The Surgeon Generals' Reports and Respiratory Diseases From 1964 to 2014

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The 1964 report, *Smoking and Health: Report of the Advisory Committee of the Surgeon General of the Public Health Service* (generally referred to as the Surgeon General's Report), has had lasting implications for clinical care and public health through its findings and methods (1). It is best known for the conclusion that smoking was a cause of lung cancer (in men), but it had other notable findings that anticipated conclusions of future reports (Table 1). Over time, many additional cancers were causally linked to smoking, as were cardiovascular diseases and diverse adverse effects of smoking on reproduction. Regarding respiratory diseases, the report found a causal association of smoking with "chronic bronchitis" and noted increased risk of death from influenza and pneumonia in smokers. Later reports concluded that passive smoking, a topic first considered in the 1972 report (2), caused diverse adverse respiratory effects for children and adults (Table 2).

Beyond these findings for smoking and health, the methodology used in the 1964 report was groundbreaking, becoming the foundation for evidence review, integration, and causal inference in subsequent reports of the Surgeon General (3). The approach of the 1964 report has also been widely used for evidence evaluation regarding causation in other domains (4). As described in the 2014 Surgeon General's report, the committee developed and applied a transparent methodology for finding and evaluating research results and interpreted

the evidence within a framework for causal inference that is still widely referred to as "the Surgeon General's guidelines" or "criteria" (5). Surgeon General Luther Terry was not directly involved in the Advisory Committee's discussions, and the Advisory Committee was composed of individuals without strong prior opinions on smoking and health. The report represents an early application of what is now referred to as systematic review; a similar approach had been taken in the 1959 evaluation of the evidence on smoking and lung cancer by Cornfield and colleagues (6). Although systematic review had long played a role in the synthesis of scientific evidence in other fields, the 1964 report is one of the first instances of its use for integrating findings of biomedical research.

The reports of the Surgeon General on tobacco and health (including smokeless tobacco) have now been published for 50 years, a total of 33 reports. Over the 5 decades, the topics have spanned a broad range, reflecting the trends of emerging scientific evidence and the directions of tobacco control. The reports have captured the ever-expanding evidence base on tobacco and health and reached scientific conclusions that have often had powerful policy implications. With the inference of causation, prevention is possible through removal of the causal agent. As stated in the 2004 report: "A causal conclusion conveys the inference that changing a given factor will actually reduce population's burden of disease..." (3, p. 19). For example, the

initial finding that smoking caused lung cancer motivated follow-up action by the U.S. Congress, which passed the Federal Cigarette Labeling and Advertising Act of 1965. Over the 50 years since the first report, the possibility of prevention has become reality, as smoking prevalence declined and lung cancer incidence and mortality rates eventually followed. Falling smoking prevalence is also a major contributor to the dramatic decline in mortality from cardiovascular disease (7).

This paper addresses how the Surgeon Generals' reports have covered the topic of smoking and respiratory diseases over the 50 years from the first to the 2014 anniversary report. They have played a critical role in capturing the effects of smoking on the lung, serving as foundational documents for research and disease control. The reports have been the principal locus for synthesizing the evidence on smoking and respiratory diseases, much involving research performed by members of the American Thoracic Society. The conclusions of the reports chart the evolution of understanding of smoking and risk for respiratory diseases, drawing on findings of the reports over the last 50 years (Tables 2, E1, and E2).

Background

A half-century in the past at the time of the 1964 report, patterns of respiratory disease

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Table 1. Conclusions reached by the Advisory Committee to the Surgeon General in 1964**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 (Chapter 9, 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 (Chapter 9, pp. 204–205).

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 men (Chapter 9, 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 (Chapter 9, p. 218).

Cancer of the urinary bladder

Available data suggest an association between cigarette smoking and urinary bladder cancer in men but are not sufficient to support a judgment on the causal significance of this association (Chapter 9, p. 225).

Stomach cancer

No relationship has been established between tobacco use and stomach cancer (Chapter 9, p. 229).

Nonneoplastic 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 occupational exposures.

Cough, sputum production, or the two combined are consistently more frequent among cigarette smokers than among nonsmokers.

Cigarette smoking is associated with a reduction in ventilator function. Among men, cigarette smokers have a greater prevalence of breathlessness than nonsmokers.

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 otherwise substantiated (Chapter 10, p. 302).

Cardiovascular disease

Male cigarette smokers have a higher death rate from coronary artery disease than nonsmoking men, but it is not clear that the association has causal significance. (Chapter 11, p. 327).

