

## **CHAPTER 1**

### **INTRODUCTION, OVERVIEW, AND SUMMARY AND CONCLUSIONS**

# CONTENTS

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## Introduction

Development and Organization of the 1985 Report  
Historical Perspective

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## Overview

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Summary and Conclusions of the 1985 Report

## **Introduction**

### **Development and Organization of the 1985 Report**

The 1985 Report was prepared by the Office on Smoking and Health of the U.S. Department of Health and Human Services as part of the Department's responsibility, under Public Law 91-222, to report new and current information on smoking and health to the United States Congress. The scientific content of this Report is the collective work of 100 scientists in the fields of both smoking and occupational health. Individual manuscripts were written by experts who are recognized for their understanding of specific content areas. Each chapter was subjected to an intensive peer review, whereby comments were solicited from four to six individuals knowledgeable in that particular area. After these comments were incorporated, the entire Report was submitted to distinguished experts representing a balance of opinion in occupational disease and smoking and health. Concurrent with this latter review, the manuscript was also submitted to various U.S. Public Health Service agencies for review.

Throughout the entire report compilation process the Office on Smoking and Health had the advice and consultation of four internationally known scientists. These individuals represent expertise in the fields of both smoking and occupation. They are Dr. Lester Breslow, University of California at Los Angeles, Dr. Marcus Key, University of Texas Health Science Center, Dr. Irving Selikoff, the Mount Sinai Medical Center, and Dr. Jesse Steinfeld, Medical College of Georgia. From the outset, this panel of experts was instrumental in recommending the Report content and outline, suggesting individual authors and reviewers, and providing overall guidance during each stage of the compilation process. Each also served as an overall reviewer of the completed manuscript.

The 1985 Report contains a Foreword by the Acting Assistant Secretary for Health, a Preface by the Surgeon General of the U.S. Public Health Service, and the following chapters:

- Chapter 1. Introduction, Overview, and Summary and Conclusions
- Chapter 2. Occupation and Smoking Behavior in the United States
- Chapter 3. Evaluation of Smoking-Related Cancers in the Workplace
- Chapter 4. Evaluation of Chronic Lung Disease in the Workplace
- Chapter 5. Chronic Bronchitis: Interaction of Smoking and Occupation
- Chapter 6. Asbestos-Exposed Workers
- Chapter 7. Respiratory Disease in Coal Miners
- Chapter 8. Silica-Exposed Workers

- Chapter 9. Occupational Exposures to Petrochemicals, Aromatic Amines, and Pesticides
- Chapter 10. Cotton Dust Exposure and Cigarette Smoking
- Chapter 11. Ionizing Radiation and Lung Cancer
- Chapter 12. Smoking Intervention Programs in the Workplace

### Historical Perspective

More than two centuries ago, the relationship between occupational exposure and health outcome was presented by a noted English practitioner of surgery. Dr. Percivall Pott (1733–1788), in his *Chirurgical Observations* (1775), described this first scientific observation as a “superficial, painful ragged, ill-looking sore with hard and rising edges” that appeared in chimney sweeps, who almost always began working when they were very young and small enough to fit down a chimney. This malady was appropriately tagged “chimney sweep’s cancer.” Soon after the turn of the 19th century, additional reports confirmed Dr. Pott’s observations.

Only shortly before Dr. Pott’s description was published, Dr. John Hill (1716?–1775), in his *Cautions Against the Immoderate Use of Snuff*, described an association between tobacco use and cancer. Hill reported on two case histories and observed that “snuff is able to produce . . . swellings and excrescences” in the nose, and he believed them to be cancerous.

Although Dr. Pott’s startling report and description of the deplorable use of children as chimney sweeps was published in 1775, it was not until nearly a century and a half later, in 1914, that Yamagawa and Ichikawa were able to demonstrate the carcinogenic nature of the hydrocarbons in soot and tar. Almost 20 years later, in 1933, the proximate carcinogen 3,4-benzopyrene was isolated from coal tar by Cook, Hewett, and Hieger.

