Lung Association, have played a significant role in educating the public about the hazards of tobacco use.

2. Individual and group smoking cessation programs evolved from an emphasis on conditioning-based approaches in the 1960s, to the cognitively based self-management procedures of the 1970s, to the relapse prevention and pharmacologically based components of the 1980s.

3. There has recently been an increased emphasis on targeting specific groups of smokers for cessation activities (e.g., pregnant women, Hispanics, blacks).

4. Packaging and marketing of self-help smoking cessation materials have become more sophisticated and there is more of an emphasis on relapse prevention, while much of the content has changed relatively little over the years.

5. Mass-mediated quit-smoking programs have become an increasingly popular strategy for influencing the smoking behavior of a large number of smokers.

6. The 1980s have seen an increase in the promotion of smoking control efforts in the workplace in response to increasing demand and opportunity for worksite wellness programs and smoking control policies.

7. In the last decade there has been an increasing interest in involving physicians and other health care professionals in smoking control efforts. Medical organizations have played a more prominent role in smoking and health during the 1980s than they had in the past.

Part III. Antismoking Advocacy and Lobbying

1. Lobbying and advocacy efforts have expanded through the increasing commitment of the national voluntary health agencies to political action and the formation of coalitions at the local, State, and national levels.

2. Antismoking advocacy and lobbying have evolved over the past 25 years and now focus on a growing number of local, State, and national legislative and regulatory initiatives designed to reduce smoking, regulate the cigarette product, and prevent the uptake of smoking by children and adolescents.

Chapter 7: Smoking Control Policies

Part I. Policies Pertaining to Information and Education

1. The Federal Government's efforts to reduce the health consequences of cigarette smoking have consisted primarily of providing the public with information and education about the hazards of tobacco use. Two of the most well-known mechanisms are the publication of Surgeon General's Reports and the requirement of warning labels on cigarette packages. A system of rotating health warning labels is now required for all cigarette and smokeless tobacco packaging and advertisements.

2. Current laws do not require health warning labels on all tobacco products and do not require monitoring of the communications effectiveness of the warnings. Furthermore, existing laws do not provide administrative mechanisms to update the
contents of labels to prevent the overexposure of current messages or to reflect advances in scientific knowledge, such as new information about the addictive nature of tobacco use.

3. There is insufficient evidence to determine the independent effect of cigarette warning labels, particularly the rotating warning labels required since 1985, on public knowledge about the health effects of smoking or on smoking behavior.

4. Information about tar and nicotine yields appears on all cigarette advertisements but not on all cigarette packages. Levels of other hazardous constituents of tobacco smoke, such as carbon monoxide, hydrogen cyanide, and ammonia, are not disclosed on packages or advertisements. Little information is available to the public about the identity or health consequences of the additives in tobacco products.

5. Declines in adult per capita cigarette consumption have occurred in years of major dissemination of information on the health hazards of smoking. These include 1964, the year of the first Surgeon General’s Report on smoking and health, and 1967–70, when antismoking public service announcements were widely broadcast on radio and television, as mandated by the Federal Communications Commission’s Fairness Doctrine.

6. In 1985, when cigarette advertising and promotion totaled 2.5 billion dollars, cigarettes were the most heavily advertised product category in the outdoor media (e.g., billboards), second in magazines, and third in newspapers. Over the past decade, the majority of cigarette marketing expenditures has shifted from traditional print advertising to promotional activities (e.g., free samples, coupons, sponsorship of sporting events).

7. An estimated 1 percent of the budget allocated to disease prevention by the U.S. Department of Health and Human Services is devoted specifically to tobacco control. These expenditures totaled 39.5 million dollars in 1986.

Part II. Economic Incentives

1. Cigarette excise taxes are imposed by the Federal Government (16 cents per pack), all State governments, and nearly 400 cities and counties. On average, Federal and State excise taxes add 34 cents per pack to the price of cigarettes. Cigarette excise tax rates have fallen since 1964 in real terms because the rate and magnitude of periodic tax increases have not kept pace with inflation.

2. Studies demonstrate that increases in the price of cigarettes decrease smoking, particularly by adolescents. It has been estimated that an additional 100,000 or more persons will live to age 65 as a result of the price increases induced by the 1983 doubling of the Federal excise tax on cigarettes.

3. In 1964, smoking status was not considered in the determination of insurance premiums. Currently, nearly all life insurers but only a few health, disability, and property and casualty insurers offer premium discounts for nonsmokers. Few health insurers reimburse for the costs of smoking cessation programs or treatment.
Part III. Direct Restrictions on Smoking

1. Restrictions on smoking in public places and at work are growing in number and comprehensiveness, as a result of both Government actions and private initiatives. Forty-two States and more than 320 communities have passed laws restricting smoking in public, and an estimated one-half of large businesses have a smoking policy for their employees.