Peptic ulcer

Epidemiological studies indicate an association between cigarette smoking and peptic ulcer, which is greater for gastric than for duodenal ulcer (Chapter 12, 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 (Chapter 12, 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 (Chapter 12, 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 (Chapter 12, 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 (Chapter 12, p. 345).

Morphological constitution of smokers

The available evidence suggests the existence of some morphological differences between smokers and nonsmokers but is too meager to permit a conclusion (Chapter 15, 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, or the existence of predisposing constitutional or hereditary factors (Chapter 14, p. 377).

Source: U.S. Department of Health Education and Welfare 1964 (1).

Table 2. Surgeon General's report conclusions on active and passive smoking and various respiratory health effects

	Strength of Causal Inferences from Latest Available Evidence			
	Sufficient	Suggestive but not sufficient	Inadequate	Suggestive of no causal relationship
Active smoking				
Asthma				
Exacerbation among adults	X			
Exacerbation among children		X		
Incidence among adults		X		
Incidence among children		X		
COPD	X			
Chronic respiratory symptoms (including cough, phlegm, wheeze, and dyspnea)	X			
Acute respiratory illnesses				
Acute respiratory illnesses including pneumonia in persons without underlying smoking-related chronic obstructive lung disease	X			
Acute respiratory infections among persons with preexisting COPD		X		
Idiopathic pulmonary fibrosis		X		
Lung cancer	X			
Lung growth and pulmonary function				
Reduced lung function and impaired lung growth during childhood and adolescence	X			
Early onset of lung function decline during late adolescence and early adulthood	X			
Premature onset and accelerated age-related lung function decline	X			
TB				
Increased risk of TB disease	X			
Mortality due to TB	X			
Recurrent TB		X		
TB infection			X	
Passive smoking				
Asthma				
Ever having asthma among children of school age	X			
Childhood-onset asthma		X		
Adult-onset asthma		X		
Worsening of asthma control among adults		X		
Chronic respiratory symptoms				
Cough, phlegm, wheeze, and breathlessness among children of school age due to parental smoking	X			
Onset of wheeze illnesses in early childhood due to parental smoking	X			
Cough, wheeze, chest tightness, and difficulty breathing among healthy adults and persons with asthma		X		
COPD		X		
Influenza, pneumonia, infections, and acute respiratory illnesses				
Lower respiratory illnesses in infants and children due to parental smoking	X			
Increased frequency of lower respiratory tract illnesses during infancy due to maternal smoking during pregnancy		X		
Lung cancer	X			
Lung growth and pulmonary function in children				
Persistent adverse effects on lung function across childhood due to maternal smoking during pregnancy	X			

(Continued)

Table 2. (CONTINUED)

	Strength of Causal Inferences from Latest Available Evidence			
	Sufficient	Suggestive but not sufficient	Inadequate	Suggestive of no causal relationship
Lower level of lung function during childhood due to secondhand smoke exposure after birth	X			
Lung function in adults				
Acute decline in lung function among persons with asthma (short-term SHS exposure)		X		
Acute decline in lung function among healthy persons (short-term SHS exposure)			X	
Small decrement in lung function in general population (chronic SHS exposure)		X		
Accelerated decline in lung function (chronic SHS exposure)			X	
Middle ear disease and adenotonsillectomy in childhood				
Acute and recurrent otitis media and chronic middle ear effusion	X			
Natural history of middle ear effusion		X		
Adenoidectomy or tonsillectomy			X	
Atopy (IgE-mediated allergy in children)			X	
Odor and irritation				
Odor annoyance	X			
Nasal irritation	X			
Increased susceptibility to nasal irritation among persons with nasal allergies or history of respiratory illnesses		X		

Definition of abbreviations: SHS = secondhand smoke; TB = tuberculosis.

occurrence were quite different from now. Tuberculosis incidence and mortality had declined greatly from the start of the 20th century, as had infectious disease mortality in general (Figure 1). The epidemic of chronic obstructive pulmonary disease (COPD) (a term not used at the time) mortality was emerging, and symptomatic chronic bronchitis was common (8). Asthma was a well-recognized disease of children and adults, but its etiology was poorly understood and its prevalence was far lower than now. Idiopathic pulmonary fibrosis (IPF) was a recognized problem, although little was known about its pathogenesis. The pneumoconioses, particularly coal workers' pneumoconiosis, asbestosis, and silicosis, were far too common and well-described clinical entities, and seminal epidemiological studies on exposures and risks were in progress. Sparked by such disasters as the London Fog of 1952 (9) and Donora, Pennsylvania in 1948 (10), epidemiological and toxicological research on air pollution had been initiated.

