
# Work, Smøking, and Health
# A NIOSH Scientific Workshop

**June 15-16, 2000**
**Washington Court Hotel**
**Washington, DC**

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## Background

Tobacco use, with its associated morbidity and mortality, is unevenly distributed across the working population. Tobacco has a significant adverse effect on people who work and their families, both alone and in combination with workplace exposures. While at work, people may be exposed to personal or environmental tobacco smoke, to workplace toxins or stressors, or both. Tobacco smoke in the home can affect the children of smoking adults, and these children are also more likely to smoke themselves. Thus, factors that encourage or discourage smoking among workers can have a profound effect on the health of future generations. Prevention of health effects related to tobacco use remains one of the highest public health priorities.

## The Workshop

On June 15-16, 2000, 79 leaders from labor, industry, academia, government, and non-governmental agencies participated in a scientific workshop titled, "Work, Smoking and Health" in Washington, D.C. Speakers included individuals with national perspectives and extensive experience in public health, tobacco control, and occupational health. During the workshop, the various interrelationships among work, work exposures, tobacco use, and health were explored. Questions addressed included:

- How do active smoking, passive smoking, and other workplace exposures combine to affect worker health?
- What are the reasons for increased rates of smoking in some occupations?
- How can workplace policies and programs be most effective in protecting workers from the combined effects of active smoking, environmental tobacco smoke, and other occupational health hazards?
- What should be done to reduce the adverse health effects from tobacco smoke among working people?

## Workshop Faculty

### Workshop Chair
**Gregory Wagner, M.D., NIOSH**

### Workshop Co-Chairs
**David Christiani, M.D.**
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**Jonathan Samet, M.D., M.S.**
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**Donald Shopland,**
**NCI**

**Glorian Sorenson, Ph.D., M.P.H.**
**Dana-Farber Cancer Institute**
**Harvard School of Public Health**

## Program

**June 14, 2000 ▬ 6:30–8:30 PM** – Registration
**June 15, 2000 ▬** Day 1 – Plenary Session and Parallel Tracks
**June 16, 2000 ▬** Day 2 – Parallel Tracks and Plenary Session Wrap Up

## Day 1: Plenary Session

**7:30–8:30 AM** – Registration
**8:30 AM** – Work, tobacco smoking, and health: What are the Issues. – **Linda Rosenstock**

**9:00 AM** – The distribution of smoking behavior among working people: Prevalence of smoking and selected quitting behaviors by industry and occupation. – **Gary Giovino/Linda Pederson**

**9:30 AM** – Environmental tobacco smoke at work: Lessons from the past and challenges for the future. – **Jonathan Samet**

**9:50 AM** – Environmental tobacco smoke in the workplace - Trends in the protection of U.S. workers. – **Donald Shopland**

**10:10 AM** – Presentation of summaries of breakout-session background papers. – **William Beckett, John Dement, Ross Brownson,** and **Glorian Sorenson**

Charge to the working groups – **Gregory Wagner**

**10:45 AM** – Break

**June 15, 2000**

## Day 1: Parallel Tracks

**Track One: Interactions between tobacco and other occupational health hazards.**

**Chair – David Christiani**
**Co-Chair – Lee Petsonk**
**Rapporteur – Robert Castellan**

**11:00 AM Session 1**: Tobacco smoke and work-related non-malignant respiratory disease (**William Beckett**)

**11:30 AM** Workgroup Discussion: Questions, Knowledge Gaps, and Priorities

**12:30 – 2:00 PM** Lunch

**2:00 PM Session 2**: Tobacco smoke and workplace hazards: Cancer, heart disease, and other occupational health risks (**John Dement**)

**2:30 PM** Workgroup Discussion: Questions, Knowledge Gaps, and Priorities

**3:15 PM** – Break

**3:30 – 5:00 PM** Workgroup Discussion: Additional data presentations, Development of Recommendations

**Track Two: Reducing tobacco related health effects through workplace based programs, policies, and regulations**.

**Chair – James Merchant**
**Co-Chair – Gregory Wagner**
**Rapporteur – Linda Goldenhar**

**11:00 AM Session 1**: Approaches to reducing the health burden associated with smoking through workplace based policies and regulations (**Ross Brownson**)

**11:30 AM** Workgroup Discussion: Questions, Knowledge Gaps, and Priorities

**12:30 – 2:00 PM** Lunch

**2:00 PM Session 2**: Smoking cessation at the workplace: What works, and what is the role of occupational health? (**Glorian Sorenson**)

**2:30 PM** Workgroup Discussion: Questions, Knowledge Gaps, and Priorities

**3:15 PM** – Break

**3:30 – 5:00 PM** Workgroup Discussion: Additional data presentations, Development of Recommendations

**June 16, 2000**

## Day 2: Parallel Tracks

**Track One: Interactions between tobacco and other occupational health hazards.**

**8:30 – 10:30 AM**: Identifying gaps in current science, policy development, and practice that impede protection

**10:30 AM** – Break

**Track Two: Reducing tobacco related health effects through workplace based programs, policies, and regulations**.

**8:30 – 10:30 AM**: Identifying gaps in current science, policy development, and practice that impede protection

## Day 2: Plenary Session – Workshop Wrap Up

**10:45 AM – Noon**: Summary reports from work groups: Recommendations for future research, policy development, and implementation of effective interventions.

# Commonly Used Terms

*Active smoking* – The act of smoking a cigarette.
*Passive smoking* – The inhalation of environmental tobacco smoke by non-smokers.

*ETS* – Environmental tobacco smoke: The mixture of sidestream smoke and exhaled mainstream smoke that pollutes air in locations where tobacco smoking is taking place. The major source of ETS is sidestream smoke.

*MS* – Mainstream smoke–Smoke from a burning cigarette inhaled into the smoker's mouth.
*SS* – Sidestream smoke–Smoke generated by smoldering tobacco between puffs and smoke escaping during puffing.

*Interactions** – Interactions between cigarette smoking and occupational exposures may be examined in the context of a biological process, as a statistical phenomenon, or as a problem in public health and individual decision making.

*Biologic interactions*: The presence of one agent influencing the form, availability, or effect of a second agent and includes physical interaction, e.g, the adsorption of carcinogens onto particulates in inspired air; process interactions, e.g., the induction by one agent of an enzyme system capable of converting a second agent into a carcinogenic metabolite; and outcome interactions, e.g., the number of tumors produced by separate and combined exposures in an animal exposure system.

*Statistical interactions*: The approach used to assess "effect modification," i.e., whether the magnitude of the effect of one factor depends on the presence of another. Alternatively, "a departure from the mathematical model used to assess the effects of the exposure variables. The model being tested may be additive, multiplicative, or some other form; the outcome of interest may be death rates, relative risks, or other outcome measures."

*Public health interactions*: The presence or level of one agent influencing the incidence, prevalence, or extent of disease produced by a second agent.

*White collar workers*** – Professional and technical occupations, managers and administrators, sales workers, clerical workers.

*Blue-collar workers*** – Craftsmen, transportation, and non-transportation operatives and laborers, e.g., construction and craft workers, plumbers, pipefitters, carpenters, laborers, roofers, painters, brick masons, and drywall installers.

*Service workers*** – Public servants and private household workers.
*Hospitality workers* – Workers in restaurants, bars, hotels and motels, and other entertainment occupations.

* Defined in: The health consequences of smoking: cancer and chronic lung disease in the workplace, a report of the Surgeon General, Chapter 3, Evaluation of smoking-related cancers in the workplace, pages 104-113 [DHHS 1995].

**National Health Interview Survey and Current Population Survey definitions.

# Introduction

Cigarette smoking is the single greatest preventable cause of lung disease in the U.S. adult population. Tobacco smoking has numerous, well-documented, adverse health effects, both alone and in combination with hazardous workplace exposures. People who work may be active smokers and be exposed to workplace toxins or stressors, including environmental tobacco smoke. Tobacco smoke in the home can also affect the children of smoking adults, and these children are more likely to take up smoking. Prevention of the health effects related to tobacco among American workers and their families remains one of the highest public health priorities.

Over the past two decades, the public health and medical communities have made substantial progress in combating cigarette smoking. However, despite overall progress, the burden of tobacco-related illness has clearly shifted toward blue-collar and service sector workers, whose cigarette smoking and exposure to environmental tobacco smoke, as well as to many other occupational health and safety hazards, is considerably more frequent than white collar workers. To address the impact of tobacco on the health of American workers, the National Institute for Occupational Safety and Health (NIOSH) convened a scientific workshop in June 2000 and invited leaders from labor, industry, academia, government, and non-governmental organizations to participate. The objectives of the workshop were to:

Describe the current state of knowledge of the complex interrelationships among work, work exposures, tobacco use, and health;

Review interventions that have been successful;

Identify key gaps in knowledge relevant to improving worker health protection;

Evaluate research, policy, and action priorities to reduce the adverse health effects of tobacco on American workers and their families;

Bring the occupational safety and health and tobacco control communities together;

Motivate further action and improve coordination in the attack on tobacco-related hazards.

# Workshop Presentations
## Plenary Session

"Tobacco, Smoking, and Health: What Are the Issues?"
Dr. Linda Rosenstock

Dr. Linda Rosenstock, NIOSH Director, welcomed workshop participants and emphasized the need for a coordinated and comprehensive view of smoking, work, and health. Noting the complexity of issues related to smoking in the workplace, she acknowledged the consistent concerns of members of the tobacco control and occupational health communities regarding the health consequences of exposure to tobacco smoke and other workplace exposures. She challenged participants to focus on workplace factors that may influence smoking rates and smoking cessation among workers, including the role of work organization in smoking, tobacco, and workplace health. Issues of job stress, contingency work, shift work, downsizing, and the overall influence of the global economy on the nature of work need special consideration. Dr. Rosenstock encouraged participants to review the state of the current knowledge of work, smoking, and health; suggest research, surveillance, evaluations, and other measures to address the gaps; and discover ways scientists and public health workers from the occupational health and tobacco control communities might work together more effectively to reduce risks and improve the health of workers.

"The prevalence of selected cigarette smoking behaviors
by occupational class in the United States."
Gary Giovino and Linda Pederson

In the initial plenary session, Drs. Gary Giovino and Linda Pederson presented information based primarily on recent National Health Interview Survey (NHIS) data. They described the distribution of smoking behavior among working people in the United States and summarized the prevalence of smoking and selected quitting behaviors by industry and occupation (see pages 22–29). Overall, smoking rates and per capita tobacco consumption have declined over the past two decades. However, higher smoking prevalence rates and smaller declines in smoking were noted among blue-collar occupations and service workers, compared to other groups. These workers had also begun smoking at a younger age, smoked more heavily, and were less likely to be offered smoking cessation programs at work. The proportion of quit attempts was similar by occupational group, but white-collar workers appeared to be more successful in quitting.

"Environmental Tobacco Smoke in the Workplace:
Lessons from the Past and Challenges for the Future"
Dr. Jonathan Samet

In the second plenary presentation, Dr. Jonathan Samet reviewed current knowledge of the health effects associated with exposure to environmental tobacco smoke (ETS), and described recent data on the distribution of risks attributable to this exposure in the workplace. Dr. Samet highlighted the large numbers of workers who remain exposed to ETS and the particular segments of industry, such as hospitality workers (see definitions), in which exposures remain prevalent. His presentation was based on two published papers (see pages 30–49).

"Trends in Workplace Protection from Environmental Tobacco Smoke"
Donald Shopland

Mr. Shopland discussed recent trends in policies designed to protect U.S. workers from occupational exposure to ETS. These trends are based upon data from the National Cancer Institute's Tobacco Use Supplement to the Current Population Survey (see pages 50–61). The data demonstrate an increasing trend over the last decade toward smoke-free workplace policies. However, the degree of protection was not uniform, and deficiencies were highlighted for teenagers and for blue-collar and service workers, especially food service workers. In addition, across the United States considerable variability was noted among states in the level of worker protection from ETS.

## Charge to the Working Groups

Dr. Gregory R. Wagner

For years, the segment of the public health community focused on tobacco control and the segment dedicated to worker health protection have been separated by a wide chasm. Tobacco use by workers has been seen as independent of work, and worker-health advocates have been concerned that excessive attention to individual smoking behaviors blames victims and deflects attention from the importance of reducing hazardous exposures from work. In contrast, tobacco control advocates point to the incredible health risk posed by tobacco smoking. This workshop attempted to bridge that gap.

With this in mind, Dr. Wagner addressed the working groups and asked that they : (1) provide a vehicle for dialog and interaction among diverse people concerned with the interrelationships of tobacco use, tobacco control strategies, the organization of work, and workplace hazards; (2) identify what is known about the interactions between work, tobacco smoking, and health and develop research recommendations to fill important knowledge gaps; (3) review the current rationale and effectiveness of workplace policies and practices intended to reduce tobacco exposures and recommend improvements; and (4) suggest what can be done to promote multi-disciplinary work utilizing the strengths of the traditional occupational health and tobacco control communities to prevent disease resulting from the combination of tobacco smoke and work exposures.

The summaries of workgroup discussions are anticipated to provide a compilation of relevant science and policy issues, suggest a research agenda, advance health protection policy and promote useful interaction and collaboration between the traditionally separated occupational health and tobacco control communities.

## Tracks One and Two

Following the Plenary papers, the Workshop divided into two parallel tracks. Track One focused on “Interactions between tobacco and other occupational health hazards.” Two invited papers preceded the discussion. Dr. William Beckett presented a paper titled "Tobacco smoke and work-related nonmalignant respiratory disease." Dr. John Dement then presented "Tobacco smoking and workplace hazards: cancer, heart disease, and other occupational risks" (see pages 62–89 for the full text of these papers).

Track Two focused on “Reducing tobacco-related health effects through workplace-based programs, policies, and regulations.” This session included two invited papers, “Policy-related approaches to reducing environmental tobacco smoke exposure in the workplace,” by Dr. Ross Brownson and “Smoking cessation at the workplace: What works, and what is the role of occupational health?” by Dr. Glorian Sorensen. The full text of these papers can be found on pages 90–119.

The **Overall Conclusions** section of this document highlights the major findings that emerged from the Workshop. The **Research Recommendations** section addresses the knowledge gaps and research needs identified during the workshop, with respect to understanding the relationships between work, smoking, and health. This section also identifies research needs related to the development of programs and policies to reduce disease related to tobacco use and occupational health hazards. The **Programs and Policies to Protect Workers from ETS Exposure and Support Smoking Cessation** section summarizes the programs and policies described by the Workshop authors as effective or holding promise in reducing the risk of disease related to active smoking and exposure to occupational hazards including environmental tobacco smoke. The final section, **Process Recommendations**, reviews the process factors suggested by Workshop participants that promote the collaborations needed to improve worker health.

# Summaries of Tracks One and Two Presentations

*Track One: Interactions between tobacco smoke and other occupational health hazards and related discussion.*

"Tobacco smoke and work-related non-malignant respiratory disease"
Dr. William Beckett

Tobacco smoke alone causes several lung diseases, including chronic bronchitis, emphysema, bronchiolitis, and chronic obstructive pulmonary disease (COPD). Exposure at work to various dusts, gases, and fumes can also cause these lung diseases. Specific occupational agents may additionally result in other respiratory diseases, such as pneumoconiosis, asthma, and hypersensitivity pneumonitis (HP). Combined exposure to cigarette smoking and occupational lung toxins may result in adverse health effects that are *additive* or greater than additive (*synergistic*). Rarely, there may also be *antagonism*, in which the combined health effects are less than additive.

An extensive list of workplace agents are known to cause airflow limitation. Numerous studies have also demonstrated an additive effect from tobacco smoking and occupational exposures in the prevalence and severity of chronic bronchitis. The specific proportion of COPD in the U.S. population attributable to occupational exposures is an active topic of current investigation. It has been estimated to be as high as 14 to 28%. Workers who smoke and are occupationally-exposed to certain high molecular weight, asthma-causing dusts may have an increased risk of immunologic sensitization and a more rapid onset of occupational asthma. In contrast, for other sensitizing occupational exposures such as diisocyanates, there is no apparent relationship to cigarette smoking and respiratory disease. Among asbestos-exposed workers, some studies have associated smoking with a slight increase (½ category, based on the International Labour Organisation Classification of Radiographs for Pneumoconiosis) in the radiographic profusion of pneumoconiosis. Physicians caring for workers may not be fully aware of the variety and severity of adverse effects of occupational exposures on the lung and may, therefore, attribute these effects to tobacco smoking alone.