2. The goal of these smoking restrictions is to protect individuals from the consequences of involuntary tobacco smoke exposure, but they may also contribute to reductions in smoking prevalence by changing the attitudes and behavior of current and potential smokers. Insufficient research has been undertaken to determine the extent, if any, of these effects.

3. There are fewer legal restrictions on children's access to tobacco products now than in 1964, despite what has been learned since then about the dangers of tobacco use, its addictive nature, and the early age of initiation of smoking.

4. As of January 1, 1988, laws in 43 States and the District of Columbia restricted the sale of cigarettes to minors. Nevertheless, tobacco products are relatively easy for children to obtain through vending machines and over-the-counter purchases because of low levels of compliance with and enforcement of current laws.

5. Tobacco products have been exempted by law or administrative decision from the jurisdiction of Federal regulatory agencies under whose authority they might otherwise fall.

Chapter 8: Changes in the Smoking-and-Health Environment: Behavioral and Health Consequences

1. All birth cohorts born between 1901 and 1960 experienced reductions in the prevalence of smoking relative to the rates that would have been expected in the absence of the antismoking campaign. By 1985, the gap between actual (reported) prevalence and that which would have been expected ranged from 6 percentage points for the eldest female cohort to 28 percentage points for the youngest male cohort.

2. In 1985, an estimated 56 million Americans 15 to 84 years of age were smokers. In the absence of the antismoking campaign, an estimated 91 million would have been smokers.

3. Adult per capita cigarette consumption has fallen 3 to 8 percent in years of major smoking-and-health events, such as publication of the first Surgeon General's Report on smoking and health in 1964. Per capita consumption fell each of the years the Fairness Doctrine antismoking messages were presented on television and radio (1967-70).

4. By 1987, adult per capita cigarette consumption would have exceeded its actual level by an estimated 79 to 89 percent had the antismoking campaign never occurred.

5. One of the most substantial behavioral responses to concerns about smoking and health has been the shift toward filtered cigarettes in the 1950s and low-tar and
low-nicotine cigarettes in the 1970s. The net health impact of these product changes is unknown.

6. As a result of the antismoking campaign, an estimated 789,000 deaths were postponed during the period 1964 through 1985, 112,000 in 1985 alone. The average life expectancy gained per postponed death was 21 years.

7. The avoidance of smoking-related mortality associated with the antismoking campaign will represent a growing percentage of smoking-related mortality over time, as the principal beneficiaries of the campaign, younger men and women, reach the ages at which smoking-related disease is most common. Campaign-induced quitting and noninitiation through 1985 will result in the postponement or avoidance of an estimated 2.1 million smoking-related deaths between 1986 and the year 2000.
References


CHAPTER 2

ADVANCES IN KNOWLEDGE OF THE HEALTH CONSEQUENCES OF SMOKING
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INTRODUCTION

The purpose of this Chapter is to summarize and compare the state of biomedical knowledge concerning tobacco and health in 1989 with that presented in the 1964 Surgeon General's Report (see Table 13). The Chapter addresses major tobacco-related disorders that are well documented in the medical literature; it does not consider many areas of current research that may prove to be important but are in an early or provisional state of investigation.

The 1964 Surgeon General's Report was a landmark publication that included a survey of more than 7,000 available scientific articles on smoking and health. The Advisory Committee that prepared the 1964 Report reviewed and assessed epidemiologic, clinical, pathological, and experimental data for evidence linking smoking to disease. To reach conclusions concerning the causality of associations between smoking and disease, the Committee constructed a framework for evaluating the evidence. With regard to causality, the Committee concluded:

The causal significance of an association is a matter of judgment which goes beyond any statement of statistical probability. To judge or evaluate the causal significance of the association between attribute or agent and the disease, or effect upon health, a number of criteria must be utilized, no one of which is an all-sufficient basis for judgment. These criteria include:

a) the consistency of the association
b) the strength of the association
c) the specificity of the association
d) the temporal relationships of the association
e) the coherence of the association (US PHS 1964).

These criteria were applied throughout the 1964 Report. When the word "cause" was used in the 1964 Report, it was felt to convey "the notion of a significant, effectual relationship between an agent and an associated disorder or disease in the host." Use of the word "cause" in relation to cigarette smoking did not exclude other agents as causes; rather, the members of the Advisory Committee shared "a common conception of the multiple etiology of biological processes."

The principal findings on the health effects of smoking were summarized in the Surgeon General’s 1964 Report as follows:
1. Cigarette smoking is associated with a 70-percent increase in the age-specific death rates of men.
2. Cigarette smoking is causally related to lung cancer in men; the magnitude of the effect of cigarette smoking far outweighs all other factors. The data for women, though less extensive, point in the same direction.
3. Cigarette smoking is the most important of the causes of chronic bronchitis in the United States and increases the risk of dying from chronic bronchitis and
emphysema. A relationship exists between cigarette smoking and emphysema, but it has not been established that the relationship is causal.