Also in the 1920s and 1930s, scientists began investigating the possible association between cigarette smoking and cancer, and near the end of World War II, several scientists had noted the higher percentages of cigarette smokers among cancer patients, particularly those with lung cancer. In 1962, when the Surgeon General’s Advisory Committee on Smoking and Health began weighing the scientific evidence for its 1964 Report, the causal significance of the association of cigarette smoking and disease was evaluated by strict criteria, none of which taken alone was sufficient for a causal judgment. These criteria today form the basis for the continued judgment that cigarette smoking is causally related to a number of disease processes.

## Overview

Cigarette smoking is clearly the major cause of lung cancer and chronic lung disease identified for the U.S. population. The role that cigarette smoking plays in the development of cancer was extensively reviewed in 1982 Report of the Surgeon General and chronic obstructive lung disease was reviewed in 1984. However, cigarette smoking is not the only cause of lung cancer or chronic lung disease in the U.S. population. A number of occupational exposures are well established as causes of cancer and chronic lung disease, and it is reasonable to expect that ongoing investigation of workplace exposures will continue to expand our understanding of the hazards of specific exposures and increase our ability to protect U.S. workers.

This Report examines the contributions of cigarette smoking and a number of workplace exposures to lung cancer and chronic lung disease among occupations in which specific hazardous exposures are known to occur. It is possible from the data presented to identify a causal role for both smoking and certain workplace exposures in lung cancer and disability from chronic lung disease. It is also known that the occupational hazards reviewed in this Report frequently occur on a substrate of risk and injury produced by cigarette smoking. The combination of exposures may influence the nature or extent of the disease produced by the isolated exposures (interact); both may act to produce the same disease, or may produce separate injuries to the lung that in combination result in more severe disability than would be expected from the isolated injuries. In addition, the worksite may represent a setting in which a substantial number of workers begin to smoke, and may provide an environment that either supports or discourages the efforts of individual workers to stop smoking. The ability to alter the adverse health outcomes of workers exposed to occupational hazards requires both an understanding of the disease risks that result from individual and combined exposure and a knowledge of how changes in the worksite can alter the pattern of disease occurrence.

Many of the major improvements in public health during the last century and the first part of this century were produced through the control of infectious diseases. The key to this success frequently was the identification of the causal agent, with the subsequent elimination of exposure to the agent or immunization against the agent. The criteria for establishing the causality of an infectious agent were expressed by Robert Koch in 1877 and are commonly referred to as Koch's postulates. They are the following:

1. The agent must be shown to be present in every case of the disease by isolation in pure culture.
2. The agent must not be found in cases of other disease.

3. Once isolated, the agent must be capable of reproducing the disease in animal experiments.
4. The agent must be recovered from the experimental disease produced.

These postulates served well in identifying the causal agents in acute infectious processes; frequently their identification was a critical part of their successful control.

The major diseases responsible for death and disability in the latter half of the 20th century are chronic heart and lung disease and cancer. These diseases, which now account for over half of all deaths in the United States annually, are commonly the result of chronic exposures to noninfectious occupational and lifestyle influences, may be caused by a number of agents acting independently, and may also result from more than one agent contributing to the disease process in any given individual. For these reasons, Koch's postulates have little relevance for establishing causality in lifestyle and occupational exposures, and new criteria for causality have been developed. These criteria rely heavily on epidemiologic data and include an examination of the consistency, strength, specificity, coherence, and temporal relationship of the association between the agent and the disease as well as the evidence of the biologic mechanisms by which the agent produced the disease.

The multifactorial etiology and chronic exposures that characterize cancer and chronic lung disease also have implications for control of these diseases in the worksite. One of the important public health achievements of this century has been the identification of hazardous agents in the workplace, with subsequent reductions in these exposures through changes in environmental levels of the agent, modification of work practices, and alteration of manufacturing practices. These changes were the result of regulation and voluntary agreement, and they reflect the action and concern of labor, management, Government, and the insurance industry. The result, in some industries, has been a dramatic reduction in the exposure to hazardous agents in the worksite and in the disease that would have been produced by these exposures.

As this Report clearly documents, however, cigarette smoking may alter the amount of disease or level of disability produced by hazardous occupational exposures. For cancer, this alteration may come in the form of adding an additional number of cancer cases, or of the combined exposure synergistically increasing the number of cancers. On an individual level, our understanding of the process of carcinogenesis suggests that both agents may contribute to individual cancer rather than some cases being caused exclusively by an occupation exposure and other cases being caused exclusively by cigarette smoking.

