

# **Chapter 4**

## **Advances in Knowledge of the Health Consequences of Smoking: From 1964–2014**

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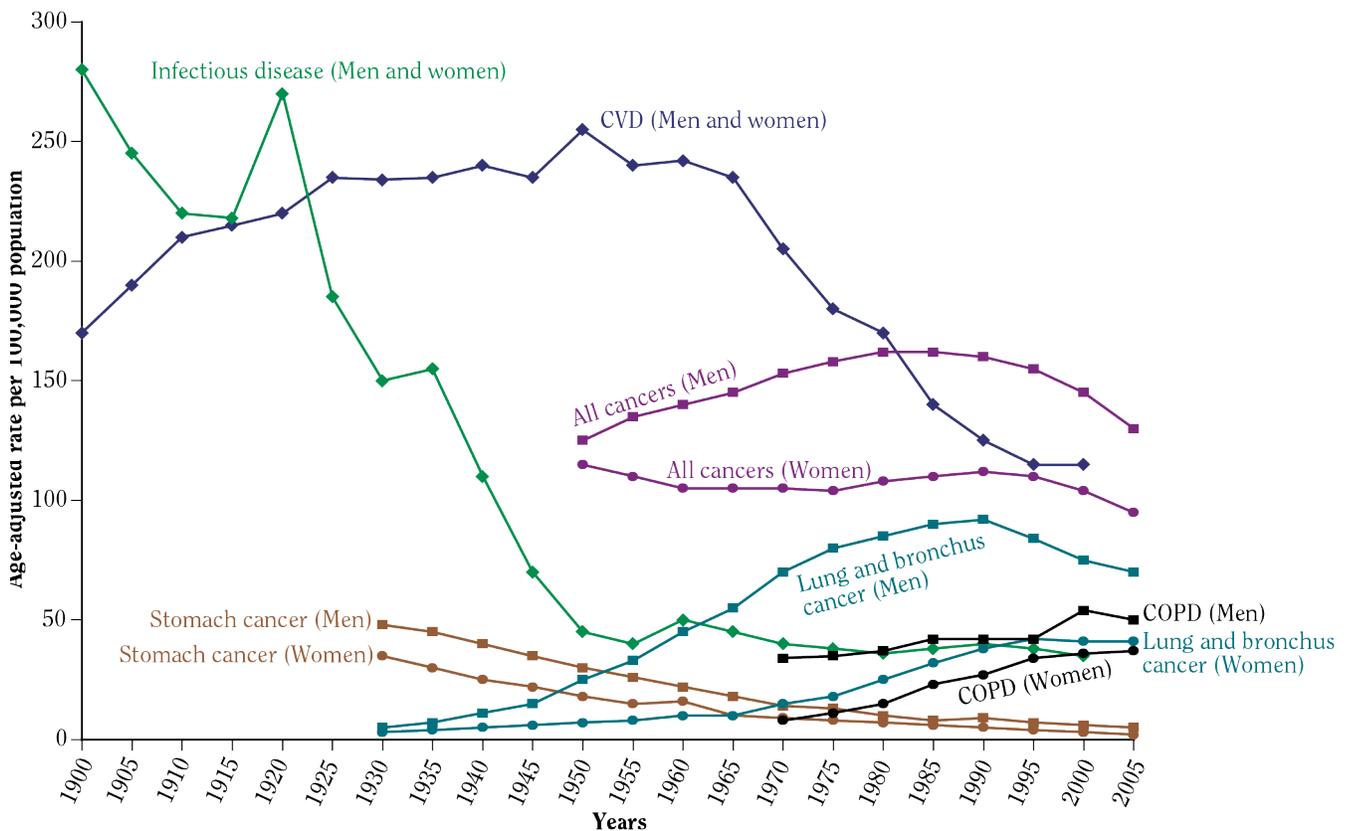
## Introduction

The 50-year span beginning in 1964 and ending in 2014 covers an era of remarkable advances in the understanding of disease etiology and opportunities for the prevention, diagnosis, and treatment of disease. There have also been striking changes seen in the incidence of disease, in mortality rates, and substantial gains in life expectancy. For example, in 1964 cancer was widely regarded as incurable and few causal agents had been identified, although tobacco smoke was already of concern because it had been identified as carcinogenic (Mukherjee 2010). Physicians and public health officials lacked today’s preventive strategies for coronary heart disease and widely used drugs, such as statins, had not yet been developed. Coronary care units for managing acute myocardial infarctions and heart rhythm disturbances were not in

existence. Chronic obstructive pulmonary disease (COPD) was recognized, but it was referred to as “chronic bronchitis” or “emphysema,” and the prevalence of what we now call COPD was far below the present level (Petty 2006; Kim and Criner 2013). Antibiotics were available for most bacterial infections, but not all infections could be cured with these drugs; antiviral agents, other than vaccines, were lacking altogether.

During the last half-century, major changes in disease occurrence have taken place that provide a critical context for the tobacco epidemic (Figure 4.1). The infectious diseases, particularly tuberculosis, declined as leading contributors to mortality to be replaced by the noncommunicable diseases: cardiovascular diseases, COPD, and cancer. Studies on the causes of these

**Figure 4.1 Mortality rates for major diseases in the United States, 1900–2005**



Source: Infectious disease and CVD rates from Cutler et al. 2006. Age-adjusted rates for stomach, lung, and bronchus cancer from American Cancer Society 2009. Age-standardized rate for all cancers from World Health Organization Mortality Database 2012. Age-adjusted rates for COPD from National Center for Health Statistics 2012.

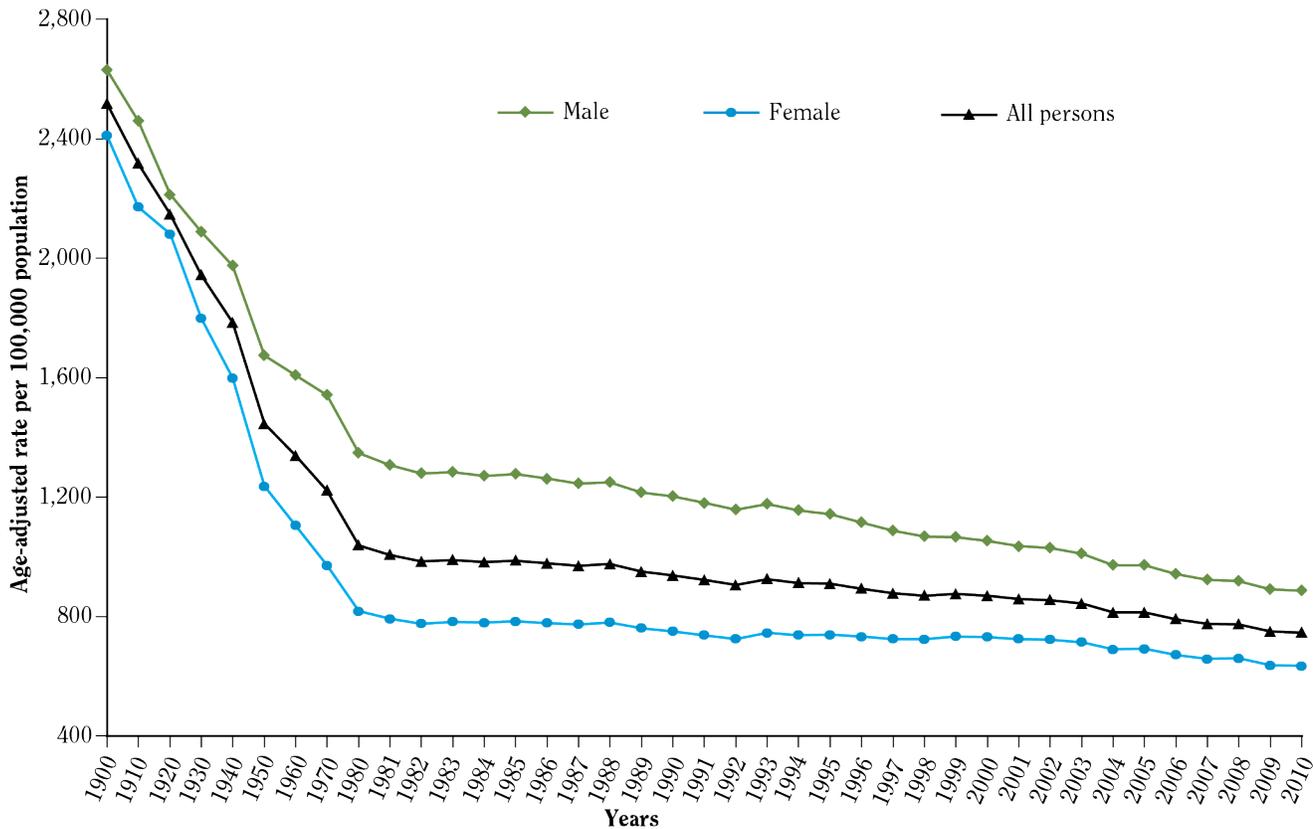
Note: COPD = chronic obstructive pulmonary disease; CVD = cardiovascular disease.

noncommunicable diseases were motivated by their rising frequency. Observational epidemiologic studies had a critical role in the search for causes, while complementary laboratory research expanded the understanding of the biological mechanisms by which risk factors caused these diseases. But even before 1964, advances had been made in characterizing the etiology of noncommunicable diseases. These advances relied on case-control and landmark cohort studies started in the late 1940s and 1950s, such as the Framingham Heart Study (which identified multiple risk factors for noncommunicable diseases, and explored blood pressure, lipids, and smoking in relationship to risk for incident coronary heart disease) (Kannel et al. 1961), the British Doctors Study in the United Kingdom (Doll and Hill 1954), and studies carried out by the American Cancer Society in the United States (Hammond and Horn 1954) linking cigarette smoking to multiple diseases. Findings from these studies figured prominently in the 1964 report, *Smoking and Health: Report of the Advisory Committee of the Surgeon General of the Public*

*Health Service*, and in subsequent reports as follow-up of participants continued and risks were tracked over time.

During the 50 years since the first Surgeon General's report on smoking and health in 1964 to this anniversary report, the observational evidence on the causes of noncommunicable diseases has continued to advance as numerous case-control and cohort studies were carried out and our understanding of the mechanistic processes leading to these diseases was greatly enhanced. Numerous risk factors were identified that have been classified by the Global Burden of Disease project into broad groups, including air pollution, tobacco smoking including exposure to secondhand smoke, alcohol and drug use, dietary risk factors and physical inactivity, physiological risk factors, and occupational risk factors (Lim et al. 2012). Many of these risk factors, such as physical inactivity, unhealthy diet, and smoking, could be avoided, making primary prevention possible. Pharmacological therapies provided control for some risk factors, such as treatment of lipid abnormalities with statins and other medications.