4. It is established that male cigarette smokers have a higher death rate from coronary artery disease than nonsmoking males. Although the causative role of cigarette smoking in deaths from coronary disease is not proven, the Committee considers it more prudent from the public health viewpoint to assume that the established association has causative meaning than to suspend judgment until no uncertainty remains.

5. Pipe smoking appears to be causally related to lip cancer. Cigarette smoking is a significant factor in the causation of cancer of the larynx in men. The evidence supports the belief that an association exists between tobacco use and cancer of the esophagus, and between cigarette smoking and cancer of the urinary bladder in men. But the data are not adequate to decide whether these relationships are causal.

6. Women who smoke cigarettes during pregnancy tend to have babies of lower birthweight. It is not known whether this decrease in birthweight has any influence on the biological fitness of the newborn.

7. Epidemiologic studies indicate an association between cigarette smoking and peptic ulcer that is greater for gastric than for duodenal ulcer.

8. The habitual use of tobacco is related primarily to psychological and social drives, reinforced and perpetuated by the pharmacologic actions of nicotine.

Since 1967, the U.S. Department of Health and Human Services has transmitted to the U.S. Congress mandated reports on the health consequences of smoking. Some of the reports have been encyclopedic reviews similar to the 1964 Report, whereas others have focused on the relationship between smoking and a specific topic. The Federal unit charged with preparing these annual reports, the Office on Smoking and Health, now has more than 57,000 documents on smoking and health in its Technical Information Center database.

Research performed during the subsequent 25 years has substantiated and strengthened the conclusions of the 1964 Advisory Committee. Studies published since 1964 have also established associations between smoking and disease in areas for which data did not exist in 1964, shed light on pathogenetic mechanisms of tobacco-related disease, and added scientific depth to areas mentioned only briefly in the 1964 Report.

**PART I: HEALTH CONSEQUENCES**

**Smoking and Overall Mortality** [See Chapter 3 for more detailed discussion]

The major prospective studies of the disease risks associated with smoking completed in the 1960s and 1970s contributed substantially to an understanding of the relationship between smoking and disease (US DHEW 1979). These studies provided estimates of both the relative and attributable risks related to cigarette and other types of smoking (Table 1) (US DHEW 1979). Male cigarette smokers had approximately 70 percent higher overall death rates than nonsmokers; the excess mortality of female.
### TABLE 1.—Mortality ratios of current cigarette-only smokers, by cause of death in eight prospective epidemiologic studies