For lung disease, the combination of cigarette smoking and exposure to a hazardous workplace agent may combine to produce similar injuries or may produce independent disease processes in the same lung that result in greater disability than with either exposure separately.

The public health importance of interaction between smoking and an occupational exposure is typified by the relationship between cigarette smoking and asbestos exposure among asbestos workers. A number of studies published in this country and abroad have demonstrated an approximately fivefold excess risk for lung cancer among nonsmoking asbestos insulation workers. Smoking in non-asbestos-exposed populations increases the lung cancer risk by approximately tenfold. However, the risk is more than fiftyfold greater if the asbestos worker also smokes. The risk in cigarette-smoking asbestos workers is greater than the sum of the risk of the independent exposures, and is approximated by multiplying the risks of the two separate exposures. Thus, the interaction of cigarette smoking and asbestos exposure is multiplicative in nature. To state this in another way, for those workers who both smoke and are exposed to asbestos, the risk of developing and dying from lung cancer is 5,000 percent greater than the risk for individuals who neither smoke nor are exposed. Among these asbestos workers, the extent of disease produced by asbestos is conditioned by the smoking habits of the asbestos-exposed population. As is also evident, attempts to control asbestos-related lung cancer can have a maximal impact only if they include successful programs to change smoking behavior as well as efforts aimed at reducing levels of asbestos dust exposure.

Elimination of the contribution made by smoking to disease and disability in the worksite is beneficial, even in the absence of synergistic interaction between smoking and workplace exposures. Even with an additive risk for an exposed population, both agents probably contribute to the cancer that develops in an individual, and removing that contribution is an important benefit to that individual. In addition, a given degree of impairment produced by an occupational agent will result in less disability in an individual without concomitant lung injury due to smoking than in a worker who has chronic obstructive lung disease due to smoking.

The focus on individuals rather than on populations when considering strategies to control occupationally related diseases also helps clarify the concept of a "safe" worksite. The same number of lung cancers may occur in a population with a high smoking prevalence and a low asbestos exposure and a population with a low smoking prevalence and a high asbestos exposure. This similarity of population risks does not suggest that the level of acceptable or "safe" dust exposure can be adjusted on the basis of the smoking

prevalence in the population. It may be reasonable to select nonsmokers for jobs in which smokers would be at much greater risk, but this approach should never be used as a justification for accepting occupational exposure levels that result in risk for those exposed. The goal should always be the elimination of as much of the disease as possible in the working population rather than the lowering of the disease rate to the population norm.

Factors in the worksite may also influence smoking initiation and smoking cessation. Chapter 2 of this Report updates the previously reported increased smoking prevalence among blue-collar workers compared with white-collar workers. It also reports two analyses that suggest the workplace may play an important role in smoking behavior. The mean age of initiation reported confirms that the majority of smokers begin smoking prior to or during high school. However, a substantial fraction also begin to smoke after high school. Little is known about the influences that may predispose individuals to become smokers at this age. One of the major life experiences occurring at the same time is entry into the workforce, particularly for blue-collar and clerical workers, and the work environment may be a major factor capable of predisposing an individual toward or away from becoming a smoker.

A second important consideration that emerges from chapter 2 is the markedly lower prevalence of successful smoking cessation among blue-collar workers compared with white-collar workers. This difference in cessation is not explained by differences in rates of initiation, and almost equal percentages of current smokers have made a serious attempt to quit and failed. This suggests that the majority of both groups of workers have attempted to become nonsmokers, but blue-collar workers have been less successful. Once again, a potential role for the workplace environment in reinforcing or inhibiting successful cessation may help to explain these differences in the prevalence of former smokers.

If a workplace is to be considered "safe," one very important criterion is the absence of exposures to agents that can cause disease. Equally important, however, is that safety should include a workplace that neither encourages initiation nor discourages cessation of cigarette smoking. As demonstrated in the final chapter of this Report, the worksite may provide a focus for the promotion of healthy behavioral change in the workforce, but at a minimum, should not be a focus that encourages behaviors that compromise a worker's health.