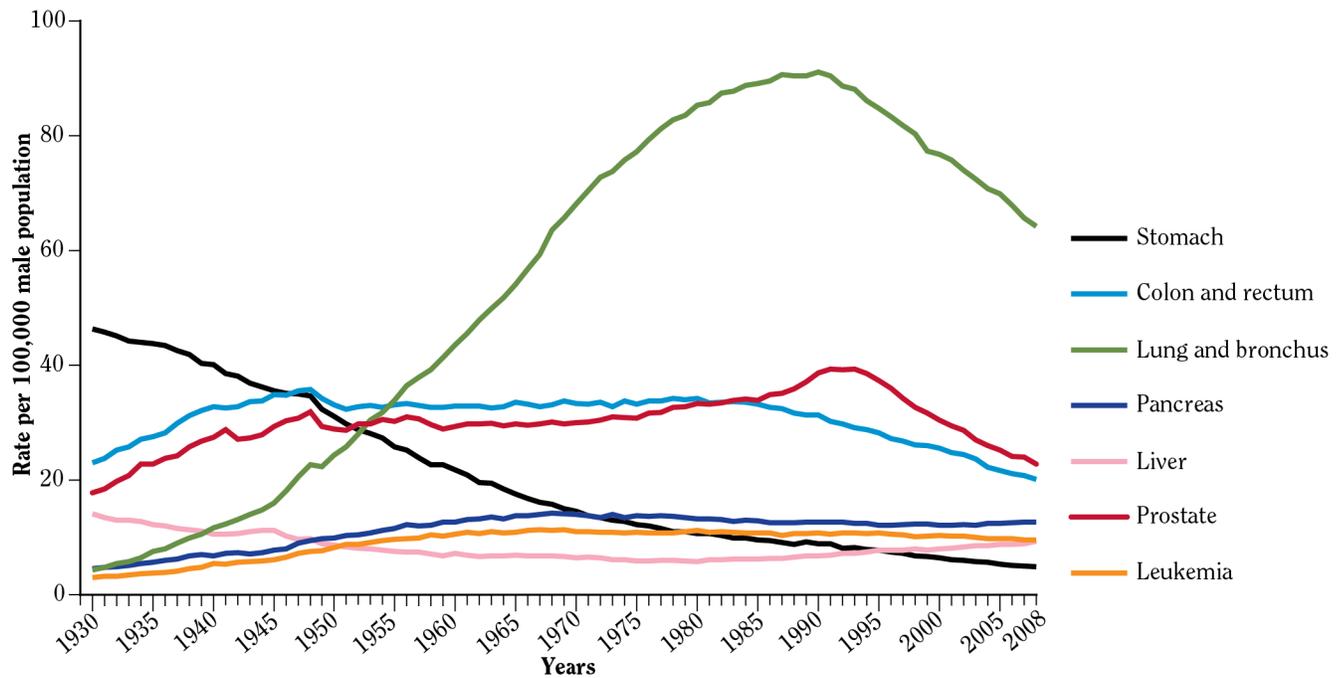
**Figure 4.2** Age-adjusted mortality rates for all causes<sup>a</sup>, United States, selected years, 1900–2010



Source: Hoyert et al. 2001; National Center for Health Statistics 2013.

<sup>a</sup>All causes of deaths combined.

**Figure 4.3 Mortality rates from selected cancers among men in the United States, 1930–2008<sup>a</sup>**



Source: Surveillance, Epidemiology, and End Results Program 2013.

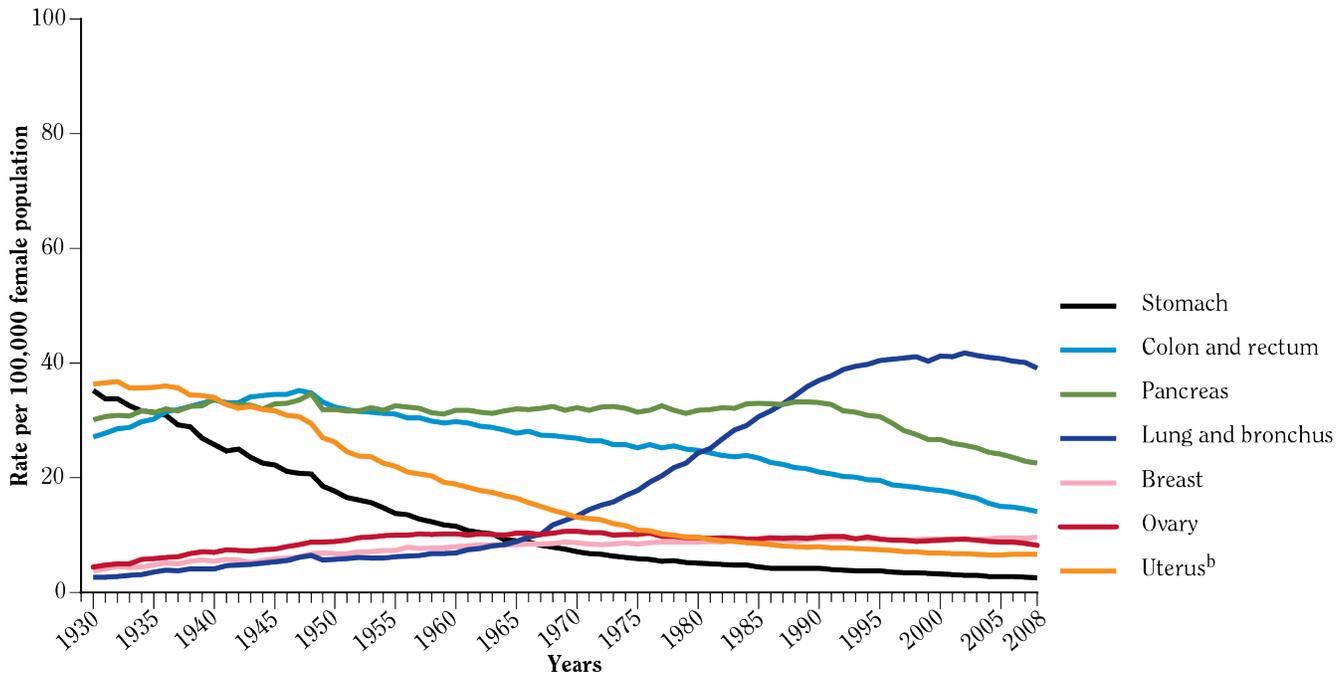
Note: Due to changes in *International Classification of Diseases* coding, numerator information has changed over time. Rates for cancer of the liver, lung and bronchus, and colon and rectum are affected by these coding changes.

<sup>a</sup>Per 100,000, age-adjusted to the 2000 U.S. standard population.

Another important advance over the last several decades has been the incorporation of genetics into research on the etiology of noncommunicable diseases, especially in the use of genetics to identify those men and women who are particularly susceptible to certain extrinsic exposures, such as cigarette smoking. For the diseases caused by smoking, emphasis has been placed on understanding why some people who are exposed to tobacco smoke develop disease while others do not. Also in the last few decades, the approaches used to explore the genetic basis of disease have evolved from family and linkage studies to genome-wide association studies (GWAS) (Wellcome Trust Case Control Consortium 2007). The GWAS approach involves comparing the distribution of markers (single nucleotide polymorphisms) across the genome between (a) people affected by the disease of interest and (b) a control population. To date, however, even though hundreds of thousands of markers across the genome have been examined, few promising associations have been found (Visscher et al. 2012), but work is in progress to further explore the GWAS-identified markers in greater depth (U.S. Department of Health and Human Services [USDHHS] 2010).

During the 50-year period reviewed in this report, there have been substantial changes in disease patterns in the United States. Figure 4.1 shows the rates for mortality for selected major diseases across the twentieth century, and Figure 4.2 shows the rates for all-cause mortality. Although the time spans covered differ for the various causes of death because of changes in coding used in the *International Classification of Diseases* and in the availability of data, major patterns are evident. These include the substantial decline in all-cause mortality (Figure 4.2) and the sharp drop in infectious disease mortality (Figure 4.1), both long antedating the general availability of modern antibiotics at mid-century. The rising mortality from lung cancer and cardiovascular disease that triggered numerous epidemiologic inquiries is also evident in Figure 4.1. In the later decades of the time period, rates for coronary heart disease mortality declined sharply, while lung cancer mortality in men reached a plateau and then began to decline around 1990. In contrast, lung cancer mortality in women rose, reaching a plateau by the century's end. Mortality from COPD, variably described across the century with labels including chronic bronchitis and

**Figure 4.4 Mortality rates from selected cancers among women in the United States, 1930–2008<sup>a</sup>**



Source: Surveillance, Epidemiology, and End Results Program 2013.

Note: Due to changes in *International Classification of Diseases* coding, numerator information has changed over time. Rates for cancer of the liver, lung and bronchus, and colon and rectum are affected by these coding changes.

<sup>a</sup>Per 100,000, age-adjusted to the 2000 U.S. standard population.

<sup>b</sup>Uterus refers to uterine cervix and uterine corpus combined.

emphysema, has risen progressively, even as death rates for other major diseases, such as cardiovascular disease and lung cancer caused by smoking have declined (Petty 2006; Kim and Criner 2013).

Figures 4.3 and 4.4, spanning 1930–2008, provide further detail on mortality rates for cancer in men and women. For both genders, the rise of lung cancer to become the leading cause of cancer death is evident. Stomach cancer, once the leading cause of cancer death in men and second among women in 1930, dropped so far as to eventually rank last among the seven cancers portrayed in Figures 4.3 and 4.4. Also during the 1930–2008 period, the uterine cancer mortality rate for women declined steeply. In addition, among women the mortality rate for lung cancer surpassed that for breast cancer in the 1980s and continued to rise to a plateau as breast cancer mortality declined. The mortality rate for pancreatic cancer rose slowly between 1930–2008 for both men and women. Although many factors have driven these changing patterns of disease, the patterns reflect, in part, the rise and fall of the prevalence of cigarette smoking across the twentieth century (USDHHS 2004; U.S. Burden of Disease

Collaborators 2013). Tobacco control measures, driven by the emerging findings on the health consequences of tobacco smoking, have been a key determinant of changes in these rates.

This chapter reviews the evolution of the conclusions in the Surgeon General's reports with regard to the health consequences of smoking. The chapters following this one review the evidence for diseases and other adverse effects for which the evidence was previously found to be suggestive, including macular degeneration, colorectal cancer, breast cancer, prostate cancer, and male sexual dysfunction. Additionally, the chapters cover several health outcomes that have not been comprehensively addressed in previous Surgeon General's reports, including general effects on the immune system and the development of several diseases in which the immune system plays a key role, such as tuberculosis, diabetes, rheumatoid arthritis, and systemic lupus erythematosus. The reviews extend to active smoking and exposure to secondhand smoke, as appropriate. New reviews in Chapter 8 cover the relationship between exposure to secondhand smoke and stroke and the potential that smokefree policies will reduce

the incidence of cardiovascular events. For lung cancer, COPD, and cardiovascular diseases—well-established major consequences of cigarette smoking—perspectives are provided on the most critical issues relative to smoking in the etiology of these diseases. Several chapters address general and nonspecific consequences of smoking tobacco. The clinically significant topic of smoking and outcomes following the diagnosis of cancer is covered for

the first time, including the impact of smoking on treatment outcomes for cancer sites that have not been causally related to smoking. Chapter 11 addresses general morbidity and all-cause mortality, and updated estimates of the burden of smoking-attributable mortality and morbidity and of the direct and indirect costs of smoking are provided in Chapter 12.