"Tobacco smoking and workplace hazards: Cancer, heart disease,
and other occupational risks"
Dr. John Dement

Both smoking habits and exposures to ETS have been shown to vary substantially by occupation, with blue-collar workers generally having higher exposures. These same populations also experience greater exposures to occupational risk factors. Thus, the potential exists for important interactions to occur between smoking and workplace exposures to physical, chemical, and biological agents. Mainstream smoke (MS) and ETS contain at least 250 chemicals known to be toxic or carcinogenic. Exposure to tobacco smoke and certain occupational agents are known to increase the risk for cancers at selected sites as well as cardiovascular diseases. Smoking also has been shown to be a risk factor for work-related injuries, injury- related deaths, and low back pain.

Many of the chemical compounds found in tobacco smoke also occur in workplaces. Thus, workers exposed to toxic chemicals at work receive additional exposures from the presence of those same toxic chemicals in tobacco smoke. In some instances, the effects of tobacco smoke and occupational exposures are greater than additive. Additional research is needed to better define the independent and interactive effects of tobacco smoke and occupational exposures. Innovative intervention and prevention programs are needed to address both smoking and occupational risk factors.

*Track Two: Reducing tobacco-related health effects through workplace-based programs, policies and regulations and related discussion.*

"Policy-Related Approaches To Reducing Environmental Tobacco Smoke Exposure In The Workplace"
Ross Brownson and David Hopkins

Dr. Brownson reviewed the rationale for restricting cigarette smoking in public settings and in the workplace, including the recognized health effects of ETS, public annoyance and discomfort from ETS exposure, and the potential benefits to smokers. The presentation covered: 1) workplace policy initiatives that have been designed to reduce ETS exposure, 2) effects and effectiveness of such policy measures, and 3) areas of future importance for policy development and research.

Occupational exposure to ETS is strongly influenced by the type of job and the specific smoking policy. Blue-collar and service workers are more likely to be exposed to ETS in the workplace than workers in other sectors. Workplace smoking bans have been demonstrated to be effective in reducing nonsmokers' exposure to ETS, and also result in decreased consumption of cigarettes among smokers. However, the impact of smoking bans on quitting behavior is less clear. Such bans are becoming more common, and enjoy considerable support among both smokers and nonsmokers.

Despite substantial progress in protecting workers from ETS, additional efforts are needed in several areas including: attention to ETS exposure and smoking policies among blue-collar workers and in smaller workplaces; studies of enforcement; effects of smoking policies on smoking cessation; and comprehensive cost-effectiveness studies.

"Smoking Cessation At The Worksite: What Works and What Is The Role Of Occupational Health?"
Dr. Glorian Sorensen

Dr. Sorensen presented a model for worksite smoking cessation that is embedded in a comprehensive approach to worker health. A comprehensive approach to worker health addresses multiple factors influencing worker health, including reduction of exposures to workplace hazards, modification of job factors to support healthy outcomes, and promotion of health-enhancing behaviors, including non-smoking. Such an approach targets multiple levels of influence, including the work environment, workplace organization, interpersonal supports, and the individual worker.

This model draws heavily on research conducted in the tobacco control arena. It extends that research to conceptualize a model for worker health that incorporates both tobacco control and occupational health. A summary of the key findings from Dr. Sorensen's research, including promising intervention strategies and priorities for future investigation, are described in the sections: Overall Conclusions and Research Recommendations.

## Workgroup Discussions

Discussion highlighted the need to develop, modify, and tailor smoking policies and cessation approaches for a range of worksites including institutional settings where residents may be allowed to smoke (e.g., prisons and psychiatric facility), small businesses, and unionized and non-unionized worksites. Discussion also emphasized the need to consider alternative approaches to protecting workers from ETS such as mandatory ventilation rates in settings where smoking bans are precluded (e.g., by collective bargaining agreements).

Discussants mentioned that pulmonary function testing (e.g., single breath diffusing capacity, nonspecific airway responsiveness, various markers of inflammation) had been established as a useful tool in investigating effects of many of the respirable occupational particulate dusts. However, the utility of these tests compared to other respiratory outcomes in investigating tobacco interactions with other exposures, including fibrogenic dusts and fine and ultrafine particulates, may benefit from further evaluation.

Norman Anderson, from the Carpenters Health and Security Trust of Western Washington, presented preliminary results of a three-year pilot smoking cessation program, begun in 1999. The program was financed through a joint labor/management insurance fund for carpenters. Ten percent of the Fund's health care costs are related to the treatment of tobacco-related illness. The program uses minimal intervention techniques, including a telephone-based disease management counseling program, combined with drug therapy. Participation in this program and 3-month quit rates among the carpenters are promising, although the long-term impact of this approach needs to be evaluated.

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# Overall Workshop Conclusions

**Occupational disparities in exposures to tobacco smoke appear to be increasing.**
- Blue collar and service workers report smoking more and quitting less often than other workers.
- Teenage workers, workers in blue collar and service jobs, and workers in certain states are offered less protection from exposure to occupational tobacco smoke at work than other workers.

**Combined adverse health effects from exposure to occupational hazards and tobacco smoke are usually greater than for either exposure alone.**

**Several specific approaches to workplace-based smoking cessation may be effective.**
- Approaches integrating health promotion and health protection offer promise.
- Joint labor/management efforts can reduce smoking among blue collar workers.

*Disparities in the Prevalence of Cigarette Smoking Among Occupational Groups*
The Workshop identified positive, as well as troubling, public health trends. Overall, smoking rates and per capita tobacco consumption have declined over the past two decades. However, higher smoking prevalence rates and smaller declines in smoking were noted among blue-collar occupations and service workers. Workers in these sectors had begun smoking at a younger age, smoked more heavily and were less likely to be offered smoking cessation programs at work. They were also more likely to be exposed to hazardous agents on the job. White-collar workers appeared to be more successful in quitting than other workers, further increasing the disparity in smoking rates.

*Disparities in Protection of Workers from Environmental Tobacco Smoke*
Although exposures to ETS have been documented in some workplaces at levels ten times higher than in the homes of smokers, recent data confirm that smoking restrictions directed at reducing exposure to ETS have become more common in U.S. workplaces. A 1999 U.S. survey found that 79% of workplaces with 50 or more employees had formal smoking policies that prohibited or limited smoking to separately ventilated areas. Data demonstrate an increasing trend over the last decade toward smoke-free workplace policies. This trend may result, in part, from an acknowledgment that workplace policies that allow smoking in designated areas, without separate ventilation, result in substantial nonsmoker exposure to ETS.

Despite the trend toward smoke-free workplace policies, Shopland and Samet described important disparities in the degree of protection from environmental tobacco smoke for teenagers; for blue-collar and service workers, especially those in food service; and for hospitality workers. Workers in smaller places of employment (< 50 workers) may not be covered by such policies. There is also great variability in the degree of worker protection from exposure to ETS among the states.

*Combined Health Impact of Cigarette Smoking and Exposure to Workplace Hazards including ETS.*
The deleterious health effects of cigarette smoking alone and exposure to environmental tobacco smoke alone have been well documented. This Workshop highlighted important differences in disease outcomes for active smokers who are also exposed to occupational hazards. There has been less research on the health effects in non-smoking workers with respect to ETS exposure in combination with other occupational hazards. However, a spectrum of disease risks comparable to that resulting from active smoking and exposure to similar occupational hazards would be anticipated.

Most health effects of these dual exposures appear to be additive, i.e., the effects of tobacco smoking and the occupational hazard combine to result in a greater adverse health effect than either agent alone would produce.

Synergistic effects have also been documented in which the combined effects of tobacco use and exposure to occupational hazards are greater than additive.

*Diseases Associated with Active Smoking and Exposure to Occupational Health Hazards*

The combination of active smoking and exposure to selected occupational hazards is known to increase the prevalence and severity of certain non-malignant lung diseases and increase the risk for cancer at selected sites, as well as the risk for cardiovascular disease. Many studies show an additive increase in the prevalence and severity of chronic bronchitis and COPD in smokers exposed to coal mine dust and silica. Excess cancers of the lung and other organs from active smoking and exposure to a number of occupational health hazards are well documented. Lung cancer risk may be multiplied when active smokers are exposed to asbestos, silica, radon daughters, arsenic, and chloromethyl ethers. The risk of bladder cancer may be markedly increased when active smokers are exposed to bladder carcinogens. Active smoking among men and women may cause multiple forms of cardiovascular disease. Additive cardiovascular toxicity is also considered likely in workplaces when active or passive smoking combines with exposure to cardiovascular stressors such as chronic job strain, lead, carbon monoxide, and carbon disulfide. Smoking has also been associated with a 61% increased risk of occupational traumatic death. Interactions between tobacco and specific workplace hazards and possible mechanisms of these diseases are discussed in the Beckett and Dement papers (pages 63–90).

*Workplace-based Smoking Cessation Programs*

Data from the 1994 National Health Interview Survey indicated that workplace-based smoking cessation programs have not achieved a high level of success, with only 4.6% of respondents reporting participation in these programs. There is a particular need to improve participation rates among blue-collar and service workers and others in high-risk industries. The Workshop highlighted the potential for improving these rates through programs that intervene at multiple levels of influence at the worksite, including the organizational level, the interpersonal level, and the individual level. These intervention programs coordinate health promotion and occupational health protection efforts at the worksite to reduce occupational exposures and encourage healthy lifestyles.

There are growing precedents for worksite programs that integrate efforts to reduce behavioral risks, including tobacco use, with health protection initiatives. While the impact of these programs has not been fully evaluated, there is promising evidence from the WellWorks Study (see Sorensen, pages 99-120) that programs integrating occupational safety and health with health promotion efforts may be more effective than health promotion programs alone in improving worker health, including smoking cessation.

The WellWorks study, conducted as part of the National Cancer Institute's national worksite initiative known as the Working Well Trial, tested the effectiveness of a model integrating health promotion and health protection. The intervention included programs targeting both workers and management that combined messages on tobacco control, nutrition, and occupational health. Of three study centers in the Working Well Trial to address smoking cessation, the WellWorks study (including 24 worksites) was the only one in which a significant increase in smoking cessation was observed.

Dr. Sorensen presented evidence that efforts by management to reduce job risks may encourage blue-collar workers to engage in programs aimed at changing smoking behavior. Workers who reported that their employer had made changes to reduce exposures on the job were significantly more likely to have participated in smoking control and nutrition programs than workers not reporting these management actions [Sorensen et al. 1996b].

Awareness of the synergistic effects of tobacco smoking and exposure to job hazards was also associated with increased interest in quitting in a blue-collar working population. Smoking cessation programs that integrate information about the combined risks of smoking and exposure to occupational hazards may improve the effectiveness of smoking cessation interventions among workers exposed to job hazards. Smoking cessation may also be emphasized in the context of the changing nature of work, i.e., downsizing, loss of benefits, contingent and part-time work, and out-reach efforts such as home-based work. Occupational and worksite “cultures” need to be understood so that smoking cessation programs can be appropriately tailored, and barriers, as well as promoters, to the acceptance of smoking cessation programs need to be defined.

Preliminary data presented at the conference suggests that well designed and targeted non-worksite based minimal smoking cessation interventions sponsored by joint labor/management insurance funds may reduce smoking among blue-collar workers.

# Recommendations

Recommendations, summarized below, reflect the sense of the deliberations. However, no attempt was made to reach consensus within the groups, nor did the participants establish research priorities. The discussants identified areas of uncertainty; gaps in current information and knowledge; and opportunities for advancing research, evaluation, and monitoring methods.

**High Risk Occupations**

Investigate ETS exposures and smoking prevalence rates in unstudied, high-risk occupations
Improve characterization of ETS exposure in blue-color and service worker settings

**Diseases, Injuries and Quality-of-life Outcomes**

Describe combined impacts of occupational exposures, ETS, and active smoking on asthma, injuries, and cardiovascular function
Improve understanding of mechanisms of interactions
Improve tools and methods for evaluating health, injury, and quality-of-life outcomes
Utilize non-traditional databases and study designs for epidemiologic research

**Programs, Policies and Interventions**

Evaluate effectiveness of comprehensive, worksite-based programs
Evaluate development and impact of smoking policies and bans
Improve utility of medical screening and monitoring programs

# Research Recommendations

*ETS Exposures and Smoking Prevalence Rates in High-Risk Occupations*

Smoking prevalence rates should be described among relatively unstudied working groups, such as migrant workers, teenage workers, and Native American and Alaskan Natives, as well as smaller workplaces (with fewer than 50 employees) and non-union settings.

The distribution of ETS exposures in blue-collar and service occupations, among hospitality workers, and in custodial facilities where residents may smoke, needs to be better characterized.

For workplaces where exposures cannot be eliminated, classification of individual ETS exposures could be enhanced with the development of more accurate modeling methods, improved use of biomarkers, and evaluation of the effects of worker ventilatory requirements on dose. Studies in these settings should also characterize the source of ETS exposure and its apportionment in settings where particulates from ETS combine with those generated by work processes. Worker exposure to degradation products of occupational agents that come in contact with a burning cigarette should also be studied (as has been done with polytetrafluoroethylene).

*Diseases, Injuries, and Quality-of-Life Outcomes Resulting from Active and Passive Smoking and Occupational Hazards*

The risk of work-related asthma (including new onset and exacerbations) in relation to active and passive smoking should be more fully investigated. Further study is also needed to explore the association of smoking with work-related chronic and acute health problems and traumatic injuries (fatal, non-fatal, near-miss).

To assess the effects of active smoking and ETS exposure on occupational cardiovascular risks, prospective epidemiologic studies are suggested, especially in blue-collar work settings. These studies should include traditional disease outcomes as well as intermediate cardiovascular effects. Other outcomes that have not been adequately studied include the combined impact of occupational factors and tobacco smoking on acute and chronic

neuro-cognitive and psychiatric outcomes, visual impairment (e.g., cataracts), hearing impairment, respiratory tract infections (e.g., pneumonia/influenza), pulmonary fibrosis, and reproductive health (e.g., fertility impairment).

More data are needed on the combined effects of tobacco use and occupational diseases and injuries on quality of life outcomes, such as functional status and ability to remain on the job, as well as associated economic costs.

*Measurement Tools and Epidemiologic Designs*

In work settings where active and passive exposure to tobacco cannot be controlled, the discussants emphasized the importance of improving the utility of several measurement tools, so that health effects from combined tobacco exposure and occupational hazards can be more effectively assessed. Further study is needed to determine the utility of early markers and intermediate outcomes (e.g., biomarkers, irritative symptoms and symptom complexes) in the evaluation of the health effects of ETS exposure combined with other workplace hazards.

Epidemiological studies of the health effects of combined exposures to tobacco smoke and job exposures will require innovative designs to avoid or account for selection/survivor biases and to make efficient use of intermediate outcomes. Such designs will also need to assess a complex range of potential confounders (e.g., data from Italy suggesting an association between specific dietary habits and exposure to ETS). Investigators should seek opportunities for occupationally relevant studies using existing National Heart Lung and Blood Institute study cohorts (e.g., the Multiple Risk Factors Intervention Trial [MRFIT]), as well as National Center for Health Statistics and managed health care data. Where possible, researchers should estimate population attributable risk (PAR) in addition to odds ratios (OR) or relative risks (RR) due to tobacco smoke and due to occupational factor(s), to better inform intervention strategies. In order to identify ethically responsible and potentially feasible risk modifications, applicable in large populations of at-risk workers, studies will be needed to better define both acquired and genetic susceptibilities (and their interactions).

