)

NOTE: Computed from Table 11 and unpublished tabulations of deaths at ages 20 years or more by cause from NCHS, 1985. Sum of individual causes may not equal totals because of rounding.

[a]Numbers in parentheses are 95-percent confidence intervals.

in 1985 were 67,000, compared with 30,000 in 1965. Even if the population had remained entirely stable during 1965 through 1985, the lethality of cigarette use in American women would have doubled.

Among men, the total of 231,000 smoking-induced deaths in 1985 represented 41 percent of total deaths from the 10 causes combined and 22 percent of all deaths among persons aged 20 years or more. Among women, the total of 106,000 smoking-induced deaths represented 25 percent of deaths from the 10 causes combined and 11 percent of deaths from all deaths among persons aged 20 years or more.

The computations in Tables 10 and 12 have omitted other causes of death that are likely to be attributable to cigarette use. If the relative risks given in Tables 6 and 7 for

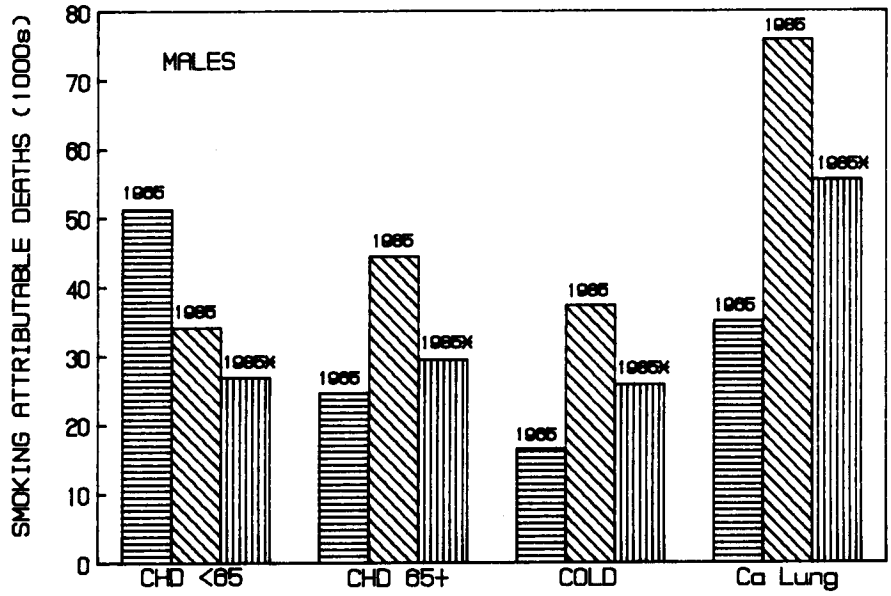

FIGURE 10.—Estimated cigarette-smoking-attributable deaths from CHD, COPD, and lung cancer, males aged 20 years or more, United States, 1965 and 1985

NOTE: For the bars marked 1985*, the estimated smoking-attributable deaths in 1985 have been corrected for population increases during 1965–85.

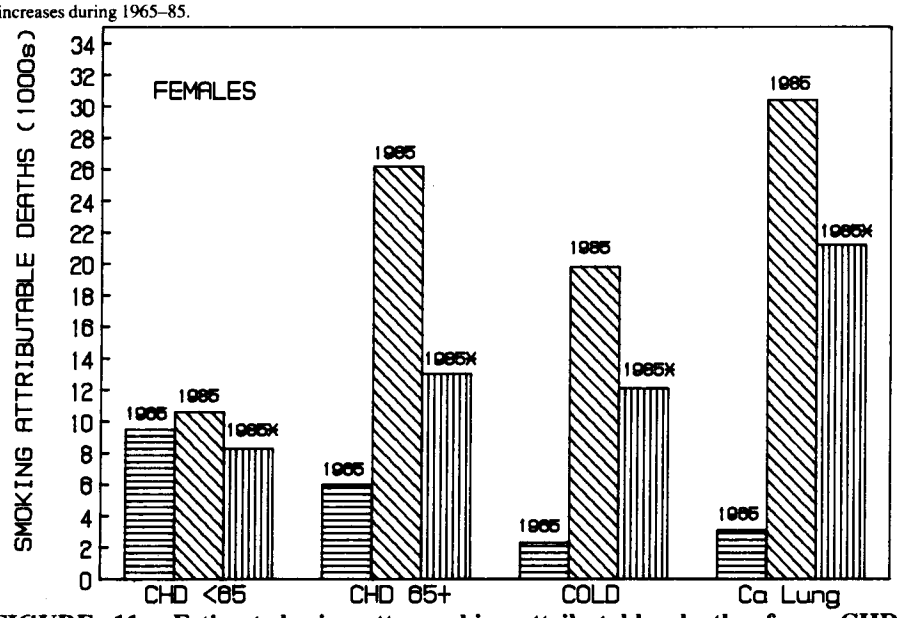

FIGURE 11.—Estimated cigarette-smoking-attributable deaths from CHD, COPD, and lung cancer, females aged 20 years or more, United States, 1965 and 1985

NOTE: For the bars marked 1985*, the estimated smoking-attributable deaths in 1985 have been corrected for population increases during 1965–85.