### **Summary and Conclusions of the 1985 Report**

The major conclusions of this Report are clear. They are the following:

**For the majority of American workers who smoke, cigarette smoking represents a greater cause of death and disability than their workplace environment.**

**In those worksites where well-established disease outcomes occur, smoking control and reduction in exposure to hazardous agents are effective, compatible, and occasionally synergistic approaches to the reduction of disease risk for the individual worker.**

Individual chapter summaries and conclusions follow.

### **Occupation and Smoking Behavior in the United States: Current Estimates and Recent Trends**

1. Among men, a substantially higher percentage of blue-collar workers than white-collar workers currently smoke cigarettes. Operatives and kindred workers have the highest rate of current smoking (approaching 50 percent), with professional, technical, and kindred workers having the lowest rates of current smoking (approximately 26 percent).
2. Among women, blue-collar versus white-collar differences are less pronounced, but still show a higher percentage of current smokers among blue-collar workers. Occupational categories with the highest rates of current smoking include craftsmen and kindred workers (approximately 45 percent current smokers) and managers and administrators (38 percent), with the lowest rate of current smoking occurring among women employed in professional, technical, and kindred occupations (26 percent).
3. Occupational differences in daily cigarette consumption are generally modest. For both men and women, the highest daily consumption of cigarettes occurs among managers and administrators and craftsmen and kindred workers.
4. Blue-collar workers (both men and women) report an earlier onset of smoking than white-collar workers. A substantial fraction of smokers report initiation of smoking at ages coincident with their entry into the workforce.
5. Blue-collar occupations have a lower percentage of former smokers than white-collar occupations; this difference is most pronounced among men. Among women, the pattern for homemakers closely parallels that of white-collar women.
6. Black workers have higher smoking rates than white workers, with black male blue-collar workers exhibiting the highest smoking rate. Black workers also have lower quit rates than white workers. In contrast, white workers of both sexes are more likely to be heavy smokers regardless of occupational category.

### **Evaluation of Smoking-Related Cancers in the Workplace**

1. Cigarette smoking and occupational exposures may interact biologically, within a given statistical model and in their public health consequences. The demonstration of an interaction at one of these levels does not always characterize the nature of the interaction at the other levels.
2. Information on smoking behaviors should be collected as part of the health screening of all workers and made a part of their permanent exposure record.
3. Examination of the smoking behavior of an exposed population should include measures of smoking prevalence, smoking dose, and duration of smoking.
4. Differences in age of onset of exposure to cigarette smoke and occupational exposures should be considered when evaluating studies of occupational exposure, particularly when the exposed population is relatively young or the exposure is of relatively recent onset.

### **Evaluation of Chronic Lung Disease in the Workplace**

1. Existing resources for monitoring the occurrence of occupational lung diseases are not comprehensive and do not include information on cigarette smoking. Other approaches, such as registries, might offer more accurate data and facilitate research related to occupational lung diseases. Because of the variability in diagnostic criteria for chronic lung disease, in studies on occupational lung diseases emphasis should be placed on measures of physiological change, roentgenographic abnormality, and other objective measures.
2. Further studies that correlate lung function with histopathology should be carried out in occupationally exposed smokers and nonsmokers.
3. The effects of cigarette smoking on the chest x ray should be clarified. In particular, the sensitivity of the ILO classification to smoking-related changes should be further evaluated in healthy populations.
4. To determine if smoking is reported with bias by occupationally exposed workers, self-reported histories should be compared with biological markers of smoking in appropriate populations.
5. Mechanisms through which specific occupational agents and cigarette smoking might interact should be systematically considered. Both laboratory and epidemiological approaches should be used to evaluate such interactions.
6. Statistical methods for evaluating interaction require further development. In particular, the biological implications of conventional modeling approaches should be explored. Further, the limitations posed by sample size for examining

independent and interactive effects should be evaluated. The consequences of misclassification by exposure estimates and of the colinearity of exposure variables should also be addressed.

7. The role of cigarette smoking in the "healthy worker effect" requires further evaluation.
8. Approaches for apportioning the impairment in a specific individual between occupational causes and cigarette smoking should be developed and validated.