## Recommendations: Policies and Interventions to Support Smoking Cessation and Reduce Occupational Exposure to ETS and Other Hazards

Workplace programs and policies designed to control exposure to ETS, support smoking cessation, and improve worker protection from occupational safety and health hazards should be objectively evaluated. Intervention programs that adopt a comprehensive approach to reducing active tobacco smoking and exposure to other occupational health and safety hazards, including ETS, should be more fully evaluated. The costs, benefits, and effectiveness of various approaches to smoking cessation need to be evaluated both in the short and long-term, including programs with and without smoking bans. Efforts should focus on high-risk populations such as blue-collar and service workers, teenage workers, hospitality workers, Native American and Alaskan natives, and smaller workplaces (with fewer than 50 employees). Uniform evaluation methods will facilitate comparisons and improve the generalizability of results; thus, development of common definitions, outcomes, and standardized metrics is recommended.

Additional research is needed to explore the many underlying factors that may influence smoking behavior. These may include the organization of work and job characteristics (such as exposure to hazardous chemicals, shift work, high workload demands and low control or decision-making latitude) and the culture at work and at home. Understanding and changing some of these underlying factors poses a difficult but necessary challenge to altering the smoking behavior of workers at high risk. Attention must be paid to ethnically and culturally appropriate communication and intervention strategies.

## Recommendations: Development and Impact of Smoking Policies and Bans

Workplace smoking bans clearly reduce the number of cigarettes consumed by smokers per day. Smoking bans and other workplace smoking policies have become a key component of an overall workplace tobacco control effort and are central to supporting smoking cessation among workers. However, additional study is needed on the changing patterns of smoking associated with worksite smoking bans. Studies should investigate and define how smoking policies are developed and implemented and should assess both intended and unintended consequences of the

different approaches. Characteristics of effective strategies should be documented. For example, there is evidence that when unions are partners in shaping smoking policies, the policies are not as likely to be contested as when policies are implemented unilaterally by management.

Questions arose regarding the impact of smoking policies. For example, do smokers and nonsmokers differentially select into jobs based on workplace smoking policies? Do smoking bans impact the health effects resulting from combined exposure to active smoking and occupational hazards (such as when tobacco smoking and workplace exposures are sequential rather than concurrent)? When bans are implemented, does the individual dose change for those who continue to smoke? Does the process of confining smokers to separately ventilated areas expose smokers to higher concentrations of sidestream smoke? Additional research may clarify the potential for increased health risks in these separate smoking rooms. Finally, the practical effectiveness of administrative and engineering controls directed at reducing ETS exposure at work needs further evaluation.

## Recommendations: Medical Screening and Monitoring Programs

Studies are also needed to improve the utility of workplace-based medical screening and monitoring programs. Abnormal medical test results among workers exposed to both tobacco smoke and occupational hazards require improved interpretation and intervention strategies. Physicians involved in medical screening programs need to be aware of the combined health effects of active or passive smoking and exposures to other workplace hazards documented in this Workshop.

## Recommendations: Policies and Programs to Protect Workers from ETS Exposure

Much can be done at this time to improve workplace policies and programs in addressing tobacco-related issues in the workplace by the following suggestions.

**Improving Workplace Policies and Programs**

*Target Interventions to High Risk Populations by*
- Communicating risks of exposure and benefits of bans
- Promoting comprehensive approaches to health promotion and protection
- Utilizing less intensive strategies and minimal intervention approaches

*Control Exposure to Environmental Tobacco Smoke with*
- Smoking bans and other policies
- Separately ventilated smoking areas

*Address Barriers to Achieving Improved Worker Health via*
- Management commitment
- Collaboration among managers, unions, workers, and health care providers

*Target Interventions to High-Risk Occupations and Populations*

A number of high risk occupations and industries have been identified. The significant occupational health and safety risks related to tobacco smoking, ETS, and other occupational exposures documented in this Workshop, and the apparent benefits of smoking bans need to be communicated widely, particularly to managers, unions, health care providers, and community-based groups with access to workers at high risk. Research on comprehensive intervention models such as the WellWorks Study, that integrate tobacco control and occupational safety and health

protection efforts should be replicated and expanded. Intervention trials that tailor and adapt smoking policies and cessation approaches to meet the needs of workers and the varying conditions at different worksites and industries, should be supported. Research needs with respect to these studies are detailed in the Sorensen paper.

Non worksite-based approaches to smoking cessation also ought to be more fully evaluated and replicated. The participation and 3-month quit rates achieved in the minimal intervention program offered to carpenters through the Carpenter Labor/Management Insurance Fund are promising, although the long-term impact is not yet known. Such less intensive strategies for reducing smoking are relatively low cost and low risk, and may be effective in reaching workers at high risk.

*Control Exposure to Environmental Tobacco Smoke*

Since ETS is a recognized human carcinogen, workplace smoking policies should focus on reducing worker exposure to ETS to the lowest feasible concentration. Smoking bans have been shown to be effective in eliminating exposure to ETS as well as reducing cigarette consumption in smokers. Smoking policies, including bans, are a key component of an overall workplace tobacco control effort and are central to supporting smoking cessation among workers.

Workshop participants noted that the Occupational Safety and Health Administration's (OSHA) proposed regulations on Indoor Air Quality recommend the designation of areas with separate ventilation and limiting smokers use to 30 minutes a day. While separately ventilated smoking areas are far superior to smoking areas not separately ventilated, the potential increased lung cancer risk for persons who enter these smoking areas (over that present from inhaling the individual's own cigarette smoke) needs to be better defined and then communicated to the workers who enter these areas. To accomplish this, workshop participants suggested that training programs required by OSHA's Hazard Communication Standard incorporate information about the adverse health effects related to ETS and to the combined effects of workplace exposures, ETS, and active smoking.

Workplaces that have adopted smoking bans should provide a variety of options directed at smoking cessation to encourage and support workers' attempts to quit smoking. These options include smoking cessation clinics, medical interventions, minimal intervention programs, incentives and competitions, and social and environmental supports. Combinations of strategies appear to increase the chances of influencing smokers at varying stages of readiness to quit (see Sorensen paper).

*Address Barriers to Achieving Improved Worker Health*

As this Workshop has documented, smoking and job exposures, alone and in combination, contribute significantly to the burden of disease and injury in workers. The Sorensen paper describes one intervention model based at the worksite that offers an unusual opportunity to reach large numbers of workers to improve their health. However, there are several barriers to overcome in implementing this model or other similar approaches. First, management and worker representatives must be committed to both smoking cessation and reduction of job risks. Second, comprehensive approaches to improving worker health will require changes in the training of professionals in both occupational safety and health and health promotion, as well as in the way government agencies and funding agencies think about worker health. New collaborations and lines of communication are needed among different agencies, management, labor, workers, occupational safety and health advocates, tobacco control and health promotion professionals, insurers, and others committed to improving worker health.

## Recommendations: Promoting Collaboration Between Tobacco Control and Occupational Safety and Health Communities

Workshop participants recommended additional activities be supported that promote improved communication among researchers, funding agencies, management, workers, and unions. Additional workshops and information exchanges will encourage increased interdisciplinary collaboration. Funding initiatives should be jointly sponsored (e.g., National Cancer Institute, National Heart Lung and Blood Institute, Office on Smoking and Health, NIOSH) to maximize the likelihood of true interdisciplinary proposals.

Requests for Applications can encourage multi-disciplinary partnerships and research teams and encourage innovative research models such as participatory or action research. Initiatives should also facilitate expansion of the research infrastructure to promote the needed intra/interdisciplinary work. The effectiveness of the personnel

who administer comprehensive worksite-based programs could be increased through the availability of professional training programs that offer students integrated interdisciplinary courses in health promotion and occupational safety and health.

NIOSH and its partners can utilize an existing framework, the National Occupational Research Agenda (NORA), to develop and support a national smoking and work initiative. NORA was initiated in 1996 to guide occupational safety and health research into the next decade, and represents a broad consensus of stakeholders. A number of research needs highlighted during this workshop had previously been identified as NORA priority research areas, and can greatly benefit from the collaborative efforts and partnerships envisioned under NORA. Finally, to track progress in this area, the workshop participants recommended that workplace health and safety issues should be regularly included in the Surgeon General's Report on Smoking and Health.

# Full Text of Presentations

# The Prevalence of Selected Cigarette Smoking Behaviors by Occupational Class in the United States

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## Introduction

As the overall prevalence of cigarette smoking decreased from 42.4% in 1965 to 24.1% in 1998, differences between occupational groups have been consistently reported [CDC 2000; Brackbill et al. 1988; Nelson et al. 1994]. In particular, blue collar and service workers have had higher smoking prevalences than white-collar workers, and these differences have been reported for both men and women [Nelson et al. 1994]. This gap may be widening even further, as the decline in smoking among white-collar workers exceeded that of other groups from the end of the 1970s to the end of the 1980s [Nelson et al. 1994]. How smoking rates by occupation vary by race or ethnicity is much less clear, because this subject has not been explored recently.

To update and expand upon earlier work on cigarette smoking by occupational class, we analyzed data from two national surveys. Our goals were to (1) examine current smoking prevalence by occupational class, not only overall, by also by gender and by race/ethnicity; (2) compare 1997 results with those from 1978 to 1980 and 1987 to 1990; (3) assess the relationship between occupational class and smoking prevalence after controlling for education, age, gender, and race/ethnicity; and (4) assess quit attempts among current smokers, former smoking among ever smokers, the availability of employer assistance with quitting, rates of heavy smoking ($\geq$ 25 cigarettes per day), and age of initiation of regular smoking.

## Methods

*Data Sources*

Two national surveys of adults (ages $\geq$ 18 years) were used, the National Health Interview Survey (NHIS) and the Current Population Survey (CPS).* Conducted by the National Center for Health Statistics, the NHIS is a household survey designed to be representative of the civilian, noninstitutionalized population of the United States. In previously published reports on smoking by occupation that were based on the NHIS, Brackbill and colleagues [Brackbill et al.1988] and Nelson and colleagues [Nelson et al. 1994] combined data from the 1978–1980 and the 1987–1990 surveys, respectively, to provide more stable estimates. Here, we add data from 1997 that, because of changes in questions and design, were analyzed separately. The 1997 NHIS had a final response rate of 80.4%; the response rate for the earlier years combined exceeded 80%.

The major purpose of the CPS, a household survey of persons aged $\geq$ 15 years—selected by probability sample to be representative of the U.S. civilian, noninstitutionalized population—is to provide national labor statistics. National Cancer Institute (NCI) Tobacco Use Supplements were added to the CPS core survey in 1992–1993, 1995–1996, and 1998–1999 as part of NCI's evaluation of the American Stop Smoking Intervention Study for Cancer Prevention (Project ASSIST) [Stillman et al. 1999]. For this analysis, the September 1995, January 1996, and May 1996 supplements were combined; yielding a response rate of 85.5%.

*Occupational Smoking Prevalence for Selected Years*

❖1978 to 1980

In their analysis, Brackbill and colleagues [1988] used 1978–1980 data to estimate cigarette smoking prevalence in 12 broad occupational groups (e.g., professional and technical, clerical) by gender and by detailed categories based on three-digit Census codes for any occupational categories (e.g., pharmacist, roofer) with a weighted population estimate of $\geq$ 100,000 workers. They also estimated prevalence by occupational class (blue-collar, white-collar, service, farming/forestry) and employment status. Three kinds of employment classification were used: employed, unemployed, and not in the labor force (the latter included housewives and persons retired, physically unable to work, or performing unpaid charity work) based on activity during the 2 weeks before the interview. The 1978–1980 data set contained 49,150 records, 28,640 for employed persons. Persons with unknown current smoking status were included in the denominators for the calculations (thus counting them as nonsmokers).

* Although the Current Population Survey includes persons $\geq$ 15 years old, we used data only for persons aged $\geq$ 18 years.

❖ 1987 to 1990

Combining 1987, 1988, and 1990 data from the NHIS, Nelson et al. (1994) created a data set of 129,640 records, including 82,358 for employed persons. They estimated smoking prevalence for the 12 broad occupational groups and four occupational classes, and the 215 occupational categories with weighted population estimates of ≥ 100,000. Unlike the analyses on the 1978–1980 data, persons whose current smoking status was unknown were excluded from the analyses.

❖1997

The redesigned version of the 1997 NHIS included a new sample frame with more primary sampling units, as well as new data collection, using an updated questionnaire administered via computer-assisted personal interviews (NCHS 2000). A total of 36,116 adults were interviewed in 1997, with 20,043 classified as employed. Unlike the earlier surveys, in 1997, employment status was determined by activity during the previous week only. Respondents were defined as employed if they were working at a job or business or if they had a job but did not work in that week. The same occupation and employment status groups and classes were used as in the earlier analyses. As with the 1987–1990 analyses, persons whose current smoking status was unknown were excluded.

❖1995 to1996 CPS

The combined CPS data set for the 3 months selected from 1995 and 1996 included 233,737 records, with 126,713 of them for employed adults. Because the sample was so large, we used it to calculate smoking prevalence by race for the four occupational classes. In addition, we used the data to determine the availability of employer-sponsored smoking cessation programs by occupational class, based on the question, "Within the past 12 months, has your employer offered any stop smoking program or any other help to employees who want to quit smoking?"

*Definitions*

For the 1978–1980 and 1987–1990 combined NHIS data sets, current smokers were those who had smoked ≥ 100 lifetime cigarettes and who smoked at the time of the survey. For the 1997 NHIS and 1995–1996 CPS, current smokers were those who had smoked ≥ 100 lifetime cigarettes and currently smoked every day or on some days.

The change in definition in the 1990s to incorporate some day smoking led to an overall increase in prevalence estimates of 0.9 percentage points [CDC 1994]. The inclusion of persons of unknown smoking status in the denominator of the study by Brackbill and colleagues [1988] led to an estimate of prevalence that was approximately 0.5 percentage points lower than when persons with unknown smoking status were excluded [Nelson et al. 1994]. The National Center for Health Statistics excludes persons with unknown smoking status, because if it leaves them in they must be in the denominator only, which assumes incorrectly that none of them smokes.

In the 1997 NHIS analyses, former smokers were defined as persons who had smoked ≥ 100 lifetime cigarettes but currently smoked "not at all." Heavy smokers were defined as current smokers who smoked ≥ 25 cigarettes per day. Age of smoking initiation was assessed as the age a person first began smoking fairly regularly.

In both the NHIS and the CPS, white-collar workers included professional and technical occupations, managers and administrators, sales workers, and clerical workers. Blue-collar workers included craftsmen, operatives except transportation, transportation operatives and laborers. Farm workers included farmers and farm managers, farm laborers and foremen, as well as fish and forestry workers. Service workers included public servants and private household workers.

*Statistical Analyses*

For both the NHIS and CPS analyses, data files were weighted to yield national estimates, and Survey Data Analysis (SUDAAN) [Shah et al. 1997] was used to estimate standard errors for prevalence estimates, taking the complex survey designs into account. Current smoking prevalences with 95% confidence intervals (CIs) were calculated for the four occupational classes stratified by gender (using NHIS data) and by race or ethnicity (using CPS data). Differences in point estimates in which 95% CIs did not overlap were judged statistically significant. Changes in prevalence were assessed for 1978–1980 to 1997 and 1987–1990 to 1997 using z scores for independent samples.

Two logistic regression models for current smoking were developed using the 1997 NHIS data to determine crude and adjusted odds ratios (ORs) for the four occupational classes. The adjusted model included gender, age in years (18–24, 25–44, 45–64, and ≥ 65 years), race or ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, Asian/Pacific Islander, and American Indian/Alaska Native), and educational attainment in years (<12, 12, 13–15, and ≥16).

The 1997 NHIS data were used to estimate the following prevalences for each of the four occupational classes: (1) among current smokers, quitting for at least one day during the previous year, (2) among ever smokers, former smoking (quit ratios), and (3) among current smokers, smoking ≥ 25 cigarettes per day (heavy smoking). Additionally, the percentages of current and former smokers who had initiated smoking by 16 and 18 years of age were calculated by occupational category.