## Evolution of Conclusions on Cigarette Smoking and Exposure to Secondhand Smoke as a Cause of Disease

During the past 50 years, both the number and strength of the conclusions on active smoking and exposure to secondhand smoke as a cause of disease and other adverse health effects have increased markedly, moving from the two specific causal conclusions on lung cancer in males and on chronic bronchitis that were drawn in the 1964 report to numerous other conclusions that span most organs and now include exposure to secondhand smoke. Tables 4.1–4.5 address the evolution of the conclusions on active smoking, listing the report in which a particular health consequence was first mentioned; the strongest conclusion(s) reached before the 2004 report, *The Health Consequences of Smoking* (in which the classification of the strength of evidence was standardized); the conclusion(s) of the 2004 report; and any subsequent conclusions. The changes in the conclusions over time are characterized in this fashion because of the variable terminology used before the 2004 report (USDHHS 2004). Tables 4.6–4.10 provide a similar listing for exposure to secondhand smoke.

Although these conclusions relate primarily to specific diseases and other adverse health effects, the Surgeon General's reports have also tracked the evolution of the understanding of the pathogenesis and adverse health effects of these diseases and conditions. This deepening understanding has supported reaching stronger conclusions on causation. The 2010 report, *How Tobacco Smoke Causes Disease: The Biology and Behavioral Basis for Smoking-Attributable Disease*, provides conclusions specific to this topic (USDHHS 2010).

### Active Cigarette Smoking

Table 4.11 provides the conclusions formally adopted by the Advisory Committee to the Surgeon General in the

1964 report. The language ranges widely in describing the findings, from the clear conclusion that smoking causes lung cancer in men to the characterizations of the uncertainty and limitations of the evidence for some diseases. In most cases, the conclusions provide summary descriptions of the state of the evidence as well. The lack of knowledge of the mechanism(s) underlying the association of smoking with birth weight is mentioned.

In Table 4.1, which deals with active smoking and cancer, there has been consistency over time in the nomenclature so that interpretation of the changes in conclusions is not complicated by shifting terminology. With the exception of stomach cancer, causal conclusions were reached within the next two decades for cancer sites other than the lung that were mentioned in the 1964 report (i.e., oral cancer, laryngeal cancer, esophageal cancer, stomach cancer, and cancer of the urinary bladder) (U.S. Department of Health, Education, and Welfare [USDHEW] 1964). The 1982 report, *Health Consequences of Smoking – Cancer*, which focused on cancer, identified smoking as a contributory factor for pancreatic cancer and kidney cancer (USDHHS 1982). The list of cancers causally linked to active smoking lengthened with the 2004 report, which added cervical cancer and acute myeloid leukemia (USDHHS 2004). That report found the evidence on causation to be suggestive for breast cancer, colorectal cancer, and liver cancer. For prostate cancer, the evidence overall was not suggestive of a causal relationship.

For cardiovascular diseases (Table 4.2), the trends in the conclusions reflect the advancing understanding of the pathogenesis of these diseases and their common mechanistic basis (see Chapter 8 “Cardiovascular Diseases”). The 1964 report commented on the higher death rates from coronary artery disease among smokers compared with nonsmokers, but it expressed uncertainty with regard to the causal significance of the association

(USDHEW 1964). The conclusions on cardiovascular diseases strengthened throughout the next several decades. The 1979 report, *Smoking and Health*, offered a causal conclusion on coronary heart disease, but one that was introduced by the phrase “In summary, for the purposes of preventive medicine ...” (USDHEW 1979, p. 1-15). This apparently cautious phrasing may have been reflective of the preventive implications of the causal conclusion, however, and not an indication that there was some doubt about the statement. Later, the 2004 report found the evidence to be sufficient to infer causation for abdominal aortic aneurysm, atherosclerosis and peripheral vascular disease, cerebrovascular disease, and coronary heart disease (Table 4.2) (USDHHS 2004).

The conclusions on respiratory diseases over the years (Table 4.3) have addressed COPD, variably designated, as well as the respiratory symptoms caused by smoking and its reduction of lung function which, if sustained, leads to COPD. The 1964 report concluded that “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” (USDHEW 1964, p. 302). Although chronic bronchitis is the term long used for chronic cough and sputum production, at the time it was also used to refer to what is now called COPD. The 1984 report, *Health Consequences of Smoking: Chronic Obstructive Lung Disease*, which focused on the respiratory consequences of smoking, classified cigarette smoking as “... the major cause of COLD [chronic obstructive lung disease] morbidity in the United States...” (USDHHS 1984, p. 9). The 2004 report used the term COPD, finding the evidence to be sufficient to infer a causal relationship between smoking and both COPD morbidity and mortality (USDHHS 2004). The Surgeon General's reports have also addressed asthma, influenza, and pneumonia.

The effects of smoking on reproductive health (Table 4.4) have been addressed since the 1964 report, covering an increasing number and diversity of topics as the multiple adverse effects of smoking on reproductive health were identified. In fact, the 1964 report considered only birth weight and devoted just one page to the topic, citing just five retrospective and two prospective studies (USDHEW 1964). Over time, the effects of smoking have been found to extend from fertility to pregnancy and its outcome as well as the subsequent development of the child. There has also been substantial advancement in the understanding of how smoking affects reproductive health, the health of the fetus, and neurodevelopment as summarized in the 2010 report (USDHHS 2010). Male sexual functioning, not directly mentioned in the 1964 report, was covered extensively in the 2004 report (USDHHS 2004), and a causal conclusion on the relationship between smoking

and male sexual dysfunction has now been reached in this 2014 report.

Numerous other diseases and adverse consequences of smoking have been addressed in the reports of the Surgeon General (Table 4.5). These have included dental diseases, cataract and macular degeneration, peptic ulcer disease, fractures and osteoporosis, and diabetes. Nonspecific consequences of smoking have also been considered. All-cause mortality was covered in the 1964 report, but a specific conclusion was not offered. Several subsequent reports identified smoking as the leading cause of avoidable premature mortality (Table 4.12). The 2004 report assembled a wide range of evidence on nonspecific consequences of smoking, such as absenteeism and postoperative complications, with the report concluding that smoking caused “diminished health status” (Table 4.5), based on a review of a wide range of evidence (USDHHS 2004). The report's conclusion stated that diminished health status may manifest as “... increased absenteeism from work and increased use of medical care services” (USDHHS 2004, p. 29).

## Exposure to Secondhand Smoke

The topic of secondhand smoke was first considered in the 1972 Surgeon General's report, *Health Consequences of Smoking*, in a chapter titled “Public Exposure to Air Pollution from Tobacco Smoke” (USDHEW 1972). The involuntary inhalation of tobacco smoke by nonsmokers has been referred to in the Surgeon General's reports as involuntary smoking or passive smoking. The smoke inhaled has been called secondhand smoke or environmental tobacco smoke. This chapter in the 1972 report reviewed the accumulating evidence on levels of air pollutants, such as carbon monoxide, in indoor environments where people were smoking. The report concluded that “An atmosphere contaminated with tobacco smoke can contribute to the discomfort of many individuals” (USDHEW 1972, p. 7). The 1982 report, which had a chapter on the relationship between exposure to secondhand smoke and lung cancer (USDHHS 1982), reviewed the findings of three epidemiologic studies, but it did not offer a conclusion, while noting the limited evidence available. The 1986 report, *The Health Consequences of Involuntary Smoking*, was the first to have involuntary smoking as its topic, and the 2006 report followed suit, as it was titled *The Health Consequences of Involuntary Exposure to Tobacco Smoke* (USDHHS 1986, 2006).

The 1984 Surgeon General's report addressed COPD, and the report's chapter on passive smoking addressed the

respiratory consequences, other than cancer, of exposure to secondhand smoke. By that time, a substantial body of literature had accumulated on the respiratory consequences of exposure to secondhand smoke in children, and there was a more limited body of evidence related to adults. Notably, the conclusions in the 1984 report were overall summaries of the evidence and not statements as to the strength of the evidence for causation.

Exposure to secondhand smoke and its effects was the sole topic of the 1986 report. With regard to the effects of parental smoking on child respiratory health, that report addressed the range of outcomes considered in the 1984 report, comprehensively reviewed the evidence, and offered summary conclusions, but it did not provide statements on the strength of evidence for causation. The 1986 report did, however, comprehensively cover the relationship of lung cancer to exposure to secondhand smoke and concluded that involuntary smoking caused lung cancer in never smokers. This causal conclusion was repeated in the 2006 report, which also addressed exposure to secondhand smoke. That report also found sufficient evidence to infer causation for the principal adverse effects considered in the earlier reports. The 2006 report covered childhood cancers as well, but the evidence was not judged to be sufficient to infer a causal relationship for any of the malig-

nancies considered.

The 2001 report, *Women and Smoking*, had considered the relationship between exposure to secondhand smoke and breast cancer, and that topic was discussed in the 2006 report as well. Other cancers considered in relation to exposure to secondhand smoke included nasal sinus cavity and nasopharyngeal carcinoma (2006), and cervical cancer (2006); the conclusions drawn were that the evidence was either suggestive (breast cancer and nasal sinus cavity) or inadequate (nasopharyngeal carcinoma and cervical cancer). Reports after 2006 expanded the topics related to exposure to secondhand smoke and childhood health to include adverse effects on reproduction, risk for sudden infant death syndrome, and neurodevelopment.

The 1986 report did not cover exposure to secondhand smoke and cardiovascular diseases because only a few studies on that topic had been reported at that time. The 2001 report was the first to consider the topic, and found that the evidence did indicate a causal relationship. Finally, the 2006 report found that the evidence for a link between exposure to secondhand smoke and coronary heart disease was sufficient to infer a causal relationship, but it designated as suggestive the evidence for a similar link with atherosclerosis and cerebrovascular disease.

## Summary

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Over the 50 years that began with the seminal 1964 report, the conclusions of the Surgeon Generals' reports on smoking and health have evolved greatly, moving from the few causal associations set forth in the 1964 report to the inference of causal relationships between not only active smoking but also exposure to secondhand smoke and a wide range of diseases and other adverse health effects. The 2004 and 2006 reports provided comprehensive coverage of the evidence on active smoking and exposure to secondhand smoke, respectively, and the 2010 report addressed the mechanisms underlying the causal relationships described in these reports. The 2012 report,

*Preventing Tobacco Use Among Youth and Young Adults*, provided additional coverage of the effects of smoking on the health of children, adolescents, and young adults, highlighting the linkages between early life events and subsequent risk for disease (USDHHS 2012).

Notably, this 2014 review extends the list of diseases and other adverse health effects caused by smoking and reaffirms the widespread consequences of smoking. In the 2004 report, it was noted that smoking affects nearly every organ of the body; the evidence in this report provides additional support for that finding.