### **Chronic Bronchitis: Interaction of Smoking and Occupation**

1. Chronic simple bronchitis has been associated with occupational exposures in both nonsmoking exposed workers and populations of exposed smokers in excess of rates predicted from the smoking habit alone. Among these exposures are coal, grain, silica, the welding environment, and to a lesser extent, sulfur dioxide and cement.
2. The evidence indicates that the effects of smoking and those occupational agents that cause bronchitis are frequently additive in producing symptoms of chronic cough and expectoration. Smoking has commonly been demonstrated to be the more important factor in producing these symptoms.

### **Asbestos-Exposed Workers**

1. Asbestos exposure can increase the risk of developing lung cancer in both cigarette smokers and nonsmokers. The risk in cigarette-smoking asbestos workers is greater than the sum of the risks of the independent exposures, and is approximated by multiplying the risks of the separate exposures.
2. The risk of developing lung cancer in asbestos workers increases with increasing number of cigarettes smoked per day and increasing cumulative asbestos exposure.
3. The risk of developing lung cancer declines in asbestos workers who stop smoking when compared with asbestos workers who continue to smoke. Cessation of asbestos exposure may result in a lower risk of developing lung cancer than continued exposure, but the risk of developing lung cancer appears to remain significantly elevated even 25 years after cessation of exposure.
4. Cigarette smoking and asbestos exposure appear to have an independent and additive effect on lung function decline. Nonsmoking asbestos workers have decreased total lung capacities (restrictive disease). Cigarette-smoking asbestos workers develop both restrictive lung disease and chronic obstructive lung disease (as defined by an abnormal  $FEV_1/FVC$ ), but the evidence does not suggest that cigarette-smoking asbestos

workers have a lower FEV<sub>1</sub>/FVC than would be expected from their smoking habits alone.

5. Both cigarette smoking and asbestos exposure result in an increased resistance to airflow in the small airways. In the absence of cigarette smoking, this increased resistance in the small airways does not appear to result in obstruction on standard spirometry as measured by FEV<sub>1</sub>/FVC.
6. Asbestos exposure is the predominant cause of interstitial fibrosis in populations with substantial asbestos exposure. Cigarette smokers do have a slightly higher prevalence of chest radiographs interpreted as interstitial fibrosis than nonsmokers, but neither the frequency of these changes nor the severity of the changes approach levels found in populations with substantial asbestos exposure.
7. The promotion of smoking cessation should be an intrinsic part of efforts to control asbestos-related death and disability.

### **Respiratory Disease in Coal Miners**

1. Coal dust exposure is clearly the major etiologic factor in the production of the radiologic changes of coal workers' pneumoconiosis (CWP). Cigarette smoking probably increases the prevalence of irregular opacities on the chest roentgenograms of smoking coal miners, but appears to have little effect on the prevalence of small rounded opacities or complicated CWP.
2. Increasing category of simple radiologic CWP is not associated with increasing airflow obstruction, but increasing coal dust exposure is associated with increasing airflow obstruction in both smokers and nonsmokers.
3. Since the introduction of more effective controls to reduce the level of coal dust exposure at the worksite, cigarette smoking has become the more significant contributor to reported cases of disabling airflow obstruction among coal miners.
4. Cigarette smoking and coal dust exposure appear to have an independent and additive effect on the prevalence of chronic cough and phlegm.
5. Increasing coal dust exposure is associated with a form of emphysema known as focal dust emphysema, but there is no definite evidence that extensive centrilobular emphysema occurs in the absence of cigarette smoking.
6. The majority of studies have shown that coal dust exposure is not associated with an increased risk for lung cancer.
7. Reduction in the levels of coal dust exposure is the only method available to reduce the prevalence of simple or complicated CWP. However, the prevalence of ventilatory disabilities in coal miners could be substantially reduced by reducing the prevalence of cigarette smoking, and efforts aimed at reducing

ventilatory disability should include efforts to enhance successful smoking cessation.