## Results

*1997 NHIS Prevalence Estimates by Occupational Class and Gender*

According to NHIS data, in 1997 there were 68.1 million white-collar workers in the U.S. civilian, noninstitutionalized population, of whom 14.2 million (20.8% [95% CI = ± 0.9%]) were current smokers (Table 1). Of 28.4 million blue-collar workers, 10.3 million (36.4 ± 1.6%) were current smokers. In addition, 826,000 (27.4 ± 4.4%) of the 3.0 million farm workers and 4.8 million (32.3 ± 2.1%) of the 14.7 million service workers were current smokers. Thus, in 1997, smoking prevalence among blue-collar workers was higher than for any other occupational class; service workers and farm workers were also more likely to smoke than white-collar workers.

Among both men and women in 1997, smoking prevalence was higher among blue-collar workers than farm or white-collar workers. Among men, both service and farm workers were more likely to smoke than were white-collar workers; among women, this was true for service workers but not farm workers. By gender, although the point estimates for 1997 were higher for men in each occupational class, none of the differences was significant.

*Trends in Prevalence by Occupational Class*

NHIS Data (1978–1980, 1987–1990, and 1997)

Both overall and for men, smoking prevalence declined from 1978–1980 to 1997 for all groups but farm workers (Table1). For women, prevalence declined for white-collar workers and service workers. Overall and for men and women, prevalence for farm workers (who had the lowest prevalence in 1978–1980) changed very little over time.

In 1978–1980, blue-collar workers were 38% more likely to smoke cigarettes than were white-collar workers, but by 1997, they were 75% more likely to do so. Similarly, in 1978–1980, service workers were 17% more likely to smoke cigarettes than white-collar workers; by 1997, they were 55% more likely to smoke.

Comparisons of 1978–1980 with 1997 showed that smoking prevalence was 65.6% as high in 1997 as it was in 1978–1980 for white-collar workers, 83.3% as high for blue-collar workers, and 86.8% as high for service workers.

1995/96 CPS Estimates by Occupational Class and Race/Ethnicity

According to CPS data, in each of the racial/ethnic groups, blue-collar and farm workers were more likely to smoke than were white-collar workers. Service workers were more likely than white-collar workers to smoke if they were white, African American, or Asian American/Pacific Islander.

Point estimates below 20% were obtained for Asian American/Pacific Islander, Hispanic, and African American white-collar workers and for Hispanic and Asian American/Pacific Islander service workers. On the other hand, point estimates were at least 30% for American Indians/Alaska Natives in each occupational class, as they were for white blue-collar and service workers and for African American farm workers.

*Multivariate Analysis on 1997 NHIS Data*

Using 1997 NHIS data and logistic regression analysis, crude and adjusted (for age, gender, race/ethnicity, and education) odds ratios (ORs) for current smoking were estimated by occupational class (Figure 1). Consistent with data presented in Table 1, the crude analysis indicated that blue-collar, farm, and service workers were all more likely to smoke than were white-collar workers. In the adjusted analysis, the differences were attenuated considerably, with the OR for farm workers dropping to 0.9, while the ORs for blue-collar and service workers remained significantly greater than the 1.0 OR of the referent white-collar group.

*Other Selected Behaviors*

In 1997, the four occupational classes did not differ in the percentage (by self-report) of current smokers who quit smoking for ≥ 1 day during the previous year (with percentages ranging from 43–48%, data not shown). In contrast, according to 1997 NHIS data, several differences by occupational class were seen in the percentage of ever smokers who were former smokers. Here, 51.3 ± 1.6% of white-collar workers, 36.8 ± 2.1% of blue-collar workers, 41.4 ± 7.0% of farm workers, and 32.8 ± 3.1% of service workers were former smokers.

CPS data from 1995–1996 indicate that very few employees reported that their employer had offered help with quitting smoking during the previous year. Among all employees, for 21.4 ± 0.4% of white-collar workers, 16.1 ± 0.5% of blue-collar workers, 4.9 ± 1.2% of farm workers, and 12.8 ± 0.7% of service workers the employer offered such assistance (by self-report). The pattern among current smokers only was similar: for 19.7 ± 0.8% of white-collar workers, 14.0 ± 0.8% of blue-collar workers, 4.7 ± 2.2% of farm workers, and 11.2 ± 1.1% of service workers the employer offered help with quitting. National data are not available to determine how many employees actually used the programs.

According to 1997 NHIS data for smokers only, blue-collar workers (27.5 ± 2.6%) were as likely as farm workers (27.0 ± 9.5%) to smoke ≥ 25 cigarettes/day. White-collar workers (18.0 ± 2.0%) and service workers (16.7 ± 3.2%) had significantly lower estimates than blue-collar workers.

Among both current and former smokers in 1997, proportionately more blue-collar workers and service workers were regular smokers at early ages (16, 18) than were white-collar workers (Table 3). Among current smokers, more farm workers than white-collar workers were regular smokers before age 16, but by age 18 the difference was not significant.

## Discussion

Not surprisingly, given historical trends, we found that in 1997 both men and women identified as blue-collar or service workers were more likely to be current smokers those workers identified as white-collar. Furthermore, the gaps between white-collar and blue-collar workers and between white-collar and service workers appear to be widening. In addition, compared to white-collar workers who've ever smoked, blue-collar and service workers who've ever smoked were less likely to have quit and were more likely to have started at a young age. To compound matters, blue-collar smokers were more likely to be heavy smokers than white-collar smokers. We found especially high smoking prevalences among American Indian/Alaska Native workers in all occupational classes, white blue-collar and service workers, and African American farm workers.

Analyses conducted for this report (data not shown) and other reports [e.g., Nelson et al. 1994; Shopland's paper in this draft document: 50–62] indicate that smoking prevalence is lowest among persons who often act as role models for young people such as clergy, educators, physicians, and dentists. Furthermore, white-collar workers such as managers, administrators, and professional and technical workers are often in positions of leadership. Thus, in many cases, they can be role models for other workers on abstaining from smoking, often by having quit successfully. Additionally, physicians and dentists, clergy, and other white-collar workers such as lawyers can influence public opinion and the formation of public and private policies regarding reimbursement for cessation activities and the provision of smoke-free indoor air.

We note that the data in this report have several limitations. First, the estimates are only for the civilian population. However, the civilian population accounts for most of the U.S. population and smoking prevalence in the military also declined in recent years (from 51.0% in 1980 to 29.9% in 1998). In 1998, pay grade in the military was inversely related to smoking prevalence, such that the adjusted (for Service, gender, race/ethnicity, education, age, family status, and region) odds for smoking was 6.4 times higher among personnel in the lowest pay grades (E1 to E3) compared to persons in the highest pay grades (O4 to O10) [Bray et al.1998]. Second, the changes in the definition of current smoking in the 1990s attenuated slightly the estimates of declines in smoking. Third, the change in the definition of active employment, which is used to select persons for the questions on occupation, may add some unreliability to the estimation of trends. Fourth, this report does not assess interactions between occupation and industry. It would be interesting to know how prevalences for persons who are in the same job category vary by industry. For example, workers who sell cars may differ substantially from those who sell lumber or other building materials [Brackbill et al. 1988]. Fifth, our analyses of racial or ethnic differences did not control for education or other potentially influential factors. Finally, although the great majority of tobacco users in this country smoke cigarettes, the use of other tobacco products was not assessed.

Our finding that differences in smoking prevalence across occupation groups are widening is consistent with a general societal trend in which selected subpopulations have much higher prevalences than others [Pierce et al. 1989]. For example, in 1980, adults with < 12, 12, and 13–15 years of formal education were 43%, 44%, and 38%, respectively, more likely to be current smokers than were adults with ≥ 16 years of education. By 1997, however, these percentages were, in order, 154%, 158%, and 116% [Office on Smoking and Health 2000].

Consistent with other reports [Johnston et al. 1999; Waldron and Lye 1989], the analyses reported here suggest that at least some of the differences in prevalence by occupational class are determined by early initiation. Men and women who start smoking when they are young are may be more likely to become employed in blue-collar and

service occupations than those who never smoke or begin at later ages. In brief, a selection process may occur in which, for various reasons, adolescents who do not fare well in school or are less interested in education are both more likely to smoke and more apt to become employed in blue-collar and service occupations. Possible relevant factors include stress, tobacco marketing practices, and susceptibility to nicotine dependence. Future research is needed to better understand the processes involved.

Stress from the job itself may influence smoking behaviors. Available evidence suggests that higher levels of job strain (operationalized as high psychological job demands and low level of work control) and perceived occupational stress are associated with increased intensity of smoking and decreased quitting [Conway et al. 1981; Green and Johnson 1990]. Occupational stressors may be more common in blue-collar and service occupations and thus make quitting more difficult [Schilling et al. 1985]. Additionally, cultural norms may also influence smoking, particularly if the worksite culture or norms are relatively supportive of smoking and more tolerant of exposure to environmental tobacco smoke.

Interestingly, we found that occupational class remained a significant predictor of current smoking after controlling for age, gender, educational attainment, and race or ethnicity. In contrast, in a previous report, Novotny and colleagues [Novotny et al. 1988] found no significance for occupational class in any of three models. These investigators used multivariate models to study socioeconomic and demographic differences in ever smoking, former smoking (among ever smokers), and heavy smoking (among current smokers) among 25–64 year old African American and white respondents to the 1985 NHIS. Variables included in the models were age, gender, race, employment status, occupational class (white-collar, blue-collar, and service only), education, marital status, and poverty status. Because these models did not directly assess relationships with current smoking, direct comparisons between their models and ours are not possible. Their work raises the possibility, however, that the associations we observed for occupational class may not persist after controlling for other variables, such as poverty status. Alternatively, Novotny and colleagues may have over-controlled, by using variables highly related to occupational class. Additionally, the interrelationships in 1985 may have differed from those in 1997. Ideally, population prospective studies should be conducted to sort out such issues. If occupational class is indeed independently related to current smoking, then factors such as job stress and workplace culture and norms remain important targets for efforts to reduce disparities in smoking by occupational class.

In conclusion, blue-collar workers have especially high prevalences of smoking. Furthermore, they are more likely to be heavy smokers, to start smoking earlier, and to be less likely to have quit smoking. Given the documented dose-response relationships regarding early age of initiation and increased intensity of smoking on disease processes [DHHS 1989], blue-collar workers are at especially high risk for smoking-attributable diseases. Appropriate programs need to be developed and implemented with some urgency.

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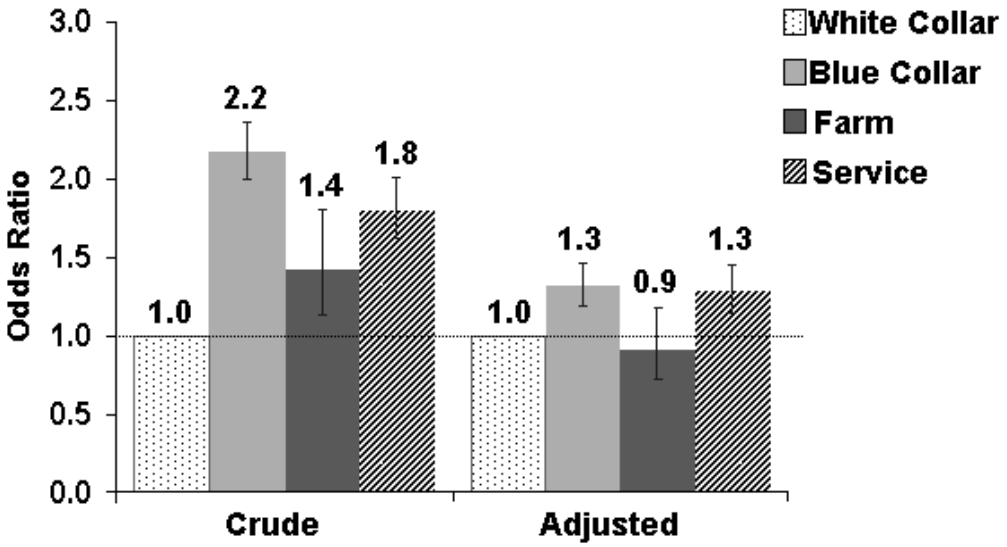
## Figure1: Crude and Adjusted* Odds Ratios for Current Smoking by Occupation Class—United States, 1997




Source: 1997 National Health Interview Survey
*Adjusted for age, sex, race/ethnicity and education. n=19935

CDC

Table 1. **Percentage of employed persons aged ≥18 years who were current smokers,* by occupational class and year of survey – United States, 1978-1980, 1987-1990, and 1997 National Health Interview Surveys†**

| | Men | | | | Women | | | | Overall | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Occupational Class | 1978-80 | 1987-90 | 1997 | Change 1978-80 | 1987-90 | 1997 | Change | 1978-80 | 1987-90 | 1997 | Change |
| White-collar | 32.0(1.2) | 24.0(0.8) | 21.3(1.3) | –10.7(1.8) | 31.4(1.0) | 24.4(0.6) | 20.4(1.0) | –11.0(1.4) | 31.7(0.8) | 24.2(0.4) | 20.8(0.9) | –10.9(1.1) |
| Blue-collar | 45.3(1.2) | 40.2(1.0) | 37.0(1.8) | – 8.3(2.1) | 36.9(2.4) | 34.8(1.6) | 33.8(3.4) | – 3.1(4.1) | 43.7(1.0) | 39.2(0.6) | 36.4(1.6) | – 7.3(1.8) |
| Farm | | 24.8(2.7) | 28.8(5.0) | + 0.9(5.8) | 20.9(6.4) | 15.1(8.4) | 19.8(9.5) | – 1.1(11.4) | 26.7(3.0) | 22.8(2.4) | 27.4(4.4) | + 0.7(5.2) |
| Service | 40.8(2.9) | 36.0(1.8) | 33.1(3.5) | – 7.7(4.6) | 35.2(1.9) | 33.6(1.4) | 31.8(2.6) | – 3.4(3.2) | 37.2(1.5) | 34.5(1.2) | 32.3(2.1) | – 4.9(2.7) |

* For data collected during 1978-1980 and 1987-1990, current smokers were those who reported having smoked ≥100 lifetime cigarettes and who reported smoking now; for data collected during 1997, current smokers were those who reported having smoked ≥100 lifetime cigarettes and who reported now smoking either every day or some days.

† Change is in percentage points. Numbers in parentheses indicate 95% confidence intervals (±)

Table 2. **Percentage of employed persons aged ≥18 years who were current smokers,* by occupational class and race/ethnicity –United States, Current Population Survey, 1995/96 National Cancer Institute Tobacco Supplement**

| | White | | African American | | Asian American/ Pacific Islander | | American Indian/ Alaska Native | |
|---|---|---|---|---|---|---|---|---|
| Occupational Class | % | 95% CI† | % | 95% CI | % | 95% CI | % | 95% CI |
| White-collar | 20.3 | (± 0.3) | 17.4 | (± 0.9) | 15.5 | (± 1.5) | 11.9 | (± 1.3) | 31.0 | (± 4.7) |
| Blue-collar | 37.8 | (± 0.6) | 29.2 | (± 1.4) | 22.9 | (± 1.8) | 24.1 | (± 2.9) | 49.2 | (± 6.1) |
| Farm | | (± 1.7) | 38.1 | (± 7.3) | 22.4 | (± 4.2) | 29.8 | (± 13.2) | 56.3 | (± 19.3) |
| Service | 33.6 | (± 0.9) | 26.7 | (± 1.7) | 18.1 | (± 2.2) | 19.7 | (± 3.4) | 42.1 | (± 7.7) |

* Persons who reported having smoked ≥100 lifetime cigarettes and who reported now smoking either every day or some days.