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>British doctors</th>
<th>Males in 25 States</th>
<th>U.S. veterans</th>
<th>Japanese study</th>
<th>Canadian veterans</th>
<th>Males in 9 States</th>
<th>Swedish</th>
<th>California occupations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer of lung and bronchus (162–163)</td>
<td>14.0</td>
<td>7.84</td>
<td>11.50</td>
<td>17.14</td>
<td>3.64</td>
<td>14.7</td>
<td>10.73</td>
<td>7.0</td>
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<td>Cancer of larynx (161)</td>
<td>6.09</td>
<td>8.99</td>
<td>9.96</td>
<td>13.59</td>
<td>13.10</td>
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<tr>
<td>Cancer of buccal cavity (140–141)</td>
<td>13.0b</td>
<td></td>
<td>4.09</td>
<td>7.04</td>
<td>3.9b</td>
<td>2.80</td>
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<td>Cancer of pharynx (145–148)</td>
<td>9.90f</td>
<td>2.93f</td>
<td>12.54</td>
<td>2.81</td>
<td></td>
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<tr>
<td>Cancer of esophagus (150)</td>
<td>4.7</td>
<td>4.17</td>
<td>1.74</td>
<td>6.17</td>
<td>2.57</td>
<td>3.3</td>
<td>6.60</td>
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<td>Cancer of bladder and other (181)</td>
<td>2.1</td>
<td>2.20</td>
<td>2.96</td>
<td>2.15</td>
<td>0.98</td>
<td>1.3</td>
<td>2.40</td>
<td>1.8</td>
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<td>Cancer of pancreas (157)</td>
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<td>2.69</td>
<td>2.17</td>
<td>1.84</td>
<td>1.83</td>
<td>2.1</td>
<td>3.1</td>
<td>2.5</td>
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<td>Cancer of kidney (180)</td>
<td>1.47</td>
<td>1.57</td>
<td>1.45</td>
<td>1.11</td>
<td>1.4</td>
<td>1.50</td>
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<td>Cancer of stomach (151)</td>
<td>1.42</td>
<td>1.26</td>
<td>1.60</td>
<td>1.51</td>
<td>1.9</td>
<td>2.30</td>
<td>0.9</td>
<td>2.3</td>
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<td>Cancer of intestines (152–153)</td>
<td>1.27</td>
<td>1.27</td>
<td>1.4</td>
<td>0.50</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Cancer of rectum (154)</td>
<td>2.7</td>
<td>1.01e</td>
<td>1.17f</td>
<td>0.98</td>
<td>0.91</td>
<td>0.6</td>
<td>0.80</td>
<td>0.9</td>
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<tr>
<td><strong>All cardiovascular disease</strong> (330–334, 400–468)</td>
<td>1.90</td>
<td>1.31</td>
<td>1.75</td>
<td></td>
<td></td>
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<tr>
<td>CHD (420)</td>
<td>1.6</td>
<td>2.08</td>
<td>1.36</td>
<td>1.74</td>
<td>1.96</td>
<td>1.6</td>
<td>1.70</td>
<td>1.7</td>
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<td>Cerebrovascular lesions (340–344)</td>
<td>1.3</td>
<td>1.38</td>
<td>1.06</td>
<td>1.52</td>
<td>1.14</td>
<td>0.9</td>
<td>1.30</td>
<td>1.0</td>
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<td>Aortic aneurysm (nonsyphilitic) (451)</td>
<td>6.6</td>
<td>2.62</td>
<td>4.92</td>
<td>5.24</td>
<td>1.8</td>
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<td>Hypertension (440–447)</td>
<td>1.40</td>
<td>1.42</td>
<td>1.67</td>
<td>2.51</td>
<td>1.6</td>
<td>1.20</td>
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<td>1.86</td>
<td>3.3</td>
<td>2.00</td>
<td>2.0</td>
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<td>Cause of death</td>
<td>British doctors&lt;sup&gt;1&lt;/sup&gt;</td>
<td>Males in 25 States&lt;sup&gt;2&lt;/sup&gt;</td>
<td>Males in 9 States&lt;sup&gt;6&lt;/sup&gt;</td>
<td>Swedish&lt;sup&gt;3&lt;/sup&gt;</td>
<td>California occupations&lt;sup&gt;5&lt;/sup&gt;</td>
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<tr>
<td>All respiratory disease (nonneoplastic)</td>
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<tr>
<td>Emphysema and/or bronchitis</td>
<td>24.7</td>
<td>10.08</td>
<td>7.7</td>
<td>2.85</td>
<td>2.9&lt;sup&gt;4&lt;/sup&gt;</td>
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<td>Emphysema without bronchitis (527.1)</td>
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<td>6.55</td>
<td>11.41</td>
<td>14.17</td>
<td>11.3</td>
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<tr>
<td>Bronchitis (500-502)</td>
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<td>4.99</td>
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<tr>
<td>Respiratory tuberculosis (001-008)</td>
<td>5.0</td>
<td>2.12</td>
<td></td>
<td>1.27</td>
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<td>Asthma (241)</td>
<td></td>
<td>3.47</td>
<td></td>
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<tr>
<td>Influenza and pneumonia (480-498)</td>
<td>1.4</td>
<td>1.86</td>
<td>1.72</td>
<td>1.87</td>
<td>1.4</td>
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<tr>
<td>Certain other conditions</td>
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<td></td>
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<tr>
<td>Stomach ulcer (540)</td>
<td>2.5&lt;sup&gt;c&lt;/sup&gt;</td>
<td>4.06</td>
<td>4.13</td>
<td>4.13</td>
<td>2.06&lt;sup&gt;c&lt;/sup&gt;</td>
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<td>Duodenal ulcer (541)</td>
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<td>2.86</td>
<td>1.50</td>
<td>2.98</td>
<td>6.9</td>
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<tr>
<td>Cirrhosis (581)</td>
<td>3.0</td>
<td>2.06</td>
<td>1.97</td>
<td>3.38</td>
<td>1.35</td>
<td></td>
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<tr>
<td>Parkinsonism (350)</td>
<td>0.4</td>
<td>0.26</td>
<td></td>
<td></td>
<td>1.17</td>
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<td></td>
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<tr>
<td>All causes</td>
<td>1.04</td>
<td>1.88</td>
<td>1.45</td>
<td>1.84</td>
<td>1.22</td>
<td></td>
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</tr>
</tbody>
</table>

<sup>1</sup>Numbers in parentheses represent International Classification of Diseases (ICD) codes.
<sup>2</sup>Includes cancers of larynx, buccal cavity, and pharynx.
<sup>3</sup>Includes cancers of larynx, buccal cavity and pharynx.
<sup>4</sup>Includes cancers of larynx, buccal cavity and pharynx.
<sup>5</sup>Includes cancers of larynx, buccal cavity, and pharynx.
<sup>6</sup>Includes cancers of larynx, buccal cavity, and pharynx.