### **Silica-Exposed Workers**

1. Silicosis, acute silicosis, mixed-dust silicosis, silicotuberculosis, and diatomaceous earth pneumoconiosis are causally related to silica exposure as a sole or principal etiological agent.
2. Epidemiological evidence, based on both cross-sectional and prospective studies, demonstrates that silica dust is associated with chronic bronchitis and chronic airways obstruction. Silica dust and smoking are major risk factors and appear to be additive in producing chronic bronchitis and chronic airways obstruction. Most studies indicate that the smoking effect is stronger than the silica dust effect.
3. Pathological studies describe mineral dust airways disease, which is morphologically similar to the small airways lesions caused by cigarette smoking.
4. A number of studies have demonstrated an increased risk of lung cancer in workers exposed to silica, but few of these studies have adequately controlled for smoking. Therefore, while the increased standardized mortality ratios for lung cancer in these populations suggest the need for further investigation of a potential carcinogenic effect of silica exposure (particularly in a combined exposure with other possible carcinogens), the evidence does not currently establish whether silica exposure increases the risk of developing lung cancer in man.
5. Smoking control efforts should be an important concomitant of efforts to reduce the burden of silica-related illness in working populations.

### **Occupational Exposures to Petrochemicals, Aromatic Amines, and Pesticides**

1. The biotransformation of industrial toxicants can be modified at least to some extent by the constituents of tobacco smoke through enzyme induction or possibly inhibition. Both tobacco smoke and some industrial pollutants contain substances capable of initiating and promoting cancer and damaging the airways and lung parenchyma. There is, therefore, an ample biologic basis for suspecting that important interactive effects between some workplace pollutants and tobacco smoke exist.
2. In mortality studies of coke oven workers and gas workers, convincing evidence has indicated that work exposures to oven effluents are causing an excess risk of lung cancer in spite of the lack of adequate information on smoking. Other mortality studies that suggest small increases in smoking-related dis-

eases, such as pancreatic cancer in refinery workers, cannot be interpreted without more information on smoking.

3. For bladder cancer, the interactions between smoking and occupational exposure are unclear, with both additive and antagonistic interactions having been demonstrated.
4. The risk of pulmonary disability in rubber workers was increased when smoking and occupational exposure to particulates were combined. There are few empirical animal experiments that demonstrate interactive effects between cigarette smoking and various industrial chemicals for lung disease.

### **Cotton Dust Exposure and Cigarette Smoking**

1. Byssinosis prevalence and severity is increased in cotton textile workers who smoke in comparison with workers who do not smoke.
2. Cigarette smoking seems to facilitate the development of byssinosis in smokers exposed to cotton dust, perhaps by the prior induction of bronchitis. Cotton mill workers of both sexes who smoke have a consistently greater prevalence of bronchitis than nonsmokers.
3. The importance of cigarette smoking to byssinosis prevalence seems to grow with rising dust levels (a smoking-cotton dust interaction). At the highest dust levels, cigarette smoke was found to interact with cotton dust exposure to substantially increase the acute symptom prevalence.
4. Nonsmokers with byssinosis have lower preshift lung function and a greater cross-shift decline in lung function than asymptomatic workers, and those workers with bronchitis generally have lower preshift lung function than those without bronchitis. In general, smokers have lower lung function than nonsmokers among cotton workers, both in those with bronchitis and in those with byssinosis.
5. Although the average forced expiration values measured at the start of a shift are reduced among smokers, the cross-shift decline in function does not seem to be affected by smoking status.
6. The contribution of the acute byssinotic symptoms (grades 1/2 and 1) to the subsequent development of what have been termed the chronic forms (grade 3) of byssinosis (which include airways obstruction) is not well documented; however, chronic airflow obstruction has been found more frequently in cotton textile workers than in control populations, and this lung function loss appears to be additive to that caused by cigarette smoking.
7. Cotton dust exposure is significantly associated with mucous gland volume and peripheral goblet cell metaplasia in non-

smokers, a pathology consistent with bronchitis. Among cigarette smokers, the interaction of cotton textile exposure and smoking is demonstrable for goblet cell hyperplasia. Centrilobular emphysema is found only in association with cigarette smoking and pipe smoking. There is no emphysema association found with cotton dust exposure.