† Confidence Interval

Table 3. **Percentage of employed persons aged ≥18 years who had started smoking regularly before ages 16 years and 18 years, by occupational class and smoking status – United States, 1997 National Health Interview Survey**

| | Current Smokers* | | | | Former Smokers* | | | |
|---|---|---|---|---|---|---|---|---|
| | Started Before | | | | Started Before | | | |
| | Age 16 Years | | Age 18 Years | | Age 16 Years | | Age 18 Years | |
| Occupational Class | % | 95% CI† | % | 95% CI | % | 95% CI | % | 95% CI |
| White-collar | 23.9 | (± 1.9) | 49.1 | (± 2.2) | 24.6 | (± 2.0) | 48.3 | (± 2.3) |
| Blue-collar | 31.2 | (± 2.6) | 57.9 | (± 2.7) | 35.4 | (± 3.5) | 60.5 | (± 3.6) |
| Farm | 35.4 | (± 9.2) | 55.9 | (± 9.7) | 25.3 | (± 10.1) | 52.8 | (± 11.4) |
| Service | 33.3 | (± 3.8) | 58.7 | (± 3.9) | 32.3 | (± 5.7) | 58.7 | (± 5.8) |

***** Current smokers are persons who reported having smoked ≥100 lifetime cigarettes and who reported now smoking either every day or some days. Former smokers are persons who reported having smoked ≥100 lifetime cigarettes and who reported now smoking not at all.

† Confidence Interval

# Workshop Summary:* Assessing Exposure to Environmental Tobacco Smoke in the Workplace

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*This article is a summary of the Workshop on Environmental Tobacco Smoke Exposure Assessment held 12-13 September 1997 in Baltimore, Maryland

# Introduction

Environmental tobacco smoke (ETS) is a term now widely used to refer to the mixture of sidestream smoke and exhaled mainstream smoke that pollutes air in locations where tobacco smoking is taking place. The health effects of active cigarette smoking have been investigated intensely since the mid-1900s. Substantial evidence has been accumulated on the characteristics of tobacco smoke and on the diseases and other adverse health effects caused by active smoking [DHHS 1989]. Although research on passive smoking—the inhalation of ETS by nonsmokers—began several decades later, there is now substantial evidence on the health effects of passive smoking as well. ETS exposure adversely affects children and adults, causing both malignant and nonmalignant diseases and other adverse health effects [DHHS 1986; EPA 1992a; Scientific Committee on Tobacco and Health 1998]. Although the adverse effects of passive smoking remain a subject of investigation, expert panels and government agencies have concluded that involuntary smoking is a cause of lung cancer and heart disease in adults, as well as other adverse consequences for children. ETS also contains irritant compounds, and its presence reduces the acceptability of indoor air quality [DHHS 1986].

Nonsmokers are exposed to ETS in the home, the workplace, and other locales where smoking is permitted. With increasing restriction in the United States on cigarette smoking in public locations and workplaces, the home is becoming an increasingly dominant locale for exposure. Nonetheless, smoking is still permitted in many workplaces. Because workers spend a substantial proportion of their time at work, ETS exposure in the workplace may pose a risk to the health of workers. Surveys of concentrations of ETS markers in workplaces confirm the occurrence of ETS exposure [Guerin et al. 1992; Hammond et al. 1995]. However, the distribution of exposures and of related health risks has not yet been well characterized in large and representative samples, and consequently, the precise magnitude of risk to workers from ETS exposure is uncertain, although there is agreement that ETS exposure is hazardous [DHHS 1986; EPA 1992a].

In 1994, the Occupational Safety and Health Administration (OSHA) published proposed new regulations on indoor air quality [OSHA 1994]. Although elements of the regulations addressed indoor air quality issues in the workplace in general, specific components of the regulations addressed ETS, and a risk assessment of ETS exposure in the workplace was included. Since preparation of this risk assessment, substantial additional data on ETS exposures in the workplace have become available. Some of these data were presented at hearings on the regulations held by OSHA from 1994 to 1995; other data have been reported in the peer-reviewed literature.

To assess [then] current understanding of ETS exposure in the workplace, the Johns Hopkins University School of Hygiene and Public Health convened a multidisciplinary workshop on September 12–13, 1997. Participants' areas of expertise included building systems, measurement of ETS, biomarkers of ETS exposure, time-activity patterns, and exposure models. Participants were asked to conduct reviews in their specific areas of expertise, relevant to the following general charge: Address key issues related to ETS exposure in the workplace, in order to prepare the groundwork for a risk assessment of the hazard that ETS exposure poses to workers.

The elements of the charge included the following

- Review exposure levels in various workplaces, based on compilation of information from the literature.
- Evaluate issues related to the accuracy and sensitivity of various exposure measurements.
- Review and evaluate available mathematic exposure models for ETS.
- Characterize properties of exposure models with respect to validity in predicting ETS exposure levels in various workplaces.
- Evaluate the chemical and physical properties of various smoke constituents to determine the appropriateness of using them as surrogates for measuring ETS exposure.
- Review and analyze public comments on OSHA's section on exposure of the proposed new rule on indoor air quality.

*This summary of the1997 workshop provides an overview of the discussions held during the two days of that workshop, the general conclusions reached by participants, and research recommendations.*

## Assessing the Risks of ETS Exposure

The ultimate goal of any exposure assessment is to provide an understanding of the risks associated with the exposure. For characterizing risks to a population, the full range of exposure is important; measures of central tendency provide only an indication of overall population exposure, and the upper end of the exposure distribution must be described, particularly at levels that may convey unacceptable risks [EPA 1992b]. Workshop participants reviewed the evidence on ETS exposure in the context of potential uses of this evidence in risk assessment. OSHA faces the task of determining if ETS exposure in the workplace is a significant hazard to workers and also of evaluating the consequences of control measures. Understanding ETS exposure in the workplace and its determinants is essential to accomplish both these tasks.

Workshop participants recognized that ETS exposure in the workplace declined sharply over the last decade as control measures have been implemented, including restrictions on smoking or outright bans on smoking in the workplace. However, the absolute number of exposed workers remains large. Presentations at the workshop on the most recent large-scale workplace surveys [Jenkins published in 1999; Hammond published in 1999] confirmed the impact of nonsmoking policies on ETS exposure. Nonetheless, some jobs and industries continue to involve exposure to ETS, e.g., the hospitality industry, and only limited information is available for certain categories of workers who may still be exposed to ETS, including those working in small workplaces and blue-collar workers. The 16-City Study conducted in 1994 [Jenkins et al. 1996] showed that some workers continue to have substantial exposures to ETS and indicated variation in levels of exposure among different worker groups.

## Overall Conclusions

Participants concluded that substantial evidence was now available on ETS exposures of workers using both the direct and indirect approaches to exposure assessment, and that these data could be used to project the distribution of exposures to ETS in the nation's workplaces. The direct approach determines exposure by actual measurement, through either personal or area monitoring. Biomarker data also provide direct assessment of ETS exposure. The indirect approach, which does not involve directly placing a monitor on a person, relies on mathematic modeling that simulates exposure distributions using a) empirical distributions of exposure in specific microenvironments, b) output from microenvironmental models, and c) human activity pattern data [Klepeis 1999]. The microenvironmental model is central to this approach [NRC 1991]. Microenvironments are locations with homogeneous concentrations of the contaminant of interest during the time of occupancy. In the indirect approach, information on concentrations in microenvironments is used along with time spent in the microenvironments to estimate personal exposure.

Combining data from both direct and indirect approaches, the 16-City Study [Jenkins et al. 1996] offers information on exposures of a large number of nonsmokers to various markers of ETS in indoor air, as well as data on levels of cotinine, a biomarker. Within the specific categories of workers, the data become sparse, but some insight can be gained concerning the shape of the exposure distributions. The 25-Site WorkWell Study of Hammond and colleagues [Hammond et al. 1995] provides additional data; a number of smaller studies are also available. The workshop participants concluded that nicotine, a semivolatile organic compound, is a good tracer for particulate matter from ETS and can be used to measure exposure to ETS as a complex mixture. Cotinine, considered to be an accurate indicator of nicotine exposure [Benowitz 1996], has been measured in participants in the National Health and Nutrition Examination Survey (NHANES) III Pirkle et al. 1996]; these measurements provide nationally representative information on ETS exposure. Two large time-activity surveys — the California study, which characterized activity patterns for a sample of Californians from 1988 to 1990 [Jenkins et al. 1992] and the national study conducted by the U.S. Environmental Protection Agency from 1990 to 1992 [Robinson and Blair 1995] — provide data on the prevalence of workplace exposure to ETS, and the U.S. EPA study provides further detail on the length of exposure and the microenvironments involved.

Mass balance models can be used to predict ETS concentrations in microenvironments [Ott 1999]. These mathematic models use the mass-balance equation, based on the physical law of conservation of mass, to calculate the concentrations in indoor settings from a knowledge of the strength of the source of the contaminant, the volume of the indoor location

into which the contaminant is emitted and diluted, the effective air exchange rate (quantity of replacement air infiltrating per unit time expressed as air changes per hour), and the rate of contaminant loss from paths other than ventilation, (e.g., deposition or chemical reactions). These models predict ETS concentration by combining estimates of the rate of generation of ETS from smoking and the rate of removal by air cleaning and air exchange [Ott 1999]. Recent models have been defined and their performance validated in selected real-world exposure circumstances [Klepeis 1999; Ott 1999; Repace 1998].

The workshop participants agreed that the exposure and time-activity databases and the mass balance models could be used to estimate the distribution of ETS exposure for workers in the United States. Although there are gaps and limitations in the available evidence, ETS exposures in the workplace can now be estimated with far greater certainty than when the initial OSHA risk assessment was prepared approximately 5 years ago. The participants appraised the available evidence and suggested an approach for describing the distribution of ETS exposures, as follows.

Data obtained by the direct approach in the 16-City Study of Jenkins and colleagues [Jenkins et al 1996] are substantial additions to previous evidence. The study provides data on a suite of ETS markers for recent exposure conditions. The results were consistent with the 25-Site WorkWell Study of Hammond and colleagues [Hammond et al. 1995]. It is uncertain whether either data set is representative of all U.S. workers, and by the nature of the approaches used to select subjects, both data sets may tend to under-represent the higher end of the exposure distribution. Both studies, however, offer reasonably robust central estimates of exposure, both overall and for broad categories of workers.

Cotinine was judged to be a valid estimator of exposure of nonsmokers to nicotine. Dietary sources of nicotine are minor and few individuals would ingest sufficient nicotine-containing foods and beverages to compromise the validity of cotinine as an estimator of exposure to nicotine in ETS [Benowitz 1996; Benowitz 1999]. The NHANES III data can be used to estimate nicotine exposure by applying an empirically derived relationship between nicotine intake and cotinine level [Pirkle et al. 1996]. Data reviewed at the workshop suggested that this relationship was relatively robust under current smoking conditions. Therefore, NHANES III, and possibly other data sets, can be used as additional bases for characterizing the national distribution of ETS exposures in the workplace. Available national data on cotinine levels, however, are compromised by the relatively small sample size. These data cannot be expected to provide a sharp picture of the upper end of the exposure distribution.

The time-activity surveys conducted in California, and nationally, draw strength from their large sizes and representative sampling designs. There is the potential to characterize exposure patterns for specific worker groups and, in the national data, exposure duration can be quantified. Both surveys are potentially limited by the need to rely on participant reports of awareness of ETS exposure. It is possible that such reports vary with education, job type, or other factors. The sampling designs of the surveys, based on random digit dialing, may tend to exclude workers at the upper end of the distribution, if such workers are less likely to have telephones or to participate in telephone surveys.

Models for predicting ETS concentrations in microenvironments were further refined during the 1990s. Ott [1999], using the mass balance principle, has developed models incorporating the real-world time dependence of ETS concentrations on smoking patterns and derived parameters for these models under real-world conditions. Model performance was assessed for specific microenvironments, including a tavern, an automobile, and an airport smoking lounge. As reviewed by Ott [1999], model predictions are in good agreement with the actual data. Models can be extended to additional microenvironments, using either assumptions about effective air exchange rates or actual measured values.

Workshop participants proposed an overall strategy for estimating the distribution of workplace exposures to ETS (Figure 1). The suggested approach involves using the parallel and complementary data sets on exposure obtained by the direct method, and using the biomarker data as an additional but distinct approach to estimating national exposure. The principal data sets providing information obtained by the direct method, include the WorkWell [Hammond et al 1995] and Oak Ridge studies [Jenkins et al 1996]. Given the uncertain representation of the nation's workers by these data sets, the NHANES III data [Pirkle et al. 1996] on cotinine levels may provide the most valid national exposure estimates. Within any particular category of workers, information will be quite limited, although for broad classes (e.g., white-collar office workers), the data may be sufficient to provide a picture of the distribution, including high-end distribution.

The time-activity survey data provide additional information on the prevalence of exposure in the early 1990s. By pairing direct exposure data with time-activity information, it may be possible to further estimate exposures for broad classes of workers. Time-activity data provide information on prevalence of exposure, and the directly measured exposures from the Oak Ridge and WorkWell studies can be used as estimates of the likely exposures for specific worker groups.

The time-activity survey data provide additional information on the prevalence of exposure in the early 1990s. By pairing direct exposure data with time-activity information, it may be possible to further estimate exposures for broad classes of workers. Time-activity data provide information on prevalence of exposure, and the directly measured exposures from the Oak Ridge and WorkWell studies can be used as estimates of the likely exposures for specific worker groups.

The mass balance-based models can be used to explore exposure under specific circumstances, such as that of the exposure of a nonsmoker who shares an office with a smoker, or levels of exposure during meetings in locations where smoking is permitted. The models also are useful tools for exploring the protection afforded by various control strategies, including ventilation and air cleaning. With additional data collection on air exchange rates in more complex office environments, the value of these models for assessing control strategies could be enhanced.

## Research Recommendations

Evidence on workplace exposure to ETS continues to mount, but there is still need for research. Workshop participants agreed on the following general recommendations for research on ETS exposure in the United States.

- Conduct larger surveys with the following characteristics:
  - assure representation of all U.S. workers
  - employ the microenvironment approach
  - characterize high-end risk
  - parallel direct approaches with biomarkers.
- Ensure ongoing monitoring through national surveys to enhance information on occupations and exposures.
- Conduct a detailed study of ETS composition and relationship of composition to various markers, with links to doses for a better understanding of the complex mixture of ETS.
- Investigate the gaps in the research, including high-end risk in the hospitality industry, small workplaces, and blue-collar occupations.
- Develop models; validate the models in various workplaces, and develop a time-activity model.
- Identify and respond to research needs regarding biomarkers, including further characterization of cotinine increments from workplace exposures.

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Figure 1.

**An Approach for Estimating Workplace Exposure to ETS**

| **A. Overall Distribution of ETS Exposure** | |
|---|---|
| **Direct Measure** | **Cotinine** |
| Oak Ridge data | NHANES III |
| 25-Site Study | Oak Ridge Study |

| **B. ETS Exposure for Worker Groups** | | |
|---|---|---|
| | $E_i = \sum_{j=1}^{J} t_{ij} c_j$ | $E_i$ = exposure for group I<br>$T_{ij}$ = time in microenvironment j for group I<br>$C_j$ = concentration in j |
| I. | Estimate $t_{ij}$ from California and EPA studies | |
| I. | Estimate $c_i$ from published reports | |

| **C. ETS Exposure for Microenvironments** | |
|---|---|
| | Use mass-balance models |
| II. | Input data on air exchange and air cleaning |

# Summary Workshop:* Health Risks Attributable to ETS Exposure in the Workplace

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*This article is an overall summary of all presentations and discussions from the Workshop on Environmental Tobacco Smoke Risk Assessment, held 9-10 July 1998 in Baltimore, Maryland.

# Introduction

The 1998 workshop was convened to address the health risks of exposure to environmental tobacco smoke (ETS) in the workplace. It was paired with a 1997 workshop on issues related to ETS exposure in work environments [Samet published in 1999a]. In the 1998 workshop, a multidisciplinary group of participants was charged with reviewing evidence on the quantitative risks to health posed by ETS and to discuss development of risk assessment methodology for the future. The overall charges for the 1998 workshop were:

- to consider various health outcomes and make recommendations regarding those health outcomes to be included in assessment of health risk resulting from ETS in the workplace;
- to consider available studies addressing these health outcomes and to evaluate the validity of data for estimating risk from occupational ETS exposure;
- to review and evaluate mathematical models useful for estimating the risk due to ETS exposure;
- to examine dose-response models and to characterize the models regarding validity and uncertainty in estimating health risk attributable to ETS exposure in the workplace.