**Table 4.1 Conclusions from Surgeon General's report on active cigarette smoking and cancer**

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2014 Surgeon General's report
Bladder	"Available data suggest an association between cigarette smoking and urinary bladder cancer in the male but are not sufficient to support a judgment on the causal significance of this association." (1964, p. 225)	"Smoking is a cause of bladder cancer; cessation reduces risk by about 50 percent after only a few years, in comparison with continued smoking." (1990, p. 10)	"The evidence is sufficient to infer a causal relationship between smoking and ... bladder cancer." (p. 26)	—
Brain (adult)	—	—	"The evidence is suggestive of no causal relationship between smoking cigarettes and brain cancer in men and women." (p. 26)	—
Breast	"Thus, active smoking does not appear to appreciably affect breast cancer risk overall. However, several issues were not entirely resolved, including whether starting to smoke at an early age increases risk, whether certain subgroups defined by genetic polymorphisms are differentially affected by smoking, and whether ETS exposure affects risk." (2001, p. 217)	—	"The evidence is suggestive of no causal relationship between active smoking and breast cancer." (p. 26)	"The evidence is sufficient to identify mechanisms by which cigarette smoking may cause breast cancer." "The evidence is suggestive but not sufficient to infer a causal relationship between active smoking and breast cancer." (Chapter 6)
Cervical	"There are conflicting results in studies published to date on the existence of a relationship between smoking and cervical cancer; further research is necessary to define whether an association exists and, if so, whether that association is direct or indirect." (1982, p. 8)	"Smoking has been consistently associated with an increased risk for cervical cancer. The extent to which this association is independent of human papillomavirus infection is uncertain." (2001, p. 224) "Smoking may be associated with an increased risk for vulvar cancer, but the extent to which the association is independent of human papillomavirus infection is uncertain." (2001, p. 224)	"The evidence is sufficient to infer a causal relationship between smoking and cervical cancer." (p. 26)	—

Table 4.1 Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2014 Surgeon General's report
Colorectal	"Women who smoke may have increased risks for...colorectal cancer." (2001, p. 231)	—	"The evidence is suggestive but not sufficient to infer a causal relationship between smoking and colorectal adenomatous polyps and colorectal cancer." (p. 26)	"The evidence is sufficient to infer a causal relationship between smoking and colorectal adenomatous polyps and colorectal cancer." (Chapter 6)
Endometrial	"Several studies have reported that endometrial cancer is less frequent among women who smoke cigarettes than among nonsmokers (Baron et al. 1986). Cigarette smoking exerts an antiestrogenic effect that may explain this inverse association. The public health significance of this association is limited because of the overall adverse impact of cigarette smoking on morbidity and mortality." (1989, p. 58)	"Current smoking is associated with a reduced risk for endometrial cancer, but the effect is probably limited to postmenopausal disease. The risk for this cancer among former smokers generally appears more similar to that of women who have never smoked." (2001, p. 224)	"The evidence is sufficient to infer that current smoking reduces the risk of endometrial cancer in postmenopausal women." (p. 26)	—
Esophageal	"The evidence on the tobacco-esophageal cancer relationship supports the belief that an association exists. However, the data are not adequate to decide whether the relationship is causal." (1964, p. 218)	"Cigarette smoking is a major cause of esophageal cancer in the United States." (1982, p. 7)	"The evidence is sufficient to infer a causal relationship between smoking and cancers of the esophagus." (p. 26)	—
Kidney	"Cigarette smoking is a contributory factor in the development of kidney cancer in the United States. The term 'contributory factor' by no means excludes the possibility of a causal role for smoking in cancers of this site." (1982, p. 7)	"There is a positive association between smoking and kidney cancer, with relative risks ranging from 1 to more than 5. The increased risk of kidney cancer due to cigarette smoking is found for both males and females, and there is a dose-response relationship as measured by the number of cigarettes smoked per day." (1989, p. 56)	"The evidence is sufficient to infer a causal relationship between smoking and renal cell, [and] renal pelvis...cancers." (p. 26)	—

Table 4.1 Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2014 Surgeon General's report
Laryngeal	"Evaluation of the evidence leads to the judgment that cigarette smoking is a significant factor in the causation of laryngeal cancer in the male." (1964, p. 212)	"Cigarette smoking is causally associated with cancer of the lung, larynx, oral cavity, and esophagus in women as well as in men...." (1980, p. 126)	"The evidence is sufficient to infer a causal relationship between smoking and cancer of the larynx." (p. 25)	—
Leukemia (acute)	"Leukemia has recently been implicated as a smoking-related disease ... but this observation has not been consistent." (1990, p. 176)	"Smoking may be associated with an increased risk for acute myeloid leukemia among women but does not appear to be associated with other lymphoproliferative or hematologic cancers." (2001, p. 231)	"The evidence is sufficient to infer a causal relationship between smoking and acute myeloid leukemia." (p. 26)	—
Liver	"Primary hepatocellular cancer has been associated with smoking in a number of recent studies." (1990, p. 176)	"Women who smoke may have increased risks for liver cancer...." (2001, p. 231)	"The evidence is suggestive but not sufficient to infer a causal relationship between smoking and liver cancer." (p. 26)	"The evidence is sufficient to infer a causal relationship between smoking and hepatocellular carcinoma." (Chapter 6)

Table 4.1 Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2014 Surgeon General's report
Lung	<p>"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." (1964, p. 196)</p>	<p>"Additional epidemiological, pathological, and experimental data not only confirm the conclusion of the Surgeon General's 1964 Report regarding lung cancer in men but strengthen the causal relationship of smoking to lung cancer in women." (1967, p. 36)</p> <p>"Cigarette smoking is causally related to lung cancer in both men and women." (1979, p. 1-16)</p> <p>"Cigarette smoking is the major cause of lung cancer in the United States." (1982, p. 5)</p> <p>"Cigarette smoking is the major cause of lung cancer among women. About 90 percent of all lung cancer deaths among U.S. women smokers are attributable to smoking." (2001, p. 13)</p>	<p>"The evidence is sufficient to infer a causal relationship between smoking and lung cancer." (p. 25)</p>	<p>"The evidence is sufficient to conclude that the risk of developing adenocarcinoma of the lung from cigarette smoking has increased since the 1960s."</p> <p>"The evidence is sufficient to conclude that the increased risk of adenocarcinoma of the lung in smokers results from changes in the design and composition of cigarettes since the 1950s."</p> <p>"The evidence is not sufficient to specify which design changes are responsible for the increased risk of adenocarcinoma, but there is suggestive evidence that ventilated filters and increased levels of tobacco-specific nitrosamines have played a role."</p> <p>"The evidence shows that the decline of squamous carcinoma follows the trend of declining smoking prevalence." (Chapter 6)</p>

**Table 4.1 Continued**

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2014 Surgeon General's report
Oral cavity and pharyngeal	<p>"The causal relationship of the smoking of pipes to the development of cancer of the lip appears to be established." (1964, p. 204)</p> <p>"Although there are suggestions of relationships between cancer of other specific sites of the oral cavity and the several forms of tobacco use, their causal implications cannot at present be stated." (1964, p. 205)</p>	<p>"Epidemiological studies indicate that smoking is a significant causal factor in the development of oral cancer. The risk increases with the number of cigarettes smoked per day." (1979, p. 1-17)</p> <p>"Cigarette smoking is a major cause of cancers of the oral cavity in the United States." (1982, p. 6)</p>	<p>"The evidence is sufficient to infer a causal relationship between smoking and cancers of the oral cavity and pharynx." (p. 25)</p>	—
Ovarian	<p>"Smoking does not appear to be associated with risk for ovarian cancer." (2001, p. 224)</p>	—	<p>"The evidence is inadequate to infer the presence or absence of a causal relationship between smoking and ovarian cancer." (p. 26)</p>	—
Pancreatic	<p>"Cigarette smoking is a contributory factor in the development of pancreatic cancer in the United States. This relationship is not as strong as that noted for the association between smoking and cancers of the lung, larynx, oral cavity, and esophagus. The term 'contributory factor' by no means excludes the possibility of a causal role for smoking in cancers of this site." (1982, p. 7)</p>	<p>"Smoking cessation reduces the risk of pancreatic cancer, compared with continued smoking, although this reduction in risk may only be measurable after 10 years of abstinence." (1990, p. 10)</p>	<p>"The evidence is sufficient to infer a causal relationship between smoking and pancreatic cancer." (p. 26)</p>	—

Table 4.1 Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2014 Surgeon General's report
Prostate cancer	—	—	“The evidence is suggestive of no causal relationship between smoking and risk for prostate cancer.” (p. 26)	“The evidence is suggestive of no causal relationship between smoking and the risk of incident prostate cancer.” “The evidence is suggestive of a higher risk of death from prostate cancer in smokers than in nonsmokers.” “In men who have prostate cancer, the evidence is suggestive of a higher risk of advanced-stage disease and less well-differentiated cancer in smokers than in nonsmokers, and— independent of stage and histologic grade—a higher risk of disease progression.” (Chapter 6)
Stomach	“No relationship has been established between tobacco use and stomach cancer.” (1964, p. 229)	“Data on smoking and cancer of the stomach ... are unclear.” (2001, p. 231)	“The evidence is sufficient to infer a causal relationship between smoking and gastric cancers.” (p. 26)	—

Note: ETS = environmental tobacco smoke.

<sup>a</sup>Refers to a general conclusion that was reached for breast cancer.

**Table 4.2 Conclusions from Surgeon General's report on active cigarette smoking and cardiovascular diseases**

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2012/2014 Surgeon General's report
Abdominal aortic aneurysm	"Cigarette smoking is a strong risk factor for atherosclerotic aortic aneurysm." (1979, p. 4-56)	"Death from rupture of an atherosclerotic abdominal aneurysm is more common in cigarette smokers than in nonsmokers." (1983, p. 195)	"The evidence is sufficient to infer a causal relationship between smoking and abdominal aortic aneurysm." (p. 27)	"The evidence is sufficient to conclude that there is a causal relationship between active smoking in adolescence and young adulthood and early abdominal aortic atherosclerosis in young adults." (2012, p. 111)
Atherosclerosis/peripheral vascular disease	"Autopsy studies suggest that cigarette smoking is associated with a significant increase in the atherosclerosis of the aorta and coronary arteries." (1969, p. 4)	"Cigarette smoking is the most powerful risk factor predisposing to atherosclerotic peripheral vascular disease." (1983, p. 8)	"The evidence is sufficient to infer a causal relationship between smoking and subclinical atherosclerosis." (p. 26)	"The evidence is suggestive but not sufficient to conclude that there is a causal relationship between smoking in adolescence and young adulthood and coronary artery atherosclerosis in adulthood." (2012, p. 111)
Cerebrovascular disease	"Additional evidence strengthens the association between cigarette smoking and cerebrovascular disease, and suggests that some of the pathogenetic [sic] considerations pertinent to coronary heart disease may also apply to cerebrovascular disease." (1967, p. 28)	"Cigarette smoking is a major cause of cerebrovascular disease (stroke), the third leading cause of death in the United States." (1989, p. 12)	"The evidence is sufficient to infer a causal relationship between smoking and stroke." (p. 27)	—
Coronary heart disease	"Male cigarette smokers have a higher death rate from coronary artery disease than non-smoking males, but it is not clear that the association has causal significance." (1964, p. 327)	"In summary, for the purposes of preventive medicine, it can be concluded that smoking is causally related to coronary heart disease for both men and women in the United States." (1979, p. 1-15)	"The evidence is sufficient to infer a causal relationship between smoking and coronary heart disease." (p. 27)	—