SOURCE: Studies cited are as follows: "Doll and Hill (1956); "Hammond (1966); "Kahn (1966); "Hirayama (1967); "Best, Josie, Walker (1961); "Hammond and Horn (1958); "Cederlof et al. (1975); "Dunn, Linden, Breslow (1960). US DHEW (1979)."
cigarette smokers was somewhat less than that of men, but it increased over the follow-up intervals. A strong dose-response relationship was found between exposure to cigarette smoke and excess mortality; cessation of cigarette smoking was associated with a decrease in this excess mortality. The relative risks were greater for smoking-related cancers and chronic obstructive pulmonary disease (COPD) than for coronary heart disease (CHD); however, because of the higher mortality rates for CHD the smoking-attributable mortality associated with CHD accounted for over one-third of the excess mortality due to smoking-related diseases.

There have been relatively few long-term longitudinal studies that have measured the overall effects of cigarette smoking since these earlier reports. Results from a new American Cancer Society (ACS) prospective study (Cancer Prevention Study II, CPS-II) and a detailed discussion of total smoking-related mortality are presented in Chapter 3. Based on this study, cigarette smoking is currently estimated to account for 21 percent of all CHD deaths, 30 percent of all cancer deaths, and 82 percent of all COPD deaths.

The Multiple Risk Factor Intervention Trial (MRFIT) is a recent prospective study that screened 361,662 men aged 35 to 57 years between 1972 and 1974 and has been following them since then, both through the Social Security Administration and the National Death Index files. To gauge smoking status, only the number of cigarettes smoked per day at enrollment was reported. Because former smokers were included in the nonsmoker category, the risk comparisons in this study between nonsmokers and smokers are conservative in estimating the effects of smoking. Findings for the 6 years of follow-up for the MRFIT enrollees screened from 1972–73 are consistent with the studies reported in the 1960s, despite changes in the type of cigarettes in terms of tar and nicotine yield and the increased use of filters (see later section of this Chapter and Chapter 5). The MRFIT study shows that smoking status and number of cigarettes smoked per day have remained powerful predictors for total mortality and the development of CHD, stroke, cancer, and COPD. In the study population, there were an estimated 2,249 (29 percent) excess deaths due to smoking, of which 35 percent were from CHD and 21 percent from lung cancer. The nonsmoker–former smoker group had 30 percent fewer total cancers than the smoking group over the 6-year follow-up.

A study of a random sample of 25,129 Swedish men between 1964 and 1979 evaluated the relationship between cigarette smoking (prevalence of 32 percent), pipe smoking (27 percent), cigar smoking (5 percent), and subsequent mortality (Table 2; Carstensen, Pershagen, Eklund 1987). The all-cause relative death rate was 1.7-fold higher for those smoking greater than 15 g of tobacco per day (estimated as 16 to 25 cigarettes equaling 20 g or a package of pipe tobacco lasting 1 to 4 days equaling 16 g). The relative risks associated with cigarette smoking were consistent both with those of the current MRFIT sample and the earlier cohorts from the 1950s and 1960s. The risks were also increased for pipe and cigar smokers for many of the causes of death.

Epidemiologic studies have shown that cigarette smoking exerts an adverse effect on mortality in older as well as younger age groups. The 17-year follow-up of the Alameda County Study (Kaplan et al. 1987) demonstrates an increased risk of death even among older cigarette smokers. The adjusted relative risk of death among smokers at entry was 1.46 (age 60 to 69) and 1.43 at age 70 or more. Smoking remained the strongest
predictor of mortality even in this older age group. Other studies have also substantiated that smoking remains an important risk factor in the older age groups (Jajich, Ostfeld, Freeman 1984).