The 1997 workshop evaluated the accuracy of exposure measurement methods for ETS and the utility of various smoke constituents as surrogates for measuring ETS exposure; it also reviewed and evaluated mathematical models for predicting ETS concentrations [Samet published in 1999b]. In their overall conclusions, the workshop participants set out a general approach for estimating the distribution of ETS exposure in the United States [Samet published in 1999b]. For a quantitative risk assessment to be conducted, information on the ETS exposure distribution should be combined with estimates of the exposure-effect relationships for the health effects of interest [Jaakola and Samet 1999]. OSHA had followed a similar type of general approach in its risk assessment in 1994 of selected risks from ETS exposure in the workplace [OSHA 1994].

The evidence on adverse effects of ETS has steadily increased over recent decades [Samet and Wang, in press]. For adults—the focus of this 2000 workshop—causal associations have now been identified between ETS exposure and lung cancer [DHHS 1986; NRC 1986; EPA 1992; CA EPA 1999; Scientific Committee on Tobacco and Health 1998] and also between ETS exposure and ischemic heart disease [CA EPA 1999; Scientific Committee on Tobacco and Health 1998; Taylor et al. 1992]. Some data indicate other adverse effects of ETS in adults, including increased risk for asthma and respiratory symptoms and reduced lung function level [Samet and Wang, in press], but there is not yet enough evidence to reach conclusions concerning causality. Other health effects linked to ETS exposure of adults include low birth-weight and increased risk for some nonrespiratory cancers [Samet and Wang, in press; CA EPA 1999]. This workshop was primarily concerned with four health outcomes: (1) heart disease and (2) lung cancer were included because a hazard has been identified; (3) asthma was considered because of its high prevalence and the known responsiveness of persons with asthma to inhaled pollutants; and (4) exposure of pregnant women was addressed because of the potential vulnerability of the fetus.

The charges given to workshop participants in relation to the four health outcomes are listed in Table 1. Workshop participants were also asked to address key methodologic issues that arise in interpreting the epidemiologic data on ETS exposure and in summarizing these data with meta-analysis. Confounding has been of particular concern, as exposure to ETS is now associated with lifestyle risk factors in some populations.

Meta-analysis—combining summary estimates from individual studies—has been used to evaluate the hazard posed by ETS and to quantitatively estimate the increased risk associated with exposure. Questions have been raised concerning the use of meta-analysis generally, as well as more specifically, regarding its application to studies of ETS. This general topic was also included in the scope of the workshop.

In this article, we synthesize the information presented in the workshop presentations and in the related discussion. There was no attempt to achieve group consensus on all issues; consequently, this summary should not be construed as necessarily reflecting the views of all participants. The peer-reviewed articles in this monograph are based on presentations at this workshop. These papers as well as the other presentations are summarized in the following sections.

## Exposure Assessment for the Purposes of Health Risk Assessment

The bridge from ETS exposure assessment issues to those of health risk assessment [Jaakola and Samet 1999] was established at the outset of the workshop. Concepts of ETS exposure assessment, relevant for health risk assessment based on human studies, were presented and data on ETS exposure levels in workplaces and residences were reviewed. The sources of variation in exposure, dose, and biologically effective dose of ETS, as well as in individual susceptibility to the health effects, were discussed and a model to describe them was presented [Jaakola and Samet 1999; Jaakola and Jaakola 1997]. A biologically driven approach to select the most appropriate ETS exposure assessment method for assessing health risk was proposed. Special reference was given to the diseases considered in the workshop. This approach accounts for the pathophysiology of the disease and the time specificity of exposure and combines this information with the time period that can be assessed with different exposure assessment methods. For example, an indicator of short-term exposure is appropriate in studies of asthma exacerbation, whereas an assessment of cumulative exposure is relevant for lung cancer.

The workshop emphasized that health risk assessment requires two types of exposure assessment. First, an unbiased estimate of the exposure-effect relation between ETS and the health effect is needed, derived from health effects studies that meet criteria for quality; meta-analysis or pooled analysis can be used to combine data from several studies. Estimates of these relationships were the topic of this [1998]workshop. Second, estimates of the distribution of ETS levels in workplaces are needed if occupational hazards due to workplace ETS is the focus of the risk assessment. These estimates were the focus of the 1997 workshop.

There is no biologic or scientific basis for expecting that the effects of ETS exposure in the workplace would differ from the effects related to home ETS exposure, if the exposure is of equal magnitude. Workplace exposures likely are more variable than residential exposures because of larger variability in: (a) size and ventilation characteristics of workspaces, (b) number of smoking co-workers, and (c) smoking policies in different workplaces. Nonetheless, median and mean indoor air concentrations of ETS markers, especially nicotine and respirable suspended particles, have been found to be essentially comparable between workplace and residential environments in the United States as well as in other countries [Jaakola and Samet 1999; EPA 1992; Guerin et al. 1992; Hammond 1999]. Some work forces, however, such as hospitality workers, may be exposed to high levels of ETS that are rarely encountered in residential settings.

Also at issue was how questionnaire-based risk estimates should be combined with ETS marker measurements used to assess the exposure distribution when assessing the proportion of disease cases attributable to workplace ETS exposure. Some estimates of the relationship between questionnaire-based assessment of exposure and indoor ETS marker concentrations have been provided by experimental and field studies [Repace et al. 1998; Klepeis 1999a; Klepeis 1999b; Ott 1999; Leaderer and Hammond 1991]. More research is recommended, however, to achieve more precise estimates of these relationships under different environmental conditions.

### Cardiovascular Diseases

The charges concerning cardiovascular diseases were broad (Table 1). The evidence on spouse's smoking and heart diseases, as well as the studies of workplace exposure in particular, were reviewed and key methodologic questions including study population selection, exposure misclassification, and confounding were addressed. In addition, data on biologic mechanisms were discussed. Usefulness of meta-analysis in estimating coronary heart disease risk from workplace ETS exposure and possibilities to model exposure-effect relationships were also addressed.

Chappell [1998] presented a thorough discussion on the use of meta-analysis in estimating coronary heart disease risk from workplace ETS exposure. Advantages as well as potential problems in applying this analytical technique to the available data were discussed. Further suggestions on how to improve the quality of the risk estimates by adjusting for duration and intensity of exposure to better reflect workplace conditions were offered. As an alternative to meta-analysis for estimating occupational risk, Chappell also suggested the use of a stochastic approach, where distributional information based on the available studies rather than simply on point estimates is considered.

The relationship of ETS exposure to subclinical measures of the development of atherosclerotic disease was addressed in a separate presentation. B-mode real-time ultrasound can be used to estimate the extent of atherosclerosis noninvasively, offering the possibility of measuring subclinical markers of disease; the intimal-media thickness of the carotid artery has been used as an index of systemic atherosclerosis. Howard and Wagenknecht [1999] described cross-sectional and longitudinal findings from the Atherosclerosis Risk in Communities (ARIC)

study, which linked ETS exposure to both greater thickness and an accelerated rate of increase in thickness of the intimal-media. This finding implies that ETS exposure accelerates the process of atherosclerosis. Other measures of subclinical disease considered in relation to ETS exposure included decreased endothelial function and silent cerebral infarction.

Several presentations covered the evidence on ETS exposure and heart disease risk. Thun and colleagues [1999] carried out a meta-analysis of 17 studies, 9 cohort and 8 case-control, on the risk of ischemic heart disease for nonsmokers married to smokers. The evidence, which encompassed more than 485,000 lifelong nonsmokers and 7,345 events, was substantially more extensive than 5 years earlier when OSHA conducted its risk assessment, using the estimate from only one study conducted in Washington County, Maryland [Sandler et al. 1989]. The meta-analysis provided an overall estimate of relative risk of 1.25 (95% confidence interval [CI], 1.17-1.33).

Together, Howard and Thun [1999] considered the various types of bias considered to be potential explanations for the association of ETS with ischemic heart disease. These primarily include confounding and information bias. There has been concern that bias may at least partially explain the association because the relative risk has been considered disproportionately elevated in relation to relative risk values for active smoking. Their analysis indicated that the association cannot be readily explained by bias. Additionally, they described effects of acute exposure to ETS that provide insight concerning mechanisms that may underlie the association of ETS with cardiovascular disease.

Information available on risk attributable to workplace exposure was specifically addressed in two presentations. Kawachi and Colditz [1999] summarized the available evidence from 5 studies, 3 case-control and 2 cohort. The point estimates of relative risk in the individual studies ranged from 1.2 to 1.9, but none of the estimates were statistically significant. It was stated that because of the imprecision of the risk estimates in all but one study, a modest increase in cardiovascular disease risk from workplace exposure to ETS could not be excluded. Additionally, in contrast to the evidence on spousal exposure to ETS and increased risk of cardiovascular disease, studies of workplace ETS exposure are still sparse and further research is needed. Steenland [1999] proposed an approach for conducting a quantitative risk assessment of workplace ETS. His approach uses a relative risk estimate derived by meta-analysis. This method leads to an estimate of approximately 340 excess ischemic heart disease deaths per year among nonsmoking U.S. workers age 35 to 70 years.

**Lung Cancer**

A causal role of ETS in induction of lung cancer is strongly established [NRC 1986; EPA 1992]. Biologic plausibility is derived from the fact that ETS contains the same carcinogenic compounds as mainstream smoke inhaled by active smokers. The workshop charges on workplace ETS exposure and lung cancer included a review of estimates of lung cancer risk associated with ETS exposure, with emphasis on workplace ETS exposure. Several contributors reviewed potential sources of bias and confounding in studies of ETS and lung cancer as well as the methods that have been applied in the studies to reduce their impact. Finally, different modeling approaches to assess the lung cancer risk related to workplace ETS exposure were reviewed.

Over 40 studies have examined the relationship between spousal smoking and risk of lung cancer. Many of them have provided evidence of an exposure-response relationship with the number of cigarettes smoked by the spouse and/or with the duration of ETS exposure at home or in the workplace [Hackshaw et al. 1997; Reynolds 1999]. The risk related specifically to workplace ETS exposure has been studied in women in 16 hospital-based or population-based case-control studies and in men in 7 hospital-based or community-based case-control studies and in 1 cohort study [Reynolds 1999; Alavanja 1998]. Most of the studies were not explicitly designed to evaluate the association between workplace ETS and lung cancer risk and consequently had low power to detect a statistically significant relationship [Reynolds 1999]. In general, the risk estimates appear to be consistent with those for exposure from a smoking spouse. Recent studies have had larger sample sizes and addressed many of the potential sources of bias. These studies have shown a statistically significant increase in lung cancer risk related to workplace ETS and have provided evidence of increasing risk with increasing duration of workplace ETS exposure [Fontham et al.1994; Kabat et al.1995; Reynolds et al. 1996; Bofetta et al. 1998].

A pooled analysis or meta-analysis of individual studies can provide a useful approach to combine data from small, individual studies to assess the risk. A meta-analysis is often a more feasible approach, but it does not provide an opportunity to assess heterogeneity of risk within subgroups, whereas a pooled analysis of raw data from studies usually offers greater flexibility in modeling [Lubin 1999]. Approaches to use the relatively large body of data on the

exposure-response relationship between lung cancer risk and spouse's smoking to assess risk related to workplace ETS were discussed [Lubin 1999; Brown 1999]. The exposure-response relationship between lung cancer and ETS exposure due to spousal smoking among nonsmoking women, using the number of cigarettes smoked by the husband as an indicator for the amount of exposure, was evaluated using a log-linear model by Brown [1999]. A total of 14 studies contributed data to this analysis. The model for all countries combined predicted an excess risk of lung cancer of 17% per 10 cigarettes/day (95% CI, 12-22%), and the excess risk from the United States alone was 13% per 10 cigarettes/day (95% CI, 5-21%). On the basis of data on smoking habits in the two large cancer prevention studies by the American Cancer Society [Stellman and Garfinkel 1986; Steenland et al. 1996], the average number of cigarettes smoked daily by U.S. men was about 24 [Brown 1999]. Applying this value to the model, the average excess risk of lung cancer in nonsmoking women due to spousal smoking was 33% (95% CI, 14-56%). According to serum cotinine measurements in a large national survey of the U.S. population [Pirkle et al. 1996], the workplace ETS exposure of nonsmoking women is on average 42% of the home exposure of nonsmoking women. This corresponds to an average exposure of 10 cigarettes/day [Brown 1999]. When this average workplace ETS exposure was used to assess the excess risk of lung cancer in nonsmoking women, an estimate of 13% was obtained. After adjustment of the estimate for ETS exposure in the reference group for sources other than the workplace exposure, the estimated excess risk is 19% (95% CI, 10-28%).

As alternative approaches, one can model lung cancer risk in current and former smokers and extrapolate the results to low levels corresponding to ETS exposures or one can model restricted data from light smokers [Lubin 1999]. The latter approach is more directly applicable to the range of exposures comparable with typical ETS exposure settings. The modeling based on data in current and former smokers should first adjust for ETS exposure in the reference group so that the effect estimate is not diluted by increased risk among ETS exposed nonsmokers. The exposure-risk patterns from light active smoking models do not indicate a threshold level below which exposures would not be expected to increase the risk of lung cancer [Lubin 1999]. Average ETS exposure in nonsmokers has been estimated to be the equivalent of actively smoking 0.1–2 cigarettes/day [EPA 1992; Lubin 1999]. The risk ratio for smoking 0.5 cigarettes/day ranges between 1.1 and 1.3 in models restricted to light smokers only [Lubin 1999]. The risk estimate of lung cancer in relation to ETS exposure is surprisingly consistent regardless of whether the modeling approach uses data on active smoking and extrapolates the estimates to low levels or whether more direct data on passive smoking are used.

Potential biases that might affect the risk estimates from lung cancer studies were discussed extensively by workshop participants [Reynolds 1999; Alavanja 1998; Wu 1999; Matanoski 1998]. Hospital-based case-control studies may be weakened by a selection bias if recruitment of cases or controls is related in some way to ETS exposure. In recent years, several population-based case-control studies have been conducted to avoid this type of bias [Reynolds 1999]. In epidemiologic studies, some degree of misclassification of exposure and of outcome is likely to occur. In many of the studies, ETS exposure was classified on the basis of smoking by the spouse only, while not capturing exposure from other sources, although as adults people are usually exposed from multiple ETS sources, including workplace and social settings. Consequently, the reference category classified as unexposed may include persons who have experienced substantial ETS exposure, thus diluting the obtained risk estimates [Wu 1999].

In case-control studies, use of proxy respondents (usually a family member) to give information on exposures for lung cancer cases who are very ill or deceased may lead to greater misclassification of ETS exposure among cases compared to controls [Reynolds 1999; Alavanja 1998]. However, information on ETS exposure variables reported by surrogate respondents has been found to agree closely with that reported by the lung cancer cases themselves. Studies comparing results in total study populations with analysis limited to cases interviewed in person have shown essentially similar results [Reynolds 1999; Alavanja 1998].

Recall bias can affect risk estimates if cases (or surrogates) consider ETS exposure as a possible explanation for the disease [Reynolds 1999]. The U.S. multicenter case-control study included controls drawn from the general population and controls recruited among patients with primary colon carcinoma [Fontham et al.1991]. The latter control group was expected to be searching for an explanation for their cancer in the same manner as lung cancer cases. The results were consistent in case-control comparisons regardless of the control group used.

Misclassification of self-reported smoking status is a concern, as some current and former smokers may report that they are “never smokers” whereas they are at higher risk for lung cancer because of their smoking [WU 1999]. The proportion of ever smokers (current or former smokers) misclassified as never smokers has been estimated to be small: 3–7% [EPA 1992; Hackshaw et al. 1997; Nyberg et al. 1997]. On the basis of measurements of urinary cotinine with a cut-off point of 50 ng/mg creatinine to indicate active smoking, the proportion of active smokers

among reported never smokers was between 3 and 5% [Fontham et al. 1994; Riboli et al. 1995]. Most of the ever smokers misclassified as never smokers have quit smoking and had smoked fewer cigarettes than an average smoker [Wu 1999]. In addition, although smoking spouses tend to marry each other, this type of differential misclassification of the smoking status does not appear to be likely with respect to workplace ETS exposure.