**Table 4.3 Conclusions from Surgeon General’s report on active cigarette smoking and respiratory diseases**

<b>Disease</b>	<b>First mention and finding(s) in a Surgeon General’s report (year)</b>	<b>Highest level conclusion(s) from subsequent Surgeon General’s reports before 2004 (year)</b>	<b>Conclusion(s) from the 2004 Surgeon General’s report</b>	<b>Additional or updated conclusion(s) from the 2012/2014 Surgeon General’s report</b>
Asthma	“Cigarette smoking does not appear to cause asthma.” (1964, p. 302)	—	<p>“The evidence is inadequate to infer the presence or absence of a causal relationship between active smoking and asthma in adults.” (p. 28)</p> <p>“The evidence is suggestive but not sufficient to infer a causal relationship between active smoking and increased nonspecific bronchial hyperresponsiveness.” (p. 28)</p> <p>“The evidence is sufficient to infer a causal relationship between active smoking and poor asthma control.” (p. 28)</p>	<p>“The evidence is sufficient to conclude that there is a causal relationship between active smoking and wheezing severe enough to be diagnosed as asthma in susceptible child and adolescent populations.” (2012, p. 111)</p> <p>“The evidence is suggestive but not sufficient to infer a causal relationship between active smoking and incidence of asthma in adolescents.” (2014, Chapter 7)</p> <p>“The evidence is suggestive but not sufficient to infer a causal relationship between active smoking and exacerbation of asthma among children and adolescents.” (2014, Chapter 7)</p> <p>“The evidence is suggestive but not sufficient to infer a causal relationship between active smoking and the incidence of asthma in adults.” (2014, Chapter 7)</p> <p>“The evidence is sufficient to infer a causal relationship between active smoking and exacerbation of asthma in adults.” (2014, Chapter 7)</p>

**Table 4.3 Continued**

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2012/2014 Surgeon General's report
COPD (Formerly designated as chronic bronchitis; emphysema; GOLD; chronic obstructive bronchopulmonary disease)	<p>"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." (1964, p. 302)</p> <p>"A relationship exists between pulmonary emphysema and cigarette smoking but it has not been established that the relationship is causal. The smoking of cigarettes is associated with an increased risk of dying from pulmonary emphysema." (1964, p. 302)</p>	<p>"Cigarette smoking is the major cause of GOLD ... morbidity in the United States; 80 to 90 percent of GOLD in the United States is attributable to cigarette smoking." (1984, p. 9)</p> <p>"Cigarette smoking is a primary cause of COPD among women, and the risk increases with the amount and duration of smoking. Approximately 90 percent of mortality from COPD among women in the United States can be attributed to cigarette smoking." (2001, p. 14)</p>	<p>"The evidence is sufficient to infer a causal relationship between active smoking and chronic obstructive pulmonary disease morbidity and mortality." (p. 28)</p>	<p>"The evidence is sufficient to infer that smoking is the dominant cause of chronic obstructive pulmonary disease (COPD) in men and women in the United States. Smoking causes all elements of the COPD phenotype, including emphysema and damage to the airways of the lung." (2014, Chapter 7)</p> <p>"Chronic obstructive pulmonary disease mortality has increased dramatically in men and women since the 1964 Surgeon General's report. The number of women dying from COPD now surpasses the number of men." (2014, Chapter 7)</p> <p>"The evidence is suggestive but not sufficient to infer that women are more susceptible to develop severe chronic obstructive pulmonary disease at younger ages." (2014, Chapter 7)</p> <p>"The evidence is sufficient to infer that severe <math>\alpha</math>-1-antitrypsin deficiency and cutis laxa are genetic causes of chronic obstructive pulmonary disease." (2014, Chapter 7)</p>

Table 4.3 Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2012/2014 Surgeon General's report
Chronic respiratory symptoms (cough, phlegm, wheeze, dyspnea, etc.)	"Cough, sputum production, or the two combined are consistently more frequent among cigarette smokers than among non-smokers." (1964, p. 302)	"Cigarette smokers have an increased frequency of respiratory symptoms, and at least two of them, cough and sputum production, are dose-related." (1979, p. 1-18)	"The evidence is sufficient to infer a causal relationship between active smoking and all major respiratory symptoms among adults, including coughing, phlegm, wheezing, and dyspnea." (p. 28)	—
Influenza, pneumonia, infections, and acute respiratory illnesses	"Although death certification shows that cigarette smokers have a moderately increased risk of death from influenza and pneumonia, an association of cigarette smoking and infectious diseases is not otherwise substantiated." (1964, p. 302)	"Smoking cessation reduces rates of respiratory symptoms such as cough, sputum production, and wheezing, and respiratory infections such as bronchitis and pneumonia, compared with continued smoking." (1990, p. 11)	"The evidence is sufficient to infer a causal relationship between smoking and acute respiratory illnesses, including pneumonia, in persons without underlying smoking-related chronic obstructive lung disease." (p. 27)	"The evidence is suggestive but not sufficient to infer a causal relationship between smoking and acute respiratory infections among persons with preexisting chronic obstructive pulmonary disease." (p. 27)

Table 4.3 Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2012/2014 Surgeon General's report
Tuberculosis	—	—	—	<p>"The evidence is sufficient to infer a causal relationship between smoking and an increased risk of <i>Mycobacterium tuberculosis</i> disease." (2014, Chapter 7)</p> <p>"The evidence is sufficient to infer a causal relationship between smoking and mortality due to tuberculosis." (2014, Chapter 7)</p> <p>"The evidence is suggestive of a causal relationship between smoking and the risk of recurrent tuberculosis disease." (2014, Chapter 7)</p> <p>"The evidence is inadequate to infer the presence or absence of a causal relationship between active smoking and the risk of tuberculosis infection." (2014, Chapter 7)</p>
Lung function level	"Cigarette smoking is associated with a reduction in ventilatory function. Among males, cigarette smokers have a greater prevalence of breathlessness than non-smokers." (1964, p. 302)	"Cigarette smoking accelerates the age-related decline in lung function that occurs among never smokers. With sustained abstinence from smoking, the rate of decline in pulmonary function among former smokers returns to that of never smokers." (1990, p. 11)	<p>"The evidence is sufficient to infer a causal relationship between active smoking in adulthood and a premature onset of and an accelerated age-related decline in lung function." (p. 27)</p> <p>"The evidence is sufficient to infer a causal relationship between sustained cessation from smoking and a return of the rate of decline in pulmonary function to that of persons who had never smoked." (p. 27)</p>	<p>"The evidence is sufficient to conclude that there is a causal relationship between active smoking and both reduced lung function and impaired lung growth during childhood and adolescence." (2012, p. 111)</p>

Table 4.3 Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2012/2014 Surgeon General's report
Respiratory effects due to active smoking during childhood and adolescence	—	"Cigarette smoking during childhood and adolescence produces significant health problems among young people, including cough and phlegm production, an increased number and severity of respiratory illnesses, decreased physical fitness, an unfavorable lipid profile, and potential retardation in the rate of lung growth and the level of maximum lung function." (1994, p. 41)	<p>"The evidence is sufficient to infer a causal relationship between active smoking and impaired lung growth during childhood and adolescence." (p. 27)</p> <p>"The evidence is sufficient to infer a causal relationship between active smoking and the early onset of lung function decline during late adolescence and early adulthood." (p. 27)</p> <p>"The evidence is sufficient to infer a causal relationship between active smoking and respiratory symptoms in children and adolescents, including coughing, phlegm, wheezing, and dyspnea." (p. 27)</p> <p>"The evidence is sufficient to infer a causal relationship between active smoking and asthma-related symptoms (i.e., wheezing) in childhood and adolescence." (p. 27)</p>	—

Note: **COLD** = chronic obstructive lung disease; **COPD** = chronic obstructive pulmonary disease.

**Table 4.4 Conclusions from Surgeon General's report on active cigarette smoking and adverse reproductive outcomes or childhood neurobehavioral disorders**

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2014 Surgeon General's report
Child physical, behavioral, and cognitive development	"According to studies of long-term growth and development, smoking during pregnancy may affect physical growth, mental development, and behavioral characteristics of children at least up to the age of 11." (1979, p. 1-21)	"Maternal smoking during pregnancy may adversely affect the child's long-term growth, intellectual development, and behavioral characteristics." (1980, p. 11)	"The evidence is inadequate to infer the presence or absence of a causal relationship between maternal smoking and physical growth and neurocognitive development of children." (p. 28)	<p>"The evidence is suggestive but not sufficient to infer a causal relationship between maternal prenatal smoking and disruptive behavioral disorders, and ADHD in particular, among children."</p> <p>"The evidence is insufficient to infer the presence or absence of a causal relationship between maternal prenatal smoking and anxiety and depression in children."</p> <p>"The evidence is insufficient to infer the presence or absence of a causal relationship between maternal prenatal smoking and Tourette syndrome."</p> <p>"The evidence is insufficient to infer the presence or absence of a causal relationship between maternal prenatal smoking and schizophrenia in her offspring."</p> <p>"The evidence is insufficient to infer the presence or absence of a causal relationship between maternal prenatal smoking and intellectual disability." (Chapter 9)</p>

**Table 4.4 Continued**

<b>Disease</b>	<b>First mention and finding(s) in a Surgeon General's report (year)</b>	<b>Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)</b>	<b>Conclusion(s) from the 2004 Surgeon General's report</b>	<b>Additional or updated conclusion(s) from the 2014 Surgeon General's report</b>
Congenital malformations	"...no conclusions can be drawn about any relationship between maternal cigarette smoking and congenital malformations at the present time." (1973, p. 137)	"The accumulated evidence does not support a conclusion that maternal smoking increases the incidence of congenital malformations." (1979, p. 1-22) "There are insufficient data to support a judgment on whether maternal and/or paternal cigarette smoking increases the risk of congenital malformations." (1980, p. 11) "Smoking does not appear to affect the overall risk for congenital malformations." (2001, p. 307)	"The evidence is inadequate to infer the presence or absence of a causal relationship between maternal smoking and congenital malformations in general." (p. 28) "The evidence is suggestive but not sufficient to infer a causal relationship between maternal smoking and oral clefts." (p. 28)	"The evidence is sufficient to infer a causal relationship between maternal smoking in early pregnancy and orofacial clefts." "The evidence is suggestive but not sufficient to infer a causal relationship between maternal smoking in early pregnancy and clubfoot, gastroschisis, and atrial septal heart defects." (Chapter 9)
Fertility	"Studies in women and men suggest that cigarette smoking may impair fertility." (1980, p. 12)	"The available information suggests that current smoking is related to low sperm density. However, these data are limited." (1990, p. 405) "Women who smoke have increased risks for conception delay and for both primary and secondary infertility." (2001, p. 307)	"The evidence is sufficient to infer a causal relationship between smoking and reduced fertility in women." (p. 28) "The evidence is inadequate to infer the presence or absence of a causal relationship between active smoking and sperm quality." (p. 28)	—
Fetal death, stillbirths, and infant mortality	"...it appears that maternal smoking during pregnancy may be associated with an increased incidence of spontaneous abortion, stillbirth, and neonatal death and that this relationship may be most marked in the presence of other risk factors." (1969, p. 5)	"Cigarette smoking is now considered to be a probable cause of ...increased infant mortality." (1989, p. 20) "The risk for perinatal mortality—both stillbirth and neonatal deaths—and the risk for sudden infant death syndrome (SIDS) are increased among the offspring of women who smoke during pregnancy." (2001, p. 307) "Women who smoke may have a modest increase in risks for... spontaneous abortion." (2001, p. 307)	—	"The evidence is suggestive but not sufficient to infer a causal relationship between maternal active smoking and spontaneous abortion." (Chapter 9)