**TABLE 2.—Mortality ratios for selected causes in Swedish males, 1964–1979, by type of smoking**

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>Cigarettes only</th>
<th>Pipe only</th>
<th>Cigars only</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer of oral cavity and larynx (140–146, 148, 161)</td>
<td>2.9 (8)</td>
<td>1.4 (3)</td>
<td>0.6 (1)</td>
</tr>
<tr>
<td>Cancer of esophagus (150)</td>
<td>3.7 (9)</td>
<td>3.6 (6)</td>
<td>6.5 (2)</td>
</tr>
<tr>
<td>Cancer of liver and biliary passages (155–156)</td>
<td>3.0 (13)</td>
<td>1.7 (5)</td>
<td>7.2 (4)</td>
</tr>
<tr>
<td>Cancer of pancreas (157)</td>
<td>3.3 (28)</td>
<td>2.8 (19)</td>
<td>1.0 (1)</td>
</tr>
<tr>
<td>Cancer of trachea, bronchus, and lung (162)</td>
<td>7.4 (77)</td>
<td>7.2 (59)</td>
<td>7.6 (11)</td>
</tr>
<tr>
<td>Cancer of bladder (188)</td>
<td>4.2 (17)</td>
<td>4.0 (16)</td>
<td>1.9 (1)</td>
</tr>
<tr>
<td>Ischemic heart disease (410–414)</td>
<td>1.48 (399)</td>
<td>1.39 (366)</td>
<td>1.16 (42)</td>
</tr>
<tr>
<td>Aortic aneurysm (nonsyphilitic) (441)</td>
<td>2.1 (11)</td>
<td>2.1 (11)</td>
<td>5.1 (4)</td>
</tr>
<tr>
<td>Bronchitis and emphysema (490–492)</td>
<td>3.3 (18)</td>
<td>3.6 (16)</td>
<td>1.3 (1)</td>
</tr>
<tr>
<td>Peptic ulcer (531–534)</td>
<td>2.0 (11)</td>
<td>2.8 (13)</td>
<td>4.0 (3)</td>
</tr>
<tr>
<td>Cirrhosis of liver (571)</td>
<td>1.8 (21)</td>
<td>0.7 (4)</td>
<td>2.7 (3)</td>
</tr>
<tr>
<td>Suicide, accidents, and violence (E800–E999)</td>
<td>1.7 (90)</td>
<td>0.9 (35)</td>
<td>2.5 (10)</td>
</tr>
<tr>
<td>All causes</td>
<td>1.45 (1,063)</td>
<td>1.29 (866)</td>
<td>1.39 (131)</td>
</tr>
</tbody>
</table>

NOTE: Death rates standardized for age and residence. Never smokers constitute the reference group. Number of deaths are given in parentheses.

*The mean grams of tobacco smoked per day in 1963, standardized for age and residence, was estimated to be 10.7 in cigarette smokers, 8.4 in pipe smokers, and 13.5 in cigar smokers.

Numbers in parentheses are ICD-8 codes.

Lung Cancer

Introduction

One of the most prominent conclusions of the 1964 Report was the determination that “Cigarette smoking is causally related to lung cancer in men; the magnitude of the effect far outweighs all other factors. The data for women, though less extensive, point in the same direction.” The epidemiologic evidence available in 1964 on smoking and lung cancer was already extensive. Sharply increasing lung cancer mortality rates in the United States across the 20th century provided indisputable documentation of a new epidemic. Clinical observations and early epidemiologic findings suggested that tobacco smoking was associated with lung cancer, but hypotheses related to air pollution, occupation, and other factors were also extant. By 1964, however, the epidemiologic data, derived from 29 retrospective and 7 prospective studies, were conclusive: smoking was causally related to cancer of the lung. Further support for this conclusion was obtained from animal studies showing that condensates of tobacco smoke were carcinogenic and from the demonstration that tobacco smoke contained carcinogens (US DHHS 1982). The evidence compiled through 1964 also provided additional insight into quantitative aspects of respiratory carcinogenesis by tobacco smoke. The risk of lung cancer was shown to increase with the amount and duration of smoking and to decline with cessation of smoking.

In the 25 years since the 1964 Report, voluminous evidence has continued to support the causal relationship between smoking and lung cancer. The new evidence has been sufficient to establish that smoking also causes lung cancer in women: more comprehensive epidemiologic data have provided expanded descriptions of dose-response relationships between smoking and lung cancer risk. Research has also been directed at environmental and host factors determining susceptibility to tobacco smoke. New investigative techniques in molecular and cellular biology are now providing insight into the molecular mechanisms of carcinogenesis by tobacco smoke.

Dose-Response Relationships

The 1964 Report reviewed evidence from retrospective and prospective epidemiologic investigations that documented dose-response relationships between lung cancer risk and measures of exposure to tobacco smoke. This evidence was cited by the 1964 Report in relation to the criterion of strength of association for determining causality. Investigation of dose-response relationships for lung cancer has subsequently been extended. Mathematical models have been applied to the epidemiologic data to gain biological insight into respiratory carcinogenesis. The cigarette has evolved substantially since 1964 with modifications designed to reduce tar and nicotine yields. Recent research has addressed the risks of smoking the newer products. Studies of lung cancer and involuntary smoking have examined lung cancer risks at low dose levels (US DHHS 1986a).

Abundant epidemiologic evidence has shown dose-response relationships of lung cancer risk with cigarettes smoked per day, degree of inhalation, and age at initiation.
of regular smoking. For the purpose of illustration, selected examples of dose–response relationships from two of the early, large prospective epidemiologic studies are reviewed here. Figure 1 shows lung cancer mortality ratios for males by the number of cigarettes smoked per day. For those who smoked more than 40 cigarettes per day, the risk of dying of lung cancer was 23 times greater than the risk experienced by non-smokers.