Women nonsmokers living with smokers have been shown to differ from those living with nonsmokers with respect to such disease risk factors as lifestyle and socioeconomic status [Matanoski 1998]. Whether the same factors are associated with workplace ETS exposure is unclear. Most studies published since the mid-1980s have adjusted for important potential confounders, although the set of confounders has differed among the studies. Most have accounted for some indicator of socioeconomic status, which can be seen as a surrogate for many lifestyle factors, but the possibility of residual confounding exists. Several studies have adjusted for exposure to other occupational hazards and for dietary habits, including low fruit and vegetable consumption and high saturated fat intake, and have shown negligible confounding by these factors of the relationship between ETS exposure and lung cancer risk [Hackshaw 1997; Reynolds 1999; Alavanja 1998; Brown 1999]. Modification of effects of ETS by other occupational and indoor exposures has not been studied [Alavanja 1998]. Potential synergism between these exposures and ETS should be studied to evaluate whether ETS is especially harmful in the workplace.

**Asthma**

Strong evidence exists for a causal role for ETS in the development and exacerbation of asthma in children [EPA 1992; CA EPA 1999]. However, there are only a few studies on ETS and adult asthma. For asthma, the charges for the workshop participants were to review data on the role of ETS in causing asthma in adults, with special emphasis on workplace exposure and to review data on effects of ETS on exacerbation of asthma in both epidemiologic and controlled exposure studies [Weiss et al. 1999].

Only four studies of ETS and adult onset asthma were identified [Greer et al.1993; Leuenberger et al.1994; Flodin et al.1995; Hu et al.1997]. Two longitudinal, one case-control, and one crosssectional studies all indicated that the risk of adult asthma is increased in relation to ETS exposure in general and in some instances specifically in relation to workplace exposure. Some methodological problems of the studies were identified including potential recall bias of exposures and bias in selection of study subjects [Weiss et al. 1999]. Only one epidemiologic study had addressed the effects of ETS exposure on exacerbation of asthma in adults and found increased emergency room visits, hospitalizations, medication use, and absence from work among ETS-exposed asthmatics, compared with unexposed asthmatic subjects [Jindal et al. 1994]. In this study, retrospective assessment of both ETS exposure and outcomes raises the question of potential recall bias. The published epidemiologic studies suggest that ETS contributes both to development and exacerbation of asthma in adults, but definite conclusions cannot be reached because of a limited number of studies and potential problems in their design.

More relevant data are available from controlled exposure studies in asthmatic and healthy volunteers [Weiss et al. 1999]. The results have been quite inconsistent, probably because of different selection criteria of subjects, small study samples, and different exposure periods. However, there is evidence of a subgroup of asthmatics who are sensitive to ETS and respond to ETS exposure with symptoms, reduction in lung function, and increase in bronchial hyper-responsiveness. The determinants of this susceptibility are not known.

The available, although limited, literature on ETS in adults suggests that ETS may have a significant impact on exacerbation of asthma in adults, and further clinical and epidemiologic studies paying special attention to design issues are needed.

**Low Birth-Weight**

For low birth-weight, the charge for the workshop participants was to consider the full spectrum of the evidence on ETS and the growth of the fetus and to address the applicability of data from outside the workplace to pregnant women working outside the home.

Active smoking during pregnancy has been causally associated with reduced birth-weight. Misra and Nguyen [1999] considered ETS exposure and reduced birth-weight. For women of child-bearing age, the workplace is a particularly important locus of exposure. The literature on ETS exposure generally shows associations with adverse health effects including reduced birth-weight. These findings were considered applicable in the workplace setting.

## Concluding Remarks

This workshop addressed four outcome measures in relation to workplace exposure to ETS: lung cancer, ischemic heart disease, asthma, and low birth-weight. The focus was on approaches for deriving quantitative risk estimates for workplace ETS exposure. In addition, cross-cutting issues were addressed including sources of bias that may have affected risk estimates and the use of meta-analysis and mathematical modeling for synthesis of the evidence. Although bias from confounding and exposure misclassification is a concern, the workshop participants found little evidence that estimates of ETS risk are substantially affected by bias.

For lung cancer, data from three types of studies are available that can be used to estimate risk related to ETS exposure: (a) active smoking, particularly at lower levels of daily consumption, (b) ETS exposure at home, and (c) ETS exposure at work. Mathematical models for lung cancer risk from active smoking can be developed and then applied with extrapolation to ETS exposure levels. Estimates from studies of ETS exposure in the home can be extended to the workplace by considering the relative exposures in the two types of locations. Finally, several studies, particularly the large U.S. multicenter study [Fontham et al. 1994], provide risk estimates based directly on reported workplace exposure.

For ischemic heart disease, there are now numerous reports of the increased risk associated with ETS exposure at home. Evidence is far more limited on ETS exposure at work. However, a meta-analysis approach was presented for estimating the hazard posed to workers by increased ischemic heart disease risk from ETS exposure.

For asthma and low birth-weight, only limited evidence on workplace exposure to ETS was available. Persons with asthma and pregnant women are groups considered susceptible to effects of inhaled pollutants in general. For children, ETS exposure at home is well characterized as exacerbating asthma and increasing medical morbidity. An effect of workplace exposure on adults with asthma is plausible, and experimental studies show that some asthmatics are sensitive to ETS. However, we need further investigation on workplace exposure to ETS as a risk factor for exacerbation of asthma.

Active smoking by the mother reduces infant birth-weight, as does ETS exposure at home, although to a much lesser degree than active maternal smoking. On a biologic basis, ETS exposures at home and at work would be expected to have the same consequences for birth-weight. Consequently, exposure of pregnant women to ETS in the workplace was considered an outcome of concern, while needing further investigation.

In combination, the two workshops—the 1997 workshop on exposure assessment and the 1998 workshop on health outcomes—provide a framework for assessing the risks of ETS exposure in the workplace. An approach for assessment of ETS exposure was proposed in the 1997 workshop. The 1998 workshop added the needed complement of dose- response assessment for lung cancer and cardiovascular disease.

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Table 1.

**Charges for the Specific Health Outcomes**

| | |
|---|---|
| Exposure Assessment | • What are important exposure assessment issues for health risk assessment based on human studies of workplace ETS exposure?<br>• Are there data available on markers of ETS exposure in the workplace?<br>• Are there data on comparative levels of ETS exposure at home and work to support extrapolation from studies of spousal smoking? |
| Cardiovascular Disease | • Has workplace exposure to ETS been investigated as a risk factor for coronary heart disease and other cardiovascular diseases? Are quantitative risk estimates available.<br>• Can the data on spouse smoking and coronary heart disease risk be extended to workplace exposure?<br>• Does the "healthy worker effect" have an impact on estimates of cardiovascular disease risks arising from workplace ETS exposure?<br>• Are there risk estimates that control for potential bias due to (1) misclassification of smoking status (some reported nonsmokers are actually smokers); and (2) confounding by lifestyle, e.g., diet and exercise?<br>• Are there data available on biologically plausible mechanisms? |
| Lung Cancer | • Are risk estimates available for workplace exposure? What is the precision of such estimates? What are the sources of uncertainty in extending them to current levels of exposure?<br>• Can the evidence on risks associated with spouse smoking be extended to the workplace?<br>• Are there any models of carcinogenesis useful for estimating workplace risks of ETS exposure? |
| Asthma | • Are there data supporting a role for ETS in causing asthma in adults? Are there data available related to workplace exposure?<br>• Has ETS exposure been shown to exacerbate asthma in adults? What are the findings of exposure of volunteers with asthma to ETS? Are there epidemiological data available on this issue? |
| Low Birth-Weight | • What is the dose-response relationship for ETS exposure and low birth-weight? Are exposures in the workplace in a range of biologic concern?<br>• Are there studies of occupational exposure to ETS and birth-weight?<br>• Can results from studies of birth-weight and ETS exposure generally be extended to the workplace? |

# Environmental Tobacco Smoke in the Workplace: Trends in the Protection of U.S. Workers

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## Introduction

In January 1971, Surgeon General Dr. Jesse Steinfeld urged that smoking be restricted in public places including worksites [Steinfeld 1972]. Dr. Steinfeld argued that the strong dose-response relationship observed between active cigarette smoking and disease suggested that even the small dose of smoke that nonsmokers received could be hazardous. Active smokers, he reasoned, experienced lung cancer death rates that were between 10 and 20 times greater than those experienced by lifelong nonsmokers [DHEW 1971]. Thus, it was biologically plausible that nonsmokers who lived and worked with smokers probably received a dose of cigarette smoke sufficient to place them at increased risk for lung cancer and other diseases.

Scientific research continued to be reported after 1971, and by 1986, the health risks of exposure to environmental tobacco smoke (ETS) were conclusively documented. Separate reports issued that year by the U.S. Surgeon General [DHHS 1986] and the National Academy of Sciences [NRC 1986] concluded that ETS was a cause of lung cancer and respiratory disease in nonsmokers. The Surgeon General's report also cautioned that the simple separation of smokers and nonsmokers within the same air space could reduce, but not eliminate, nonsmoker exposure to ambient tobacco smoke. Subsequent reports [EPA 1992; CA EPA 1999] confirmed and extended these findings. It is now generally accepted that ETS is responsible for approximately 30,000 to 60,000 nonsmoker deaths each year in the United States.

As the scientific evidence linking ETS exposure to a host of diseases continued to accumulate, the general public became more vocal in its demand for smoke-free public accommodations [Gallup 1987]. In 1988, smoking was banned on virtually all domestic airlines [Shopland et al. 1990]. All international flights entering the United States became smoke-free in 2000 [Associated Press 2000]. Smoking is either not permitted or severely restricted in shopping malls, health care facilities, hotels and motels, schools, sporting events, restaurants, and in bars in some jurisdictions. However, it is the American worksite that has received the most attention regarding smoking restrictions [Gerlach et al. 1997]. This paper examines trends in workplace policies that protect workers from exposure to ETS.

## The Current Population Survey

The Current Population Survey (CPS) has been conducted monthly by the Bureau of the Census since 1940. The CPS focuses on labor force indicators for the civilian noninstitutionalized U.S. population, aged 15 years and older. In 1992, a 40-item Tobacco Use Supplement was developed by National Cancer Institute staff and pre-tested by trained Census Bureau interviewers prior to full field implementation. Among other measures, questions on tobacco use practices, presence and characteristics of workplace smoking policies, rules about smoking in the home, and attitudes toward smoking restrictions in public places were added to the CPS for the September 1992 survey and continued in January 1993 and in May 1993. The Supplement questions were repeated in the September 1995, January 1996, and May 1996 surveys [NCI 2000]. A third round of the Tobacco Use Supplement was conducted by the Census Bureau in September 1998 and in January and May 1999. In the text and tables that follow, these survey results are often grouped and referred to simply as 1993, 1996, and 1999.

The data presented here reports on results from the 1992–93, 1995–96, and 1998–99 CPS. Results from the 1998–99 CPS are preliminary.

*CPS Methodology* – The complete CPS methodology is published elsewhere [Hansen 1978; U.S. Dept of Commerce 2000]. The CPS sample is based on household addresses. The three main sources are households listed in the most recent decennial census, updated building permits, and area sampling where no address lists exist from the Bureau of the Census. Individuals eligible for CPS interviews are the civilian noninstitutionalized population of the United States 15 years of age and older. [However, information for respondents 15 years of age is not available from the January 1996 CPS]. For the purposes of this paper, however, data are presented for adults ages 18 years or older at the time of the interview. All strata are defined within state boundaries, and the sample is allocated among the states to produce state and Census region and division, as well as national labor force estimates.

Normally for the CPS, interviews are conducted with a knowledgeable household respondent who reports for all eligible household members. Typically, slightly more than half of all interviews are self-respondents, and the

remainder are proxy [Marcus et al.1989a; Marcus et al.1989b]. For the NCI Tobacco Use Supplement, however, the 1992–93 Supplement questionnaire was administered on a form separate from the standard labor force core questionnaire, in order to minimize the proportion of proxy responses. This procedure reduced proxy responses to under 20%. For this analysis, only self-responses were utilized. In 1995, the Census Bureau began administering the CPS using a Computer Assisted Personal Interviewing (CAPI) technique; all responses to questions are recorded directly into pre-programmed computer software.

Response rates to the CPS are typically around 95%, and the corresponding response rates for the Tobacco Use Supplement range from 84–89% of those responding to the CPS core. All initial household contacts for the CPS were done in person. However, approximately 75% of all interviews were conducted by telephone and 25% in person. For Spanish language interviews, a fully translated Tobacco Use Supplement was utilized. In all cases where estimates for whites and blacks are presented, they are non-Hispanic [Gerlach et al. 1997; NCI 2000].

*Determination of Smoking Status* – Questions for determining smoking status are identical to those adopted in 1992 by the National Center for Health Statistics and currently in use on other national surveys. All respondents were asked, "Have you smoked at least 100 cigarettes in your entire life?" Those responding "no" were considered never smokers; those responding "yes" were asked, "Do you now smoke cigarettes every day, some days, or not at all?" Respondents answering either "every day" or "some days" were considered current smokers; those responding "not at all" were considered former smokers.

*Occupational and Workplace Definitions* – Labor force questions from the CPS core were used to determine each respondent's employment status (including those who were self-employed) and to categorize each worker into a standard occupational group. In the CPS, there are 500 employment categories, which the Census Bureau aggregates into 45 detailed groups. The classification uses a three-digit system (000-905) developed from the 1980 Standard Occupational Classification [U.S. Department of Commerce 1994].

Because NCI was primarily interested in measuring the extent of official workplace smoking policies for indoor working environments, additional questions from the Tobacco Use Supplement were used to identify eligible employees. To be included in the analysis, an individual must have been (a) 15 years of age or older at the time of interview; (b) currently employed outside the home, but not self-employed; (c) not working outdoors or in a motor vehicle; (d) not traveling to multiple buildings or sites; and (e) not working in someone else's home.

All eligible respondents were queried, "Does your place of work have an official policy that restricts smoking in any way?" Those who responded "yes" were further asked, "Which of these best describes your place of work's smoking policy for indoor public or common areas, such as lobbies, rest rooms and lunch rooms?" and "Which of these best describes your place of work's smoking policy for work areas?" Response choices for each were: "Not allowed in ANY....Allowed in SOME....[or] Allowed in ALL...."

Based on this algorithm, the most restrictive policies were those in which the worker reported an official workplace policy that did not allow smoking in any public or common areas of the workplace nor in the work areas of the workplace. Beginning in 1995, the question "During the past two weeks has anyone smoked in the area in which you work?" was asked of workers in an effort to gauge the level of compliance with workplace smoking policies which did not permit smoking in the work area.

Attitudes Toward Smoking Restrictions in Public Places – The NCI was also interested in measuring the general public's attitudes and beliefs about smoking restrictions in various public settings. The question asked, "In ( place ) do you THINK that smoking should be allowed in all areas, allowed in some areas, or not allowed at all?" Six specific indoor settings were identified: restaurants, hospitals, indoor work areas, bars and cocktail lounges, indoor sporting events, and indoor shopping malls.

## Results

In 1986, less than 3% of workers reported that smoking was banned in their place of employment. This figure rose to 46% in 1992–93, and by 1995–96 had increased to 63% of all U.S. indoor workers. Preliminary data from the 1998–99 CPS suggests a further increase to 69%. The adoption of smoke-free workplace rules increased markedly in the 10-year period from 1986–96, but the rate of increase appears to have slowed recently. Nonetheless, nearly 7 out of every 10 U.S. workers are now covered by a policy which protects them from ETS exposures on the job.

## Summary

In 1992, the National Cancer Institute sponsored a Tobacco Use Supplement to the Census Bureau's Current Population Survey [U.S. Department of commerce 1994] which included a series of questions about official worksite smoking policies. NCI repeated the supplement in 1995–96 and again in 1998–99. The important finding from these survey are:

1) In 1998–99, nearly 7 out of 10 indoor workers across the United States reported that smoking was not permitted in work areas and the public or common areas of their place of employment. In 1986, the corresponding figure was just three.