**Table 4.4 Continued**

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2014 Surgeon General's report
Infant birth weight	<p>"Women who smoke cigarettes during pregnancy tend to have babies of lower birth weight." (1964, p. 343)</p>	<p>"Infants born to women who smoke during pregnancy have a lower average birth weight...than infants born to women who do not smoke." (2001, p. 307)</p> <p>"Infants born to women who smoke during pregnancy ...are more likely to be small for gestational age than are infants born to women who do not smoke." (2001, p. 307)</p>	<p>"The evidence is sufficient to infer a causal relationship between maternal active smoking and fetal growth restriction and low birth weight." (p. 28)</p>	<p>—</p>
Male sexual function	<p>"...element of masculinity as indicated by external morphologic features" ... "weakness of the masculine component is significantly more frequent in smokers than in nonsmokers, and most frequent in heavy smokers." (1964, pp. 383-4)</p>	<p>"In summary, the level of sexual activity does not appear to be affected by cigarette smoking. Cigarette smoking may be associated with impaired male sexual performance. ... Because of limited and uncontrolled data, no conclusions can be drawn regarding sexual performance or PBI among former smokers." (1990, pp. 403-4)</p>	<p>"The evidence is suggestive but not sufficient to infer a causal relationship between smoking and erectile dysfunction." (p. 29)</p>	<p>"The evidence is sufficient to infer a causal relationship between smoking and erectile dysfunction." (Chapter 9)</p>

**Table 4.4 Continued**

<b>Disease</b>	<b>First mention and finding(s) in a Surgeon General's report (year)</b>	<b>Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)</b>	<b>Conclusion(s) from the 2004 Surgeon General's report</b>	<b>Additional or updated conclusion(s) from the 2014 Surgeon General's report</b>
Pregnancy complications	<p>"Maternal smoking increases the risk of fetal death through maternal complications such as abruptio placenta, placenta previa, antepartum hemorrhage, and prolonged rupture of membranes." (1979, p. 1-22)</p> <p>"Smoking during pregnancy is associated with increased risks for preterm premature rupture of membranes, abruptio placentae, and placenta previa, and with a modest increase in risk for preterm delivery." (2001, p. 14)</p> <p>"Women who smoke may have a modest increase in risks for ectopic pregnancy and spontaneous abortion." (2001, p. 14)</p> <p>"Women who smoke during pregnancy have a decreased risk for preeclampsia." (2001, p. 14)</p>	<p>"Smoking during pregnancy is associated with increased risks for preterm premature rupture of membranes, abruptio placentae, and placenta previa, and with a modest increase in risk for preterm delivery." (2001, p. 14)</p> <p>"Women who smoke may have a modest increase in risks for ectopic pregnancy and spontaneous abortion." (2001, p. 14)</p> <p>"Women who smoke during pregnancy have a decreased risk for preeclampsia." (2001, p. 14)</p>	<p>"The evidence is sufficient to infer a causal relationship between maternal active smoking and premature rupture of the membranes, placenta previa, and placental abruption." (p. 28)</p> <p>"The evidence is sufficient to infer a causal relationship between maternal active smoking and shortened preterm delivery and shortened gestation." (p. 28)</p> <p>"The evidence is sufficient to infer a causal relationship between maternal active smoking and a reduced risk for preeclampsia." (p. 28)</p> <p>"The evidence is suggestive but not sufficient to infer a causal relationship between maternal active smoking and ectopic pregnancy." (p. 28)</p> <p>"The evidence is suggestive but not sufficient to infer a causal relationship between maternal active smoking and spontaneous abortion." (p. 28)</p>	<p>"The evidence is sufficient to infer a causal relationship between maternal active smoking and ectopic pregnancy." (Chapter 9)</p>

**Table 4.4** Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2014 Surgeon General's report
Respiratory effects in infants and children due to maternal active smoking	—	"In utero exposure to maternal smoking is associated with reduced lung function among infants...." (2001, p. 14)	"The evidence is sufficient to infer a causal relationship between maternal smoking during pregnancy and a reduction of lung function in infants." (p. 27)  "The evidence is suggestive but not sufficient to infer a causal relationship between maternal smoking during pregnancy and an increase in the frequency of lower respiratory tract illnesses during infancy." (p. 27)  "The evidence is suggestive but not sufficient to infer a causal relationship between maternal smoking during pregnancy and an increased risk for impaired lung function in childhood and adulthood." (p. 27)	—
Sudden infant death syndrome (SIDS)	"Smoking by pregnant women contributes to the risk of their infants being victims of the "sudden infant death syndrome." (1979, p. 1-22)	"... the risk for sudden infant death syndrome (SIDS) are increased among the offspring of women who smoke during pregnancy." (2001, p. 307)	"The evidence is sufficient to infer a causal relationship between sudden infant death syndrome and maternal smoking during and after pregnancy." (p. 28)	—

*Note:* **ADHD** = attention deficit hyperactivity disorder; **PBI** = penile-brachial index.

**Table 4.5 Conclusions from Surgeon General’s report on active cigarette smoking and other adverse health effects**

<b>Disease</b>	<b>First mention and finding(s) in a Surgeon General’s report (year)</b>	<b>Highest level conclusion(s) from subsequent Surgeon General’s reports before 2004 (year)</b>	<b>Conclusion(s) from the 2004 Surgeon General’s report</b>	<b>Additional or updated conclusion(s) from the 2014 Surgeon General’s report</b>
Accidents	<p>“Smoking is associated with accidental deaths from fires in the home.” (1964, p. 39)</p> <p>“No conclusive information is available on the effects of smoking on traffic accidents.” (1964, p. 39)</p>	—	—	—
Dental diseases	<p>“Tobacco use, excessive alcohol use, and inappropriate dietary practices contribute to many diseases and disorders. In particular, tobacco use is a risk factor for oral cavity and pharyngeal cancers, periodontal diseases, candidiasis, and dental caries, among other diseases.” (2000, p. 6)<sup>a</sup></p>	—	<p>“The evidence is sufficient to infer a causal relationship between smoking and periodontitis.” (p. 29)</p> <p>“The evidence is inadequate to infer the presence or absence of a causal relationship between smoking and coronal dental caries.” (p. 29)</p> <p>“The evidence is suggestive but not sufficient to infer a causal relationship between smoking and root-surface caries.” (p. 29)</p>	<p>“The evidence is suggestive but not sufficient to infer a causal relationship between active cigarette smoking and dental caries.”</p> <p>“The evidence is suggestive but not sufficient to infer a causal relationship between cigarette smoking and failure of dental implants.” (Chapter 10)</p>
Diabetes mellitus	—	<p>“Smoking appears to affect glucose regulation and related metabolic processes, but conflicting data exist on the relationship of smoking and the development of type 2 diabetes mellitus and gestational diabetes among women.” (2001, p. 14)</p>	—	<p>“The evidence is sufficient to infer that cigarette smoking is a cause of diabetes.”</p> <p>“The risk of developing diabetes is 30–40% higher for active smokers than nonsmokers.”</p> <p>“There is a positive dose-response relationship between the number of cigarettes smoked and the risk of developing diabetes.” (Chapter 10)</p>

**Table 4.5 Continued**

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2014 Surgeon General's report
Diminished health status	—	—	<p>“The evidence is sufficient to infer a causal relationship between smoking and diminished health status that may manifest as increased absenteeism from work and increased use of medical care services.” (p. 29)</p> <p>“The evidence is sufficient to infer a causal relationship between smoking and increased risks for adverse surgical outcomes related to wound healing and respiratory complications.” (p. 29)</p>	—
Eye diseases	<p>“Tobacco amblyopia had been related to pipe and cigar smoking by clinical impressions. The association has not been substantiated by epidemiological or experimental studies.” (1964, p. 342)</p>	<p>“Women who smoke have an increased risk for cataract.” (2001, p. 15)</p> <p>“Women who smoke may have an increased risk for age-related macular degeneration.” (2001, p. 15)</p> <p>“Studies show no consistent association between smoking and open-angle glaucoma.” (2001, p. 15)</p>	<p>“The evidence is sufficient to infer a causal relationship between cigarette smoking and neovascular and atrophic forms of age-related macular degeneration.”</p> <p>“The evidence is suggestive but not sufficient to infer that smoking cessation reduces the risk of advanced age-related macular degeneration.” (Chapter 10).</p>	—
Hip fractures	—	<p>“Women who currently smoke have an increased risk for hip fracture compared with women who do not smoke.” (2001, p. 321)</p>	<p>“The evidence is sufficient to infer a causal relationship between smoking and hip fractures.” (p. 29)</p>	—
Liver cirrhosis	<p>“Increased mortality of smokers from cirrhosis of the liver has been shown in the prospective studies. The data are not sufficient to support a direct or causal association.” (1964, p. 342)</p>	—	—	—

Table 4.5 Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2004 (year)	Conclusion(s) from the 2004 Surgeon General's report	Additional or updated conclusion(s) from the 2014 Surgeon General's report
Low bone density	—	"Postmenopausal women who currently smoke have lower bone density than do women who do not smoke." (2001, p. 321)	"In postmenopausal women, the evidence is sufficient to infer a causal relationship between smoking and low bone density." (p. 29)	—
Peptic ulcer	"Epidemiological studies indicate an association between cigarette smoking and peptic ulcer which is greater for gastric than for duodenal ulcer." (1964, p. 340)	"The relationship between cigarette smoking and death rates from peptic ulcer, especially gastric ulcer, is confirmed. In addition, morbidity data suggest a similar relationship exists with the prevalence of reported disease from this cause." (1967, p. 40)	"The evidence is sufficient to infer a causal relationship between smoking and peptic ulcer disease in persons who are <i>Helicobacter pylori</i> positive." (p. 29)	—

<sup>a</sup>U.S. Department of Health and Human Services 2000. *Oral Health in America: A Report of the Surgeon General*.

**Table 4.6 Conclusions from Surgeon General's report on exposure to secondhand smoke and cancer<sup>a</sup>**

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2006 (year)	Conclusion(s) from the 2006 Surgeon General's report	Additional or updated conclusion(s) from the 2014 Surgeon General's report
Breast	—	“Several studies suggest that exposure to environmental tobacco smoke is associated with an increased risk of breast cancer, but this association remains uncertain.” (2001, p. 13)	“The evidence is suggestive but not sufficient to infer a causal relationship between secondhand smoke and breast cancer.” (p. 15)	“The evidence is suggestive but not sufficient to infer a causal relationship between exposure to secondhand tobacco smoke and breast cancer.” (Chapter 6)
Cervical	—	—	“The evidence is inadequate to infer the presence or absence of a causal relationship between secondhand smoke exposure and the risk of cervical cancer among lifetime nonsmokers.” (p. 15)	—
Lung	“Although the currently available evidence is not sufficient to conclude that passive or involuntary smoking causes lung cancer in nonsmokers, the evidence does raise concern about a possible serious public health problem.” (1982, p. 9)	“Involuntary smoking can cause lung cancer in nonsmokers.” (1986, p. 13) “Exposure to ETS is a cause of lung cancer among women who have never smoked.” (2001, p. 350)	“The evidence is sufficient to infer a causal relationship between secondhand smoke exposure and lung cancer among lifetime nonsmokers. This conclusion extends to all secondhand smoke exposure, regardless of location.” (p. 15)	—
Nasal sinus cavity and nasopharyngeal carcinoma	—	—	“The evidence is suggestive but not sufficient to infer a causal relationship between secondhand smoke exposure and a risk of nasal sinus cancer among nonsmokers.” (p. 15) “The evidence is inadequate to infer the presence or absence of a causal relationship between secondhand smoke exposure and a risk of nasopharyngeal carcinoma among nonsmokers.” (p. 15)	—

*Note:* **ETS** = environmental tobacco smoke.