Figure 2 illustrates the lung cancer mortality ratios for males by self-reported degree of inhalation of cigarette smoke. These data confirm that even those who reported "just puffing" on cigarettes still had a significantly increased risk of lung cancer. Those who reported inhaling "none" or "slightly" experienced a risk of developing lung cancer that was eight times greater than that of non-smokers. The relative risk increased to 17 for those who inhaled deeply.

Figure 3 shows lung cancer mortality ratios for males by the age they began smoking. The risk of developing lung cancer was greatest for those who began smoking at an early age.

Mathematical modeling of dose–response relationships, in the biological framework of a multistage model of carcinogenesis, has provided further insight into the nature of dose–response relationships for smoking and lung cancer. Using data from the prospective study of British doctors, Doll and Peto (1978) have performed the most widely cited analysis. They compared regular smokers and lifelong non-smokers and showed that lung cancer incidence increased with the square of the amount smoked daily, but with the duration of smoking raised to a power of 4 to 5. This finding implies that duration of smoking is the stronger determinant of lung cancer risk and that initiation of smoking during the teenage years will have serious consequences for lung cancer risk (Peto 1986).

Commercial cigarettes have continuously evolved through the addition of filters and other modifications designed to reduce tar and nicotine yields (US DHHS 1981). Since extensive modification of the cigarette began in the 1950s, it has only recently become possible to investigate smokers with predominant use of the newer products. Evidence from prospective and case-control studies and assessment of temporal trends of lung cancer mortality indicate somewhat lower risks for cigarettes with reduced tar and nicotine yield, although the risks remain markedly higher than for non-smokers (US DHHS 1982).

Doll and Peto (1981) examined trends of lung cancer mortality in males in the United States, Britain, and other European countries. They concluded that the international differences and the temporal trends were generally consistent with the tar yields and tar intakes across time and across countries.

Relevant information is also available from case-control and prospective studies. In the United States, investigations spanning the 1960s and 1970s have shown somewhat reduced lung cancer risks in smokers who switched from nonfilter to filter cigarettes (Bross and Gibson 1968; Wynder, Mabuchi, Beattie 1970; Hammond et al. 1976; Wynder and Stellman 1979). More recent studies continue to document lower risks in smokers of filter cigarettes compared with smokers of nonfilter cigarettes. In a case-control study conducted in Western Europe, the relative risk for lifelong nonfilter cigarette smokers was approximately twice that for smokers of filter cigarettes alone.
3. Mortality Ratio

FIGURE 1.—Lung cancer mortality ratio for males by cigarettes smoked per day

FIGURE 2.—Lung cancer mortality ratio for males by degree of inhalation
SOURCE: CPS-I (Hammond 1956).

FIGURE 3.—Lung cancer mortality ratio for males by age began smoking
However, dose–response relationships could not be demonstrated between relative risk and the proportion of years nonfilter brands were smoked or with a cigarette tar index. Among sustained smokers, switching from nonfilter to filter cigarettes was associated with a small reduction in risk (Lubin et al. 1984a). The results from another recent case–control study conducted in Cuba also did not show a convincing association between tar intake and relative risk of lung cancer (Joly, Lubin, Caraballos 1983). In New Mexico, a case–control study found that lifelong filter cigarette smokers and smokers of both filter and nonfilter cigarettes were at lower risk than lifelong smokers of nonfilter cigarettes only (Pathak et al. 1986). However, there was no evidence of decreasing risk as the extent of filter smoking increased. In addition, few data are available on the reduced risk of smoking low-tar or filter cigarettes for any other smoking-related disease (see Chapter 3).

Women and Lung Cancer

In 1964, at the time of the first Surgeon General’s Report, lung cancer was the leading cause of cancer mortality in males, but was only the fifth leading cause of cancer mortality among women. In 1964, the male–female ratio of death rates from lung cancer was 6.7. The 1964 Report did not determine that smoking was causally related to lung cancer in women, although the suggestive nature of the evidence was cited in the Report’s conclusion on lung cancer. The consistency of the male–female differences in lung cancer mortality with temporal trends of smoking was noted.

In the 25 years that have elapsed since the 1964 Report, lung cancer mortality has increased dramatically in women. In 1986, lung cancer and breast cancer were the leading causes of cancer death in U.S. women, accounting for approximately equal numbers of cancer deaths (Figure 4); lung cancer deaths are now projected to have surpassed breast cancer deaths (American Cancer Society 1988). Lung cancer mortality for women now equals that observed for men three decades earlier and the male–female ratio of death rates has now fallen to 2.0.