the broader categories of cardiovascular and nonneoplastic respiratory disease are applied to deaths from hypertensive heart disease, arteriosclerosis, aortic aneurysm, and influenza and pneumonia, then smoking-attributable deaths would increase to 256,000 among men and 126,000 among women. Inclusion of deaths among newborns and infants due to smoking during pregnancy would add an additional 2,500 to the total (CDC 1987b; McIntosh 1984; Kleinman et al. 1988); this does not include fetal loss due to smoking (Stein et al. 1981). Inclusion of lung cancer deaths among nonsmokers due to environmental tobacco smoke (NRC 1986) would add 3,800 and inclusion of deaths from cigarette-caused fires (Hall 1987) would add 1,700 to total attributable deaths. Inclusion of deaths due to cervical cancer caused by smoking would add 1,500. Including these additional causes of death, the smoking-attributable mortality in 1985 is then estimated to be approximately 390,000. Recent studies have also noted increased risks among smokers for hepatic cancer (Trichopoulos et al. 1987), penile cancer (Hellberg et al. 1987), leukemia (Kinlen and Rogot 1988), and anal cancer (Daling et al. 1987).

Among all persons at risk during 1985, an estimated 52 million were also cigarette smokers in 1965. The remaining 42 million were new cigarette smokers. In 1985, only about 4,400 deaths occurred among the latter group, which consists of persons in their teens, twenties, and thirties. Thus, 99 percent of deaths attributable to cigarette use in 1985 occurred among people who started smoking in 1965 or earlier. The vast majority of these people started smoking before the release of the 1964 Surgeon General's Report

TABLE 13.--Estimated risks of various activities

Activity or cause	Annual fatalities per 1 million exposed persons
Active smoking	7.000[a]
Alcohol	541
Accident	275
Disease	266
Motor vehicles	187
Alcohol-involved	95
Non-alcohol-involved	92
Work	113
Swimming	22
Passive smoking[b]	19
All other air pollutants[b]	6
Football	6
Electrocution	2
Lightning	0.5
DES in cattlefeed	0.3
Bee sting	0.2
Basketball	0.02

NOTE: Activities are not mutually exclusive; there are overlaps between categories. Differences in fatalities do not imply proportionate differences in years of life lost.

[a]Number of deaths per million smokers who began smoking before 1965.

[b]Cancer deaths only.

SOURCE: Active smoking, CPS-II; NHISs 1965, 1985; U.S. Bureau of the Census (1974, 1986). Other activities or causes, U.S. President (1987).

and before the 1965 Federal Cigarette Labeling and Advertising Act. For this group, the annual smoking-attributable fatality rate is about 7 deaths per 1,000 at risk, or about 7,000 deaths per 1 million persons, As shown in the Economic Report of the President (U.S. President 1987) this rate far exceeds the rates for other risks of death (Table 13).

Conclusions

1. Lung cancer death rates increased two- to fourfold among older male smokers over the two decades between the American Cancer Society's two Cancer Prevention Studies (CPS-I, 1959-65, and CPS-II, 1982-86). Lung cancer death rates for younger male smokers fell about 30 to 40 percent during this period.
2. Lung cancer death rates increased four- to sevenfold among female smokers aged 45 years or older in CPS-II compared with CPS-I, while lung cancer death rates among younger women declined 35 to 55 percent.
3. The two-decade interval witnessed a two- to threefold increase in death rates from chronic obstructive pulmonary disease (COPD) in female smokers aged 55 years or older.
4. There was no change in the age-adjusted death rates for lung cancer and COPD between CPS-I and CPS-II among men and women who never smoked regularly.
5. Overall death rates from coronary heart disease (CHD) declined substantially between CPS-I and CPS-II. The decline in CHD mortality among nonsmokers, however, was notably greater than among current cigarette smokers.
6. In CPS-II, the relative risks of death from cerebrovascular lesions were 3.7 and 4.8 for men and women smokers under age 65. Increased risks of stroke were also observed among older smokers and former smokers. Along with the recently reported results of other studies, these findings strongly support a causal role for cigarette smoking in thromboembolic and hemorrhagic stroke.
7. In 1985, smoking accounted for 87 percent of lung cancer deaths, 82 percent of COPD deaths, 21 percent of CHD deaths, and 18 percent of stroke deaths. Among men and women less than 65 years of age, smoking accounted for more than 40 percent of CHD deaths.
8. The large increase in smoking-attributable mortality among American women between 1965 and 1985 was a direct consequence of their adoption of lifelong cigarette smoking, especially from their teenage years onward.
9. In 1985, 99 percent of smoking-attributable deaths occurred among people who started smoking before the 1964 Surgeon General's Report. For this group, the annual smoking-attributable fatality rate is about 7,000 deaths per 1 million persons at risk.
10. For 10 causes of death, a total of 337,000 deaths were attributable to smoking in 1985. These represented 22 percent of all deaths among men and 11 percent among women. If other cardiovascular, neoplastic, and respiratory causes of death were included-as well as deaths among newborns and infants resulting from maternal smoking, deaths from cigarette-caused residential fires, and lung cancer deaths among nonsmokers due to environmental tobacco smoke--the total smoking--attributable mortality was about 390,000 in 1985.

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