Substantial attention of researchers was focused on smoking and obstructive airways disease. By 1964, cigarette consumption

had approached its peak; average per capita consumption for all persons 18 years of age and older, including smokers and nonsmokers, was more than 4,000 cigarettes per year or 200 packs (Figure 2). Research on smoking and respiratory health had also been initiated beginning in the 1950s. As late as the 1940s, there was little recognition of the role of smoking as a cause of nonmalignant respiratory diseases.

By 1964, the basic tools for carrying out population studies on smoking and respiratory diseases had been developed (8, 11). Standardized respiratory symptoms questionnaires were in use, and methods for carrying out lung function testing in field settings were available (8). These methods had been used in epidemiological studies of exposed workers and in the general population to address adverse effects of smoking and air pollution, primarily in cross-sectional designs, but cohort studies had also been implemented (e.g., Fletcher 1978 [12]). Consequently, by the time of the 1964 report, the chapter on "Non-Neoplastic Respiratory Diseases, Particularly Chronic Bronchitis and Pulmonary Emphysema" was lengthy (55 pages) and covered disease trends,

mechanisms, and toxicology, and epidemiological studies (1). In addition to covering what would now be referred to as COPD, it considered smoking and the upper airway, asthma, and infectious diseases. Subsequent reports returned to these topics as well as IPF, childhood asthma, and occupational lung disease. Beginning in 1982, passive smoking was considered as a risk factor for lung cancer, and both cancer and noncancer effects were examined in subsequent reports.

The Surgeon Generals' Reports and Respiratory Diseases

Lung Cancer

The 1964 conclusion on smoking and lung cancer was only the first of many conclusions in the Surgeon Generals' reports on the topic. The 1964 report was comprehensive in its review of the evidence available at the time, including knowledge of the constituents of tobacco smoke, experimental and observational studies, and coherence of disease patterns with a causal role of smoking. The report gave careful

Figure 1. Mortality rates for major diseases in the United States, 1900-2005. COPD = chronic obstructive pulmonary disease; CVD = cardiovascular disease. Reprinted from Reference 5.

consideration to alternative hypotheses, specifically to the “constitutional hypothesis” that some people were predisposed by some underlying “constitutional factor” to both smoke and to develop lung cancer. This hypothesis, attributed to R. A. Fisher (13), was set aside by the Advisory Committee, although today’s research on the genetic basis of lung cancer supplies some plausibility for it. The causal conclusion was for smoking among men only, as the evidence was quite limited for women at the time.

The Surgeon Generals’ reports are dynamic and continually updated as the evidence evolves. Subsequent reports returned repeatedly to the topic of smoking and lung cancer. By 1967, smoking was noted to be a cause of lung cancer in women: “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” (14, p. 36). Later conclusions addressed the dose-response relationships of risk with amount smoked and the decline in risk after quitting, and mechanisms of carcinogenesis were covered in multiple reports, most recently the 2010 report (15). That report covered the emerging information on genetic determinants of risk for cancer in smokers. The reports have now causally linked all major types of lung cancer to smoking and

commented on the relationship between the rise of adenocarcinoma and changes in cigarette design and the topography of smoking. The reports have also tracked the changing risks of smoking in men and women, most recently finding that risks of lung cancer are now the same in men and women who smoke cigarettes; 50 years after the first report, sex equality in smoking has, unfortunately, been achieved (5).

As the evidence on smoking and cancer emerged, the tobacco industry responded, initially by bringing more filter-tip cigarettes into the marketplace and then with cigarettes with reduced yields of machine-measured tar and nicotine in comparison with then conventional products. These cigarettes were marketed with labels such as “mild” and “light,” implicitly providing a message of reduced risk to health. This important topic was addressed in the 1981 report (16) and far more comprehensively in the 2004 report (3), which found no reduction in risk for lung cancer or other smoking-caused diseases associated with smoking such products. The 2004 report of the Surgeon General, along with Monograph 13 in the series of tobacco control monographs published by the National Cancer Institute (17), were critical in the 2006 decision by Judge Gladys Kessler that the tobacco industry was guilty of fraud under the Racketeer Influenced and Corrupt Organizations Act (18). The

2004 report had as its third major conclusion: “Smoking cigarettes with lower machine-measured yields of tar and nicotine provides no clear benefit to health” (18, p. 25). As for lung cancer and passive smoking (*see below*), the findings of the reports played an important role in this landmark litigation as well as in the litigation filed by the states to recover expenditures for healthcare costs by Medicaid.