<sup>a</sup>General conclusion on cancers other than lung: “The associations between cancers, other than cancer of the lung, and involuntary smoking require further investigation before a determination can be made about the relationship of involuntary smoking to these cancers.” (1986, p. 14)

**Table 4.7 Conclusions from Surgeon General’s report on exposure to secondhand smoke and cardiovascular diseases<sup>a</sup>**

Disease	First mention and finding(s) in a Surgeon General’s report (year)	Highest level conclusion(s) from subsequent Surgeon General’s reports before 2006 (year)	Conclusion(s) from the 2006 Surgeon General’s report	Additional or updated conclusion(s) from the 2014 Surgeon General’s report
Atherosclerosis/ subclinical vascular disease	—	—	“Studies of secondhand smoke and subclinical vascular disease, particularly carotid arterial wall thickening, are suggestive but not sufficient to infer a causal relationship between exposure to secondhand smoke and atherosclerosis.” (p. 15)	—
Cerebrovascular disease	—	—	“The evidence is suggestive but not sufficient to infer a causal relationship between exposure to secondhand smoke and an increased risk of stroke.” (p. 15)	“The evidence is sufficient to infer a causal relationship between exposure to secondhand smoke and increased risk of stroke.”  “The estimated increase in risk for stroke from exposure to secondhand smoke is about 20–30%.” (Chapter 8)
Coronary heart disease	“The presence of such levels” as found in cigarettes “indicates that the effect of exposure to carbon monoxide may on occasion, depending upon the length of exposure, be sufficient to be harmful to the health of an exposed person. This would be particularly significant for people who are already suffering from...coronary heart disease.” (1972, p. 7)	“Epidemiologic and other data support a causal relationship between ETS exposure from the spouse and coronary heart disease mortality among women nonsmokers.” (2001, p. 356)	“The evidence is sufficient to infer a causal relationship between exposure to secondhand smoke and increased risks of coronary heart disease morbidity and mortality among both men and women.” (p. 15)	—

*Note:* ETS = environmental tobacco smoke.

<sup>a</sup>General conclusion on cardiovascular disease: “Further studies on the relationship between involuntary smoking and cardiovascular disease are needed in order to determine whether involuntary smoking increases the risk of cardiovascular disease.” (1986, p. 14). [“The evidence is sufficient to infer that smoking is the dominant cause of chronic obstructive pulmonary disease (COPD) in men and women in the United States. Smoking causes all elements of the COPD phenotype, including emphysema and damage to the airways of the lung” (2014)].

**Table 4.8 Conclusions from Surgeon General's report on exposure to secondhand smoke and respiratory effects in children**

<b>Disease</b>	<b>First mention and finding(s) in a Surgeon General's report (year)</b>	<b>Highest level conclusion(s) from subsequent Surgeon General's reports before 2006 (year)</b>	<b>Conclusion(s) from the 2006 Surgeon General's report</b>	<b>Additional or updated conclusion(s) from the 2012/2014 Surgeon General's report</b>
Asthma	"The limited existing data yield conflicting results concerning the relationship between passive smoke exposure and pulmonary function changes in patients with asthma." (1984, p. 13) <sup>a</sup>	—	"The evidence is sufficient to infer a causal relationship between parental smoking and ever having asthma among children of school age." (p. 14)  "The evidence is suggestive but not sufficient to infer a causal relationship between secondhand smoke exposure from parental smoking and the onset of childhood asthma." (p. 14)	—
Chronic respiratory symptoms (cough, phlegm, wheeze, dyspnea, etc.)	"Chronic cough and phlegm are more frequent in children whose parents smoke compared with children of nonsmokers. The implications of chronic respiratory symptoms for respiratory health as an adult are unknown and deserve further study." (1986, p. 13)	—	"The evidence is sufficient to infer a causal relationship between parental smoking and cough, phlegm, wheeze, and breathlessness among children of school age." (p. 14)  "The evidence is sufficient to infer a causal relationship between secondhand smoke exposure from parental smoking and the onset of wheeze illnesses in early childhood." (p. 14)	—

Table 4.8 Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2006 (year)	Conclusion(s) from the 2006 Surgeon General's report	Additional or updated conclusion(s) from the 2012/2014 Surgeon General's report
Influenza, pneumonia, and acute respiratory illnesses	"The children of smoking parents have an increased prevalence of reported respiratory symptoms, and have an increased frequency of bronchitis and pneumonia early in life." (1984, p. 13)	"The children of parents who smoke have an increased frequency of a variety of acute respiratory illnesses and infections, including chest illnesses before 2 years of age and physician-diagnosed bronchitis, tracheitis, and laryngitis, when compared with the children of nonsmokers." (1986, p. 13)  "The children of parents who smoke have an increased frequency of hospitalization for bronchitis and pneumonia during the first year of life when compared with the children of nonsmokers." (1986, p. 13)	"The evidence is sufficient to infer a causal relationship between secondhand smoke exposure from parental smoking and lower respiratory illnesses in infants and children." (p. 14)	—
Lung growth and pulmonary function	"The children of smoking parents appear to have measurable but small differences in tests of pulmonary function when compared with children of nonsmoking parents. The significance of this finding to the future development of lung disease is unknown." (1984, p. 13)	"The children of parents who smoke have small differences in tests of pulmonary function when compared with the children of nonsmokers. Although this decrement is insufficient to cause symptoms, the possibility that it may increase susceptibility to chronic obstructive pulmonary disease with exposure to other agents in adult life, e.g., [sic] active smoking or occupational exposures, needs investigation." (1986, p. 13)	"The evidence is sufficient to infer a causal relationship between maternal smoking during pregnancy and persistent adverse effects on lung function across childhood." (p. 14)  "The evidence is sufficient to infer a causal relationship between exposure to secondhand smoke after birth and a lower level of lung function during childhood." (p. 14)	—

**Table 4.8 Continued**

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2006 (year)	Conclusion(s) from the 2006 Surgeon General's report	Additional or updated conclusion(s) from the 2012/2014 Surgeon General's report
Middle ear disease and adenotonsillectomy	"A number of studies report that chronic middle ear effusions are more common in young children whose parents smoke than in children of nonsmoking parents." (1986, p. 14)	—	"The evidence is sufficient to infer a causal relationship between parental smoking and middle ear disease in children, including acute and recurrent otitis media and chronic middle ear effusion." (p. 14)	—
			"The evidence is suggestive but not sufficient to infer a causal relationship between parental smoking and the natural history of middle ear effusion." (p. 14)	
			"The evidence is inadequate to infer the presence or absence of a causal relationship between parental smoking and an increase in the risk of adenoidectomy or tonsillectomy among children." (p. 14)	
Atopy	—	—	"The evidence is inadequate to infer the presence or absence of a causal relationship between parental smoking and the risk of immunoglobulin E-mediated allergy in their children." (p. 14)	—

Note: **TB** = tuberculosis.

<sup>a</sup>General conclusion without specification of outcome in children or adults.

**Table 4.9** Conclusions from Surgeon General’s report on exposure to secondhand smoke and respiratory effects in adults

Disease	First mention and finding(s) in a Surgeon General’s report (year)	Highest level conclusion(s) from subsequent Surgeon General’s reports before 2006 (year)	Conclusion(s) from the 2006 Surgeon General’s report	Additional or updated conclusion(s) from the 2012/2014 Surgeon General’s report
Asthma	“The limited existing data yield conflicting results concerning the relationship between passive smoke exposure and pulmonary function changes in patients with asthma.” (1984, p. 13) <sup>a</sup>	—	“The evidence is suggestive but not sufficient to infer a causal relationship between secondhand smoke exposure and adult-onset asthma.” (p. 16)  “The evidence is suggestive but not sufficient to infer a causal relationship between secondhand smoke exposure and a worsening of asthma control.” (p. 16)	—
Chronic respiratory symptoms (cough, phlegm, wheeze, dyspnea, etc.)	—	—	“The evidence is suggestive but not sufficient to infer a causal relationship between secondhand smoke exposure and acute respiratory symptoms including cough, wheeze, chest tightness, and difficulty breathing among persons with asthma.” (p. 15)  “The evidence is suggestive but not sufficient to infer a causal relationship between secondhand smoke exposure and acute respiratory symptoms including cough, wheeze, chest tightness, and difficulty breathing among healthy persons.” (p. 15)  “The evidence is suggestive but not sufficient to infer a causal relationship between secondhand smoke exposure and chronic respiratory symptoms.” (p. 15)	—

Table 4.9 Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2006 (year)	Conclusion(s) from the 2006 Surgeon General's report	Additional or updated conclusion(s) from the 2012/2014 Surgeon General's report
Chronic obstructive pulmonary disease (Formerly designated as chronic bronchitis; emphysema; chronic obstructive lung disease; chronic obstructive bronchopulmonary disease)	"Healthy adults exposed to environmental tobacco smoke may have small changes on pulmonary function testing, but are unlikely to experience clinically significant deficits in pulmonary function as a result of exposure to environmental tobacco smoke alone." (1986, pp. 13–14)	—	"The evidence is suggestive but not sufficient to infer a causal relationship between secondhand smoke exposure and risk for chronic obstructive pulmonary disease." (p. 16)  "The evidence is inadequate to infer the presence or absence of a causal relationship between secondhand smoke exposure and morbidity in persons with chronic obstructive pulmonary disease." (p. 16)	—
Lung function	"Other components of tobacco smoke, such as particulate matter and the oxides of nitrogen, have been shown in various concentrations to affect adversely animal pulmonary...function. The extent of the contributions of these substances to illness in humans exposed to the concentrations present in an atmosphere contaminated with tobacco smoke is not presently known." (1972, pp. 7–8)	"...some studies suggest that high levels of involuntary [tobacco] smoke exposure might produce small changes in pulmonary function in normal subjects. ... Two studies have reported differences in measures of lung function in older populations between subjects chronically exposed to involuntary smoking and those who were not. This difference was not found in a younger and possibly less exposed population." (1984, p. 13)	"The evidence is suggestive but not sufficient to infer a causal relationship between short-term secondhand smoke exposure and an acute decline in lung function in persons with asthma." (p. 16)  "The evidence is inadequate to infer the presence or absence of a causal relationship between short-term secondhand smoke exposure and an acute decline in lung function in healthy persons." (p. 16)  "The evidence is suggestive but not sufficient to infer a causal relationship between chronic secondhand smoke exposure and a small decrement in lung function in the general population." (p. 16)  "The evidence is inadequate to infer the presence or absence of a causal relationship between chronic secondhand smoke exposure and an accelerated decline in lung function." (p. 16)	—

Table 4.9 Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2006 (year)	Conclusion(s) from the 2006 Surgeon General's report	Additional or updated conclusion(s) from the 2012/2014 Surgeon General's report
Odor and irritation	<p>"An atmosphere contaminated with tobacco smoke can contribute to the discomfort of many individuals." (1972, p. 7)</p> <p>"Cigarette smoke in the air can produce an increase in both subjective and objective measures of eye irritation." (1984, p. 13)</p>	<p>"The main effects of the irritants present in ETS occur in the conjunctiva of the eyes and the mucous membranes of the nose, throat, and lower respiratory tract. These irritant effects are a frequent cause of complaints about poor air quality due to environmental tobacco smoke." (1986, p. 252)</p>	<p>"The evidence is sufficient to infer a causal relationship between secondhand smoke exposure and odor annoyance." (p. 15)</p> <p>"The evidence is sufficient to infer a causal relationship between secondhand smoke exposure and nasal irritation." (p. 15)</p> <p>"The evidence is suggestive but not sufficient to conclude that persons with nasal allergies or a history of respiratory illnesses are more susceptible to developing nasal irritation from secondhand smoke exposure." (p. 15)</p>	—
Tuberculosis	—	—	—	<p>"The evidence is inadequate to infer the presence or absence of a causal relationship between exposure to secondhand smoke and the risk of tuberculosis infection." (2014, Chapter 7)</p> <p>"The evidence is inadequate to infer the presence or absence of a causal relationship between exposure to secondhand smoke and the risk of tuberculosis disease." (2014, Chapter 7)</p>

Note: ETS = environmental tobacco smoke.