8. The evidence does not currently suggest an excess risk of lung cancer among cotton textile workers.

### **Ionizing Radiation and Lung Cancer**

1. There is an interaction between radon daughters and cigarette smoke exposures in the production of lung cancer in both man and animals. The nature of this interaction is not entirely clear because of the conflicting results in both epidemiological and animal studies.
2. The interaction between radon daughters and cigarette smoke exposures may consist of two parts. The first is an additive effect on the number of cancers induced by the two agents. The second is the hastening effect of the tumor promoters in cigarette smoke on the appearance of cancers induced by radiation, so that the induction-latent period is shorter among smokers than nonsmokers and the resultant cancers are distributed in time differently between smokers and nonsmokers, appearing earlier in smokers.

### **Smoking Intervention Programs in the Workplace**

1. Smoking modification and maintenance of nonsmoking status among initial quitters has the promise of being more successful in worksite programs than in clinic-based programs. Higher cessation rates in worksite programs are achieved with more intensive programs.
2. Incentives for nonsmoking appear to be associated with higher participation and better success rates. Further research is needed to specify the optimal types of incentive procedures.
3. Success of a worksite smoking program depends upon three primary factors: the characteristics of the intervention program, the characteristics of the organization in which the program is offered, and the interaction between these factors.
4. Research is needed on recruitment strategies and participation rates in worksite smoking programs and on the impact of interventions on the entire workforce of a company.
5. More investigations are needed on worksite characteristics associated with the success of occupational programs and on comprehensive programs including components such as quit-smoking contests, no-smoking policies, physician messages, and self-help materials in addition to smoking cessation clinics.

6. The implementation of broadly based health promotion efforts in the workplace should be encouraged, with smoking interventions representing a major component of the larger effort to improve health through a worksite focus.

## **CHAPTER 2**

# **OCCUPATION AND SMOKING BEHAVIOR IN THE UNITED STATES: CURRENT ESTIMATES AND RECENT TRENDS**

# CONTENTS

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Introduction

---

Patterns of Employment

---

Smoking Prevalence

---

Daily Cigarette Consumption

---

Age of Initiation

---

Quitting Behavior

---

Recent Changes in Smoking Behavior

---

Birth Cohorts

---

Race

---

Summary and Conclusions

---

Technical Addendum: National Health Interview Survey  
Estimates

---

References

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Appendices

## **Introduction**

Estimates of current smoking behavior reported in this section of the Surgeon General's Report were obtained from the 1978, 1979, and 1980 National Health Interview Surveys (NHIS). A data tape was prepared by the National Center for Health Statistics to allow linkages across surveys, thereby permitting analyses of the combined 1978-1980 NHIS (n=49,715). The majority of the analysis presented in this chapter were conducted on the population aged 20 to 64 (n=38,527). Given the large samples and exceptionally high response rates of NHIS, these estimates are generally regarded as the best available estimates of national smoking patterns. To examine recent 10-year changes in smoking behavior by occupational category, the 1978-1980 NHIS estimates have also been compared with the 1970 NHIS estimates for selected smoking variables. A more detailed description of the NHIS data base is provided in the Technical Addendum to this section.

## **Patterns of Employment**

Before characterizing the smoking behavior of the U.S. adult workforce, it will be useful to describe the patterns of employment for men and women. As is shown in Table 1, men are more likely to be employed in professional and technical, management, and blue-collar occupations. Women are more likely to be employed in professional and technical and clerical and service occupations or to be homemakers. Although there was an increase in participation by women in white-collar occupations between 1970 and 1980, the ranking of occupational categories by their relative frequency for both sexes remained about the same in 1980 as it did in 1970. Because of their low relative frequency, farm, sales, and clerical workers, laborers, and service workers have less impact on the smoking behavior of the total male workforce, and female farm workers, laborers, craftsmen and kindred workers, sales workers, and managers and administrators have a modest impact on the smoking behavior of the total female workforce.