COPD

At the time of the 1964 report, the clinical components of the COPD phenotype had been well documented, including chronic respiratory symptoms and loss of lung function level, and the lungs of affected people were known to have airway inflammation and thickening and emphysema. The terms “chronic bronchitis” and “emphysema” were used by clinicians, with labeling differing between the United States and the UK. Later such names as “chronic nonspecific lung disease” and “chronic obstructive lung disease,” the title of the 1984 report, were used (8, 19).

The 1984 report was one of series of reports published during the 1980s that focused on specific diseases caused by smoking (19). The 10 chapters of the report covered all aspects of COPD, including pathology, pathogenesis, and epidemiology. A 168-page chapter addressed smoking and measures of COPD morbidity, whereas

Figure 2. Adults aged ≥ 18 years per capita cigarette consumption and major smoking and health events, United States, 1900–2012. FDA = U.S. Food and Drug Administration. Reprinted from Reference 5.

a subsequent 34-page chapter addressed smoking and COPD mortality. Not surprisingly, in a clear overall conclusion, the report stated: “Cigarette smoking is the major cause of chronic obstructive lung disease in the United States for both men and women” (19, p. 8). This conclusion was echoed in the 2004 report, which also found little evidence that cigarette tar yield, measured by a machine, was associated with respiratory symptoms, lung function decline, or COPD mortality (3). The 2010 report addressed the mechanisms by which smoking causes COPD, exploring oxidative injury, protease–antiprotease imbalance, and genetic determinants of risk for COPD in smokers (15). The 1990 and 2010 reports emphasized the critical role of smoking cessation in arresting the processes of lung injury leading to the development of COPD.

Respiratory Infections

The 1964 report considered the risk of respiratory infections, particularly upper

respiratory illnesses and tuberculosis, in relationship to smoking. The evidence for upper respiratory illnesses was limited, and a positive association with tuberculosis was attributed to confounding by alcohol. The chapter on smoking and mortality noted the findings of the cohort studies, which showed about 50% higher mortality from influenza and pneumonia among smokers compared with nonsmokers. The data, coming from seven cohort studies, were considered as too limited for further evaluation. Further data from cohort studies confirmed the association of smoking with increased mortality from pneumonia and influenza, a common cause of death among older persons (20, 21). The 2014 report was the first to cover the effects of smoking on the immune system in depth; it showed diverse effects of smoking on immune responses, some diminishing function and others activating function (5). The review strengthened the biological basis for earlier conclusions related to smoking and

acute respiratory illnesses, including pneumonia.

The 1964 report had set aside the increased tuberculosis mortality as reflecting confounding. Over the last 2 decades, however, substantial epidemiological evidence from both case-control and cohort studies identified smoking as strongly associated with tuberculosis (22–24). These studies took into account the potential for confounding by alcohol and other risk factors for tuberculosis. Additionally, further evidence on smoking and immune responses strengthened the plausibility of a causal association of smoking with tuberculosis. The 2014 report considered the potential for smoking to increase risk for infection with *Mycobacterium tuberculosis*, to increase risk for incident tuberculosis disease, to increase risk for recurrence of tuberculosis, and to increase risk for death from tuberculosis. The evidence was found to be sufficient to infer that smoking increases risk for incident

tuberculosis. Epidemiological observations suggest that smoking is a major contributor to the burden of tuberculosis in some low-income countries (25–28).

Asthma

The 1964 report briefly considered smoking and asthma, opining that "...it is clear that cigarette smoking is of no importance as a cause of asthma" (1, p. 276). Subsequently, the 2012 report had concluded that smoking among youth causes severe wheezing that may be diagnosed as asthma. Since 1964, smoking and asthma has been investigated in both cohort and case-control studies, and the relationship between smoking and nonspecific airways reactivity has been examined in observational and experimental research. Epidemiological studies have documented increased frequency of wheezing, along with other respiratory symptoms, in smokers compared with nonsmokers. Active smoking and asthma was considered in the 2004 reports and again in the 2014 report, culminating with the 2014 conclusion that active smoking exacerbates asthma in adults.

Idiopathic Pulmonary Fibrosis

This topic was first addressed in the 2004 report. By then, there had been substantial research on the pathogenesis of IPF and recognition of the critical role of inflammation, although understanding of triggers was lacking. Smoking was considered a plausible risk factor, and by 2004 findings of four case-control studies had been published. Although three of the four showed significant associations of smoking with risk for IPF, the evidence was considered to be "inadequate." The topic was covered again in the 2014 report. Additional epidemiological studies continued to show an association of smoking with increased risk for IPF. The new studies were sufficient to raise the classification of the evidence to "suggestive" of a causal association.