<sup>a</sup>General conclusion without specification of outcome in children or adults.

**Table 4.10 Conclusions from Surgeon General's report on exposure to secondhand smoke and reproductive and developmental effects**

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2006 (year)	Conclusion(s) from the 2006 Surgeon General's report	Additional or updated conclusion(s) from the 2012/2014 Surgeon General's report
Child physical and cognitive development	—	—	<p>“The evidence is inadequate to infer the presence or absence of a causal relationship between exposure to secondhand smoke and cognitive functioning among children.” (p. 13)</p> <p>“The evidence is inadequate to infer the presence or absence of a causal relationship between exposure to secondhand smoke and behavioral problems among children.” (p. 13)</p> <p>“The evidence is inadequate to infer the presence or absence of a causal relationship between exposure to secondhand smoke and children’s height/growth.” (p. 13)</p>	—
Congenital malformations	—	—	<p>“The evidence is inadequate to infer the presence or absence of a causal relationship between exposure to secondhand smoke and congenital malformations.” (p. 13)</p>	—
Fertility	—	—	<p>“The evidence is inadequate to infer the presence or absence of a causal relationship between maternal exposure to secondhand smoke and female fertility or fecundability. No data were found on paternal exposure to secondhand smoke and male fertility or fecundability.” (p. 13)</p>	—
Fetal death, stillbirths, and infant mortality	<p>“Studies of ETS exposure and the risks for delay in conception, spontaneous abortion, and perinatal mortality are few, and the results are inconsistent.” (2001, p. 372)</p>	—	<p>“The evidence is inadequate to infer the presence or absence of a causal relationship between exposure to secondhand smoke and neonatal mortality.” (p. 13)</p>	—
Sudden infant death syndrome (SIDS)	—	—	<p>“The evidence is sufficient to infer a causal relationship between exposure to secondhand smoke and sudden infant death syndrome.” (p. 13)</p>	—

Table 4.10 Continued

Disease	First mention and finding(s) in a Surgeon General's report (year)	Highest level conclusion(s) from subsequent Surgeon General's reports before 2006 (year)	Conclusion(s) from the 2006 Surgeon General's report	Additional or updated conclusion(s) from the 2012/2014 Surgeon General's report
Infant birth weight	“...maternal exposure to ETS appears to be causally associated with detrimental effects on fetal growth.” (2001, p. 364)	—	“The evidence is sufficient to infer a causal relationship between maternal exposure to secondhand smoke during pregnancy and a small reduction in birth weight.” (p. 13)	—
Pregnancy complications	—	—	“The evidence is inadequate to infer the presence or absence of a causal relationship between maternal exposure to secondhand smoke during pregnancy and spontaneous abortion.” (p. 13)  “The evidence is suggestive but not sufficient to infer a causal relationship between maternal exposure to secondhand smoke during pregnancy and preterm delivery.” (p. 13)	—

Note: ETS = environmental tobacco smoke.

**Table 4.11 Conclusions reached by the Advisory Committee to the Surgeon General in 1964**

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**Lung Cancer**

“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.

The risk of developing lung cancer increases with duration of smoking and the number of cigarettes smoked per day, and is diminished by discontinuing smoking.

The risk of developing cancer of the lung for the combined group of pipe smokers, cigar smokers, and pipe and cigar smokers, is greater than for nonsmokers, but much less than for cigarette smokers. The data are insufficient to warrant a conclusion for each group individually.” (p. 196)

**Oral Cancer**

“The causal relationship of the smoking of pipes to the development of cancer of the lip appears to be established. Although there are suggestions of relationships between cancer of other specific sites of the oral cavity and the several forms of tobacco use, their causal implications cannot at present be stated.” (pp. 204–5)

**Cancer of the Larynx**

“Evaluation of the evidence leads to the judgment that cigarette smoking is a significant factor in the causation of laryngeal cancer in the male.” (p. 212)

**Cancer of the Esophagus**

“The evidence on the tobacco-esophageal cancer relationship supports the belief that an association exists. However, the data are not adequate to decide whether the relationship is causal.” (p. 218)

**Cancer of the Urinary Bladder**

“Available data suggest an association between cigarette smoking and urinary bladder cancer in the male but are not sufficient to support a judgment on the causal significance of this association.” (p. 225)

**Stomach Cancer**

“No relationship has been established between tobacco use and stomach cancer.” (p. 229)

**Non-Neoplastic Respiratory Diseases, Particularly Chronic Bronchitis and Pulmonary Emphysema**

“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.

A relationship exists between pulmonary emphysema and cigarette smoking but it has not been established that the relationship is causal. The smoking of cigarettes is associated with an increased risk of dying from pulmonary emphysema.

For the bulk of the population of the United States, the importance of cigarette smoking as a cause of chronic bronchopulmonary disease is much greater than that of atmospheric pollution or occupational exposures.

Cough, sputum production, or the two combined are consistently more frequent among cigarette smokers than among non-smokers. Cigarette smoking is associated with a reduction in ventilatory function. Among males, cigarette smokers have a greater prevalence of breathlessness than non-smokers.

Cigarette smoking does not appear to cause asthma.

Although death certification shows that cigarette smokers have a moderately increased risk of death from influenza and pneumonia, an association of cigarette smoking and infectious diseases is not otherwise substantiated.” (p. 302)

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**Table 4.11 Continued**

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**Cardiovascular Disease**

“Male cigarette smokers have a higher death rate from coronary artery disease than non-smoking males, but it is not clear that the association has causal significance.” (p. 327)

**Peptic Ulcer**

“Epidemiological studies indicate an association between cigarette smoking and peptic ulcer which is greater for gastric than for duodenal ulcer.” (p. 340)

**Tobacco Amblyopia**

“Tobacco amblyopia (dimness of vision unexplained by an organic lesion) has been related to pipe and cigar smoking by clinical impressions. The association has not been substantiated by epidemiological or experimental studies.” (p. 342)

**Cirrhosis of the Liver**

“Increased mortality of smokers from cirrhosis of the liver has been shown in the prospective studies. The data are not sufficient to support a direct or causal association.” (p. 342)

**Maternal Smoking and Infant Birth Weight**

“Women who smoke cigarettes during pregnancy tend to have babies of lower birth weight. Information is lacking on the mechanism by which this decrease in birth weight is produced. It is not known whether this decrease in birth weight has any influence on the biological fitness of the newborn.” (p. 343)

**Smoking and Accidents**

“Smoking is associated with accidental deaths from fires in the home. No conclusive information is available on the effects of smoking on traffic accidents.” (p. 345)

**Morphological Constitution of Smokers**

“The available evidence suggests the existence of some morphological differences between smokers and non-smokers, but is too meager to permit a conclusion.” (p. 387)

“The overwhelming evidence points to the conclusion that smoking—its beginning, habituation, and occasional discontinuation—is to a large extent psychologically and socially determined. This does not rule out physiological factors, especially in respect to habituation, nor the existence of predisposing constitutional or hereditary factors.” (p. 377)

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Source: U.S. Department of Health, Education, and Welfare 1964.

**Table 4.12 Conclusions from previous Surgeon General's reports related to smoking and all-cause mortality**

Year	Conclusion
1964	"Cigarette smoking is associated with a 70 percent increase in the age specific death rates of males, and to a lesser extent with increased death rates of females. The total number of excess deaths causally related to cigarette smoking in the U.S. population cannot be accurately estimated. In view of the continuing and mounting evidence from many sources, it is the judgment of the Committee that cigarette smoking contributes substantially to mortality from certain specific diseases and to the overall death rate." (p. 31)
1967	<p>"1. Cigarette smokers have substantially higher rates of death and disability than their nonsmoking counterparts in the population. This means that cigarette smokers tend to die at earlier ages and experience more days of disability than comparable nonsmokers.</p> <p>2. A substantial portion of earlier deaths and excess disability would not have occurred if those affected had never smoked." (p. 3)</p>
1968 (supplement to 1967)	"Previous findings reported in 1967 indicate that cigarette smoking is associated with an increase in overall mortality and morbidity and leads to a substantial excess of deaths in those people who smoke." (p. 3)
1978	<p>"1. Overall mortality rates for cigarette smokers are about 70 percent higher than those for nonsmokers.</p> <p>2. Overall mortality risk increases with the amount smoked. For the two-pack-a-day cigarette smoker, the risk of premature death is approximately twice that of the nonsmoker.</p> <p>3. Overall mortality ratios of smokers compared to nonsmokers are highest at earlier ages and decline with increasing age. For cigarette smokers, the risk of premature death is twice that of nonsmokers at age 40.</p> <p>4. Overall mortality ratios are higher for those who begin smoking at a young age compared to those who begin later. For those who begin smoking before the age of 15, the risk of premature death is about 86 percent higher than that for nonsmokers." (pp. 44–5)</p>
1979	<p>"1. The overall mortality ratio for all male current cigarette smokers, irrespective of quantity, is about 1.7 (70 percent excess) compared to nonsmokers.</p> <p>2. Mortality ratios increase with amount smoked. The two-pack-a-day male smoker has a mortality ratio of 2.0 compared to nonsmokers.</p> <p>3. Overall mortality ratios are directly proportional to the duration of cigarette smoking. The longer one smokes, the greater the risk of dying.</p> <p>4. Overall mortality ratios are higher for those who initiated their cigarette smoking at younger ages compared to those who began smoking later.</p> <p>5. Overall mortality ratios are higher among cigarette smokers who inhale than among those who do not." (p. 1-10)</p>
1980	<p>"1. The mortality ratio for women who smoke cigarettes is about 1.2 or 1.3.</p> <p>2. Mortality ratios for women increase with the amount smoked. In the largest prospective study the mortality ratio was 1.63 for the two-pack-a-day smoker as compared to nonsmokers.</p> <p>3. Mortality ratios are generally proportional to the duration of cigarette smoking; the longer a woman smokes, the greater the excess risk of dying.</p> <p>4. Mortality ratios tend to be higher for those women who begin smoking at a young age as compared to those who begin smoking later." (p. 6)</p>
1989	"Smoking is responsible for more than one of every six deaths in the United States. Smoking remains the single most important preventable cause of death in our society." (p. 11)

**Table 4.12** Continued

Year	Conclusion
2001	“1. Cigarette smoking plays a major role in the mortality of U.S. women. 2. The excess risk for death from all causes among current smokers compared with persons who have never smoked increases with both the number of years of smoking and the number of cigarettes smoked per day.” (p. 12)
2004	“There have been more than 12 million premature deaths attributable to smoking since the first published Surgeon General’s report on smoking and health in 1964. Smoking remains the leading preventable cause of premature death in the United States.” (p. 30)
2006 <sup>a</sup>	“Secondhand smoke causes premature death and disease in children and in adults who do not smoke.” (p. 11)

<sup>a</sup>Exposure to secondhand smoke.

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