## **Smoking Prevalence**

Surveys have repeatedly shown that blue-collar workers are more likely than white-collar workers to smoke cigarettes (US DHEW 1979). Recent estimates from NHIS continue to substantiate this finding (Table 2). Overall, smoking rates for blue-collar men (47.1 percent) exceed that of white-collar men (33.0 percent). The same pattern holds for women, but is less pronounced, with smoking rates among blue-collar women (38.1 percent) exceeding that of white-collar women (31.9 percent). Among women, this white-collar-blue-

**TABLE 1.—Estimates of the occupational distribution of men and women, aged 20 to 64 years, United States, 1970–1980**

Occupation	Men		Women	
	1970	1978–80	1970	1978–80
Currently employed	87.8	85.1	47.9	57.3
White-collar total	39.2	39.2	31.1	40.5
Professional, technical, and kindred workers	14.2	14.9	7.9	11.4
Managers and administrators, except farm	13.3	13.5	2.6	4.9
Sales workers	5.0	5.3	3.4	3.6
Clerical and kindred workers	6.8	5.5	17.1	20.6
Blue-collar total	43.1	40.8	9.0	9.3
Craftsmen and kindred workers	19.9	20.7	0.8	1.5
Operatives and kindred workers	18.1	14.6	8.0	7.2
Laborers, except farm	5.1	5.5	0.2	0.6
Service	5.4	6.1	10.3	10.8
Farm	3.7	2.9	0.5	0.6
Unemployed	3.6	4.1	3.2	4.3
Usual activity, homemaking	—	—	52.5	41.7

NOTE: The white-collar, blue-collar, service, and farm occupational categories are mutually exclusive; however, those classified as "Homemaking" or "Unemployed" may also be classified in an occupational group on the basis of a recent or part-time job, resulting in a small degree of overlap between categories.

SOURCE: National Center for Health Statistics, National Health Interview Surveys, 1970 and 1978–1980 (combined). (See Technical Addendum.)

collar difference exists only for the younger age group (aged 20 to 44); for older women (aged 45 to 64) there is virtually no difference in smoking prevalence between these two categories of workers.

For men, the highest rates of current smoking occur among craftsmen and kindred workers, operatives and kindred workers, laborers, service workers, and the unemployed. The lowest smoking rates for men occur among professional, technical, and kindred workers, managers and administrators, clerical and kindred workers, and farm workers.

**TABLE 2.—Estimates of the percentage of current smokers by sex, age, and occupation, aged 20 to 64 years, United States, 1978–1980**

Occupation	Women			Men		
	Total	20–44	45–64	Total	20–44	45–64
Total	33.2	34.2	31.4	40.9	41.4	39.8
Currently employed	33.3	34.0	31.8	39.9	40.9	37.7
White-collar total	31.9	31.9	31.9	33.0	33.5	32.2
Professional, technical, and kindred workers	26.5	26.1	27.9	25.7	25.3	26.6
Managers and administrators, except farm	38.3	37.8	39.2	36.3	38.9	32.2
Sales workers	33.3	33.2	33.5	40.6	42.0	38.0
Clerical and kindred workers	33.2	33.9	31.4	37.7	36.4	40.4
Blue-collar total	38.1	41.3	31.9	47.1	48.7	43.6
Craftsmen and kindred workers	44.6	45.4	43.0 *	46.1	47.8	42.6
Operatives and kindred workers	37.0	40.2	30.8	48.6	50.4	44.5
Laborers, except farm	36.2	43.0 *	14.1 *	46.8	47.3	45.1
Service	37.4	39.8	32.7	47.5	48.3	46.0
Farm	22.6	31.3 *	7.1 *	31.5	28.9	34.5
Unemployed	39.6	41.7	30.4	53.1	53.9	50.8
Usual activity, homemaking	33.0	35.1	30.4	—	—	—

\* < 100 cases in the denominator (unweighted sample).

SOURCE: National Center for Health Statistics, National Health Interview Surveys, 1978–1980 (combined). (See Technical Addendum.)

For women 20 to 64 years of age, the highest smoking rates are found among craftsmen and kindred workers and managers and administrators. Among women 20 to 44 years of age, there are also relatively high smoking rates among operatives and kindred workers, service workers, and the unemployed. The lowest rates of current smoking occur among professional, technical, and kindred workers, regardless of age. For homemakers, the category representing nearly 42 percent of all women aged 20 to 64, the prevalence of smoking among those aged 20 to 44 is midway between the