Passive Smoking

The topic of passive smoking was first considered in the 1972 report in a chapter entitled: "Public Exposure to Air Pollution from Tobacco Smoke" (2). That report highlighted the high levels of pollution indoors that could be generated by smoking; the available literature covered a period when smoking was the social norm and smoking was often quite heavy in social settings.

The 1979, 1982, and 1984 reports had chapters on the topic and addressed the emerging information on the health consequences of secondhand smoke. The first reports on secondhand smoke exposure and lung cancer risk in never smokers were published in 1981 (29, 30), and the evidence on this association was considered insufficient in the 1982 report, which focused on cancer (31). The 1984 report covered the contribution of smoking to indoor air pollution and the elevation of cotinine levels in nonsmokers exposed to tobacco smoke (19). The finding that a biomarker of smoke exposure, cotinine—the major metabolite of nicotine—was present in nonsmokers supported the plausibility of secondhand smoke as a cause of disease. The chapter's conclusions highlighted the emerging evidence on parental smoking and the respiratory health of children.

The 1986 report, *The Health Consequences of Involuntary Smoking*, was released by Surgeon General Everett Koop (32). By then, research findings on lung cancer and on nonmalignant effects of exposure to secondhand smoke were mounting, as was controversy concerning the evidence. The tobacco industry, recognizing the policy implications of the demonstration of adverse effects of secondhand smoke, initiated a broad campaign to raise questions about the scientific findings and create doubt as to their validity. The 1981 paper in the *British Medical Journal* by Hirayama (29), for example, was followed by a wave of critical letters to the editor, many written by paid consultants to the tobacco industry. Across the 1980s and into the 1990s, much of the evidence on secondhand smoke and its adverse effects came from research performed by members of the American Thoracic Society and presented at the annual meeting. At the scientific sessions, the tobacco industry's presence was often obvious.

The most powerful conclusion of the report was its first: "Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers" (32, p. 7). The second addressed the adverse consequences of parental smoking for children, and the third noted that "The simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, the exposure of nonsmokers to environmental tobacco smoke" (32, p. 7). As noted by Surgeon General Koop: "The right of smokers to smoke ends where their

behavior affects the health and well-being of others...." Thus, the report provided a powerful basis for action to end the exposures of nonsmokers to secondhand smoke in public places, including workplaces, and in homes, the principal locus of exposure of children. The report fueled the drive toward smoke-free environments and hastened the denormalization of smoking. The decline of cigarette consumption accelerated in the 1980s as a result (Figure 2).

Twenty years later, the 2006 report comprehensively reviewed passive smoking and health in its 709 pages. Most importantly, Surgeon General Carmona described the evidence as "massive and conclusive" and noted the changes in social norms and reduction of exposure to secondhand smoke since the 1986 report. Regarding adverse respiratory effects of secondhand smoke, the list lengthened to cover multiple outcomes related to the respiratory health of children. The adverse effects begin with exposure during pregnancy and may have lasting impact across the full life course. For adults, the evidence was considered suggestive for secondhand smoke as a factor that exacerbates asthma. By the time of the 2014 report, studies were available on the benefits of smoking bans for people with asthma who work in venues (e.g., bars) where implementation of bans ended their exposure.

The 2006 report, like the 1986 report, was used to support the implementation of smoking bans, as it was published at a time when a number of states were implementing statewide bans. It also added coronary heart disease to the list of diseases caused by secondhand smoke. From the policy perspective, one of the overall conclusions had substantive policy implications: "The scientific evidence indicates that there is no risk-free level of exposure to secondhand smoke" (33, p. 11).

Conclusions

Over the 5 decades spanned by the 1964 and 2014 reports of the Surgeon General, enormous gains have been made in our understanding of how smoking harms respiratory health (Table 2). The conclusions have expanded from the initial findings regarding lung cancer and chronic bronchitis to cover most of the major respiratory diseases, lung function level, and the occurrence of respiratory

symptoms and infections. The initial studies addressed active smoking and readily demonstrated adverse effects of the high doses of smoke delivered to the respiratory tract of active smokers. Within 20 years of the first report, research showed that the far lower doses of secondhand smoke also adversely affected respiratory

health. With the most recent 50th anniversary report, further endpoints have now been causally linked to smoking. Notably, smoking has been identified as a cause of tuberculosis, bringing the possibility of reducing the burden of tuberculosis, still substantial at the global level, through tobacco control. Through

their authoritative findings, the reports of the Surgeon General have had policy consequences that have benefited respiratory health in the United States and worldwide. ■

Author disclosures are available with the text of this article at www.atsjournals.org.

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