Since the late 1970s, the rise in the age-adjusted death rates of lung cancer among men began to level off (Horm and Kessler 1986). In contrast, lung cancer death rates among women continue to climb (Figure 4). As Figures 4 and 5 demonstrate, lung cancer is the only major cancer whose death rates have increased substantially and steadily since the 1930s. The dramatic increase among women began approximately 30 years after the increase for men, consistent with the later adoption of smoking by women; the slope of the curve for women appears to be nearly identical to that of men 30 years earlier. Figure 4 also demonstrates that among women, the lung cancer death rate closely approximated the breast cancer death rate in the mid-1980s. Illustrative of the importance of lung cancer in overall cancer mortality is the fact that, excluding lung cancer, the Nation’s age-adjusted cancer death rate fell by 13 percent from 1950 through 1982. Including lung cancer, the rate increased by 8 percent (Bailar and Smith 1986).

The mounting evidence on smoking and lung cancer in women led to a strengthening of the tentative conclusion in the 1964 Report. The 1971 Report concluded that “Cigarette smoking is a cause of lung cancer in women but accounts for a smaller
Age-Adjusted Cancer Death Rates* for Selected Sites, Females, United States, 1930-1986

FIGURE 4.—Age-adjusted cancer death rates* for selected sites, females, United States, 1930-86

*Adjusted to the age distribution of the 1970 U.S. Census population.

SOURCES OF DATA: National Center for Health Statistics; U.S. Bureau of the Census.

proportion of cases than in men” (US DHEW 1971). The conclusion of the 1979 Report was similar (US DHEW 1979). The 1980 Report (US DHHS 1980), concerned with smoking and women, and the 1982 Report (US DHHS 1982), concerned with smoking and cancer, comprehensively reviewed the epidemiologic data and reaffirmed the earlier conclusions concerning the causal association of smoking and lung cancer in
women; the evidence also provided comprehensive descriptions of dose–response relationships with findings similar to those reported previously for men. Recently reported dose–response relationships from the American Cancer Society Cancer Prevention Study II for lung cancer and women extend these observations (Figure 6).
These data also dramatically illustrate that the current lung cancer epidemic in women is confined to those who smoke cigarettes (Figure 7).

**FIGURE 6.**—Lung cancer mortality ratios of female cigarette smokers, compared to never smokers, by daily cigarette consumption

**SOURCE:** CPS-II 1982-86, ACS.

**FIGURE 7.**—Lung cancer death rates among females over time

**SOURCE:** CPS-I and CPS-II, ACS.
Type of Lung Cancer and Smoking

At the time of the 1964 Surgeon General’s Report, the Kreyberg classification of lung tumors was being investigated. Group I Kreyberg tumors included the epidermoid and small-cell histology types; Group 2 Kreyberg tumors included adenocarcinoma and bronchioalveolar cell types. It was felt at that time that the Group 1 tumors, but probably not the Group 2 tumors, were associated with smoking. The 1982 Surgeon General’s Report noted that smoking was related to all four major types of lung cancer: epidermoid, small cell, large cell, and adenocarcinoma.

A detailed study of trends in type of lung cancer has been reported from Olmsted County, MN, a region where a large percentage of medical care is provided through the Mayo Clinic. The investigators measured the incidence by type of lung cancer over a 45-year period. The incidence rates for squamous (epidermoid), adenocarcinoma, small-cell, and large-cell lung cancer all increased during this time (Figure 8) (Beard et al. 1985). Adenocarcinomas are more common than other cell types among nonsmokers, in whom lung cancer is rare.

Pipe and Cigar Smoking

Mortality ratios for lung cancer in those who have always smoked only cigars or pipes are significantly higher than in nonsmokers (US DHHS 1982). The mortality ratios are lower, however, than among those who have always smoked cigarettes. The risk of lung cancer increases in relation to the number of cigars smoked per day, the number of pipeful smoked per day, and the degree of smoke inhalation. The lower risk of lung cancer among pipe and cigar smokers compared with cigarette smokers is due to the lesser amount of tobacco smoked and the lower degree of inhalation.

Chemical analysis of the smoke from pipes, cigars, and cigarettes indicates that carcinogens are found in similar levels in the smoke of all these tobacco products. Additionally, experimental studies have shown that in a variety of animal models, smoke condensates from pipe and cigars are equally, if not more, carcinogenic than condensates from cigarettes (US DHEW 1979).

Determinants of Susceptibility

Since the 1964 Report, substantial epidemiologic and experimental investigation has been directed at the determinants of susceptibility to tobacco smoke; both environmental exposures and host characteristics have been investigated. The identification of determinants of susceptibility not only would further understanding of the mechanisms of carcinogenesis by tobacco smoking, but would offer new approaches for prevention of lung cancer by identification of smokers at higher risk. Synergistic interactions among risk factors may place persons with particular combinations of exposures at higher risk for lung cancer.

Interactions among risk factors, such as cigarette smoking and occupational exposures, may be either synergistic or antagonistic; synergism refers to an increased effect of the independent exposures when both are present, whereas antagonism refers to