2) Smoke-free policies at work cover only about 50% of working teens, compared to nearly 70% of the adult workforce.

3) Smoke-free workplace policies cover almost 75% of all white-collar workers, compared to 52% of service workers, and only 45% of blue-collar employees.

4) Women are significantly more likely than men to report working in a smoke-free workplace.

5) Considerable variation exists among the states in the United States, with respect to worksite smoking policies. In 1995–96, Utah and Maryland had the highest rates of coverage, with >80% of the indoor workforce reporting a smoke-free workplace, while in Arkansas, Kentucky, and Nevada, <50% of the workforce indicated that they were covered by smoke-free policies.

Significant progress has been made in the United States, with respect to workplace smoking restrictions, especially the proportion of workers now covered by an official smoke-free workplace policy. As the scientific evidence mounts documenting the adverse health effects of ETS among non-smokers, employees, unions, and worksite managers have increasingly demanded a smoke-free work environment. However, large numbers of workers, including teenagers, blue-collar and service workers, still remain at risk from the established hazards of ETS.

# Tables 1 & 2

*Smoke-free policies – Among the major racial and ethnic groups in 1995–96, the proportion of Native-Americans working under smoke-free policies was lowest, for both males and females (Table 3). Few significant differences exist in smoke-free workplace coverage for White, African-American, and Hispanic workers among either men or women.*

Table 1

**Indoor workers covered by various workplace policies among U.S. males, Current Population Survey 1993 and 1996, ages 15 and older and 95 % CI**

| Age | Smoke-free % | 95 % CI | No smoking work area only % | 95 % CI | Smoking allowed % | 95 % CI | No official policy % | 95 % CI |
|---|---|---|---|---|---|---|---|---|
| **Males 1993** | | | | | | | | |
| 15-19 | 28.2 | 2.1 | 27.8 | 2.1 | 16.6 | 1.7 | 27.4 | 2.1 |
| 20-24 | 34.6 | 1.5 | 22.4 | 1.3 | 17.4 | 1.2 | 25.5 | 1.4 |
| 25-34 | 38.9 | 1.0 | 20.7 | 0.8 | 18.6 | 0.8 | 21.8 | 0.8 |
| 35-44 | 42.3 | 1.0 | 18.7 | 0.8 | 19.6 | 0.8 | 19.3 | 0.8 |
| 45-54 | 43.9 | 1.3 | 18.8 | 1.0 | 19.8 | 1.0 | 17.5 | 1.0 |
| 55 + | 40.6 | 1.7 | 18.2 | 1.3 | 19.0 | 1.3 | 22.2 | 1.4 |
| Total | 39.8 | 0.5 | 20.2 | 0.4 | 18.9 | 0.4 | 21.2 | 0.4 |
| **Males 1996** | | | | | | | | |
| 15-19 | 47.5 | 2.3 | 14.5 | 1.6 | 13.4 | 1.6 | 24.6 | 2.0 |
| 20-24 | 49.7 | 1.7 | 15.1 | 1.2 | 13.2 | 1.1 | 22.0 | 1.4 |
| 25-34 | 57.0 | 1.0 | 13.1 | 0.7 | 13.2 | 0.7 | 16.7 | 0.8 |
| 35-44 | 59.0 | 1.0 | 12.6 | 0.7 | 14.1 | 0.7 | 14.3 | 0.7 |
| 45-54 | 61.1 | 1.2 | 11.2 | 0.8 | 13.9 | 0.9 | 13.8 | 0.9 |
| 55 + | 60.6 | 1.7 | 10.1 | 1.0 | 12.8 | 1.1 | 16.5 | 1.3 |
| Total | 57.4 | 0.5 | 12.6 | 0.4 | 13.6 | 0.4 | 16.5 | 0.4 |

Table 2

**Indoor workers covered by various workplace policies among US females, Current Population Survey 1993 and 1996, ages 15 and older and 95 % CI**

| Age | Smoke-free % | 95 % CI | No smoking work area only % | 95 % CI | Smoking allowed % | 95 % CI | No official policy % | 95 % CI |
|---|---|---|---|---|---|---|---|---|
| **Females 1993** | | | | | | | | |
| 15-19 | 35.3 | 2.1 | 23.1 | 1.9 | 14.4 | 1.6 | 27.2 | 2.0 |
| 20-24 | 46.3 | 1.5 | 19.8 | 1.2 | 12.8 | 1.0 | 21.1 | 1.2 |
| 25-34 | 50.9 | 1.0 | 19.4 | 0.8 | 13.3 | 0.7 | 16.3 | 0.7 |
| 35-44 | 54.8 | 1.0 | 18.9 | 0.8 | 12.2 | 0.6 | 14.1 | 0.7 |
| 45-54 | 54.1 | 1.2 | 19.7 | 1.0 | 12.3 | 0.8 | 14.0 | 0.8 |
| 55 + | 50.9 | 1.6 | 20.1 | 1.3 | 12.0 | 1.0 | 16.9 | 1.2 |
| Total | 51.2 | 0.5 | 19.6 | 0.4 | 12.7 | 0.3 | 16.5 | 0.4 |
| **Females 1996** | | | | | | | | |
| 15-19 | 53.6 | 2.1 | 13.7 | 1.5 | 10.8 | 1.3 | 21.9 | 1.8 |
| 20-24 | 62.0 | 1.6 | 11.9 | 1.0 | 8.8 | 0.9 | 17.2 | 1.2 |
| 25-34 | 67.6 | 1.0 | 11.2 | 0.6 | 8.9 | 0.6 | 12.2 | 0.7 |
| 35-44 | 70.7 | 0.9 | 10.8 | 0.6 | 7.9 | 0.5 | 10.6 | 0.6 |
| 45-54 | 72.9 | 1.0 | 10.1 | 0.7 | 7.6 | 0.6 | 9.5 | 0.7 |
| 55 + | 68.7 | 1.5 | 9.5 | 0.9 | 7.4 | 0.8 | 14.4 | 1.1 |
| Total | 68.2 | 0.5 | 10.9 | 0.3 | 8.3 | 0.3 | 12.5 | 0.3 |

Table 3

*Coverage by type of worker – Smoke-free workplace policies varied significantly by occupation. White-collar workers reported much greater levels of protection from ETS than either blue-collar or service workers. All groups showed gains in coverage by smoke-free policies, with blue-collar workers experiencing the greatest relative increase (relative change between 1992–93 and 1995–96 was: white-collar + 33%; blue-collar + 61%; and service workers + 46%).*

**Indoor workers covered by smoke-free* worksite policies, by gender and race-ethnicity, ages, 15 years and older and 95 % CI Current Population Survey 1993 and 1996**

| | 1993 | | 1996 | |
|---|---|---|---|---|
| **Gender**<br>**Race-ethnicity** | **%**<br>**Smoke-free** | CI | **%**<br>**Smoke-free** | CI |
| **Males**<br>White | 40.0 | 0.6 | 57.3 | 0.6 |
| Hispanic | 38.8 | 2.4 | 54.0 | 2.4 |
| African-American | 36.6 | 1.7 | 58.4 | 1.7 |
| Asian-PI | 46.7 | 2.9 | 64.5 | 2.6 |
| Native American | 34.7 | 7.5 | 48.0 | 6.8 |
| **Females**<br>White | 51.5 | 0.6 | 68.7 | 0.5 |
| Hispanic | 51.1 | 2.7 | 65.9 | 2.4 |
| African-American | 47.6 | 1.5 | 67.0 | 1.4 |
| Asian-PI | 57.7 | 3.0 | 68.2 | 2.6 |
| Native American | 45.5 | 6.3 | 62.9 | 5.8 |

*Smoke-free is defined as not permitting smoking in both the public and common areas nor in work areas.

Table 4

*Coverage: Smoke-free policies [smoking not permitted in public and common areas of the worksite nor in work areas] – In 1992–93, indoor workers from only 18 states reported that 50% or more of their workplaces were covered by a smoke-free policy. By 1995–96, < 50% coverage was noted in only three states: Nevada, Arkansas, and Kentucky. In 1992–93, no state reported a rate of 70% or higher coverage by smoke-free workplace policies. By 1995–96, workers from 13 states reported that level of coverage. Preliminary data from the 1998–99 CPS suggests that in 21 states, 70% or more of the indoor workforce was covered by smoke-free worksite policies. Only 3 states had rates < 60%. In Nevada, < 50% (48.7%) of the workforce was covered by a smoke-free policy.*

**Percentage of U.S. indoor workers, ages 18 and older, covered by a smoke-free* workplace policy in 1992-93 and 1995-96, by state, and relative change (%) between the two time periods**

| State | % of workers covered in 1992-93 | % of workers covered in 1995-96 | Relative change % |
|---|---|---|---|
| Alabama | 39.0 | 55.4 | 42.0 |
| Alaska | 58.7 | 69.9 | 19.0 |
| Arizona | 56.8 | 65.1 | 14.5 |
| Arkansas | 32.5 | 48.0 | 47.6 |
| California | 58.4 | 76.1 | 30.2 |
| Colorado | 53.5 | 71.5 | 33.7 |
| Connecticut | 48.3 | 67.3 | 39.4 |
| Delaware | 50.3 | 66.0 | 31.2 |
| District of Columbia | 51.9 | 74.7 | 43.9 |
| Florida | 53.6 | 66.5 | 24.1 |
| Georgia | 47.4 | 56.7 | 19.8 |
| Hawaii | 47.1 | 61.3 | 30.0 |
| Idaho | 59.5 | 70.9 | 19.1 |
| Illinois | 40.0 | 60.8 | 52.0 |
| Indiana | 35.1 | 51.3 | 46.1 |
| Iowa | 45.1 | 62.2 | 38.0 |
| Kansas | 49.4 | 63.2 | 27.8 |
| Kentucky | 28.6 | 49.3 | 72.2 |
| Louisiana | 39.3 | 56.6 | 43.9 |
| Maine | 55.7 | 73.4 | 31.7 |
| Maryland | 52.9 | 83.2 | 57.2 |

| | | | |
|---|---|---|---|
| Massachusetts | 48.7 | 71.1 | 45.8 |
| Michigan | 39.6 | 53.1 | 34.3 |
| Minnesota | 54.5 | 67.9 | 24.5 |
| Mississippi | 40.3 | 54.2 | 34.5 |
| Missouri | 39.1 | 58.5 | 49.7 |
| Montana | 43.7 | 58.8 | 34.6 |
| Nebraska | 44.4 | 63.6 | 43.3 |
| Nevada | 33.8 | 40.5 | 19.8 |
| New Hampshire | 53.3 | 72.9 | 36.7 |
| New Jersey | 46.3 | 68.0 | 46.7 |
| New Mexico | 55.3 | 65.5 | 18.5 |
| New York | 42.7 | 64.6 | 51.1 |
| North Carolina | 31.0 | 54.7 | 76.5 |
| North Dakota | 47.5 | 61.0 | 28.4 |
| Ohio | 37.9 | 56.6 | 49.5 |
| Oklahoma | 41.5 | 58.0 | 39.6 |
| Oregon | 59.9 | 66.9 | 11.6 |
| Pennsylvania | 42.2 | 59.9 | 42.0 |
| Rhode Island | 44.9 | 69.6 | 55.1 |
| South Carolina | 37.7 | 58.8 | 55.9 |
| South Dakota | 43.8 | 62.3 | 42.4 |
| Tennessee | 36.2 | 53.5 | 47.8 |
| Texas | 51.2 | 64.8 | 26.7 |
| Utah | 65.4 | 83.7 | 28.0 |
| Vermont | 58.7 | 78.9 | 34.4 |
| Virginia | 43.8 | 62.6 | 43.0 |
| West Virginia | 38.6 | 58.9 | 52.6 |
| Washington | 68.3 | 73.2 | 7.2 |
| Wisconsin | 43.8 | 62.0 | 41.7 |
| Wyoming | 48.4 | 61.2 | 26.6 |

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# Tobacco Smoke and Work Related Non-malignant Respiratory Disease

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## Introduction

The respiratory system is particularly susceptible to injury from inhaled materials because of its large surface area and the vast volume of air breathed. All of the major categories of respiratory system disease—upper airway irritation and allergies, bronchitis, bronchiolitis, asthma, chronic obstructive pulmonary disease (COPD), hypersensitivity pneumonitis (HP), pulmonary fibrosis, and lung and pleural cancer—can be caused by workplace exposures (Figure 1). The one exception is pulmonary vascular disease. Cigarette smoke, although chemically quite complex, has a narrower spectrum of adverse effects on the respiratory system.

The prevalence of cigarette smoking in the United States rose dramatically during the course of the 20th century, as shown for successive birth cohorts of males in Figure 2. In the second half of the 20th century, as tuberculosis was brought under control, cigarette smoking was recognized as the single most preventable cause of lung disease in the general population unexposed to occupational respiratory hazards.

Physicians caring for workers with occupational lung diseases have often been unaware of the variety and severity of adverse effects of occupational exposures on the lungs. They may assume that chronic lung disease in those with occupational exposures is caused only by smoking. There have been many instances in which workers who did not smoke but who suffered with severe occupational lung disease were advised by physicians to "quit smoking."

Multiple factors combine to determine the risk for developing lung disease and the severity of lung disease. Genetic predisposition, in utero and perinatal events, respiratory viral infections occurring at key times during immunologic development and lung maturation, diet, home environment, and occupational environment are all important in the pathogenesis of a number of common lung diseases.

The potential for seriously disabling or fatal lung disease has often been greater for those with lower educational levels and lower incomes. Historically, lower-paying jobs were commonly "dirtier" jobs—those with a higher likelihood of exposing workers to damaging levels of workplace toxins. At the same time, those with less educational opportunity and lower income in the United States have had higher rates of smoking. For example, a 1983 study of lung disease in welders found that the prevalence of smoking in full-time welders was higher than the national average [Kilburn et al. 1989]. The association of smoking with socioeconomic status continues to this day. However, the association of smoking with harmful occupational exposure may now be much weaker than it was a generation ago.

For many, but not all of the occupational lung diseases, there can be important relationships to cigarette smoking. Other kinds of tobacco use—particularly cigar and pipe smoking—may also influence the effects of occupational exposures. However, cigarette smoking, in most instances, has a markedly more potent influence.

Most joint effects between active cigarette smoking and occupational exposures are "positive." For example, cigarette smoking increases the amount of damage caused by the occupational exposure ( Table 1). In some cases, the damaging effects of active cigarette smoking and exposure to occupational lung toxins are independent of one another. The individual is, nonetheless, at risk for injury both from smoking and from the occupational exposures. In a few instances, cigarette smoking, while harmful, modifies the effects of occupational exposures and even appears to reduce the risk of acquiring certain kinds of allergic lung disease. Following is a review of the effects of active tobacco smoking on work-related, non-malignant respiratory disease.

## Combined Health Effects of Occupational Exposures and Active Cigarette Smoking

Combined effects of occupational exposures and active cigarette smoking may be measured using one or more indices of disease occurrence and severity (Table 2). Two different inhaled substances can be thought of as affecting the lungs in one of several possible ways Table (3).

Additive Effects – First, the effect may be the same as the sum of each of the individual effects. In this case the interaction is said to be "additive." The magnitude of the effect of each of the two exposures is no different than it would be if it occurred in the absence of the other exposure. The effect of each of the two exposures may be large or negligible depending on the amount of exposure (inhaled concentration and duration) and on the biological response of the host. Among healthy workers, there is marked variability in the response to commonly inhaled agents. For example, among cigarette smokers, only a small proportion—about 15 %—who smoke one pack per day for an adult

lifetime will go on to develop clinically disabling COPD. Similarly, among individuals exposed on a daily basis to high levels of asbestos fibers over a working lifetime, only a minority will go on to develop disabling pulmonary fibrosis from asbestosis. We do not yet know which biologic factors pre-dispose an individual to the damaging effects of cigarette smoke or of asbestos. But individuals exposed to both substances may develop severe COPD from smoking, severe pulmonary fibrosis from asbestos exposure, neither of these diseases, or both diseases at once.

*Multiplicative or Synergistic Effects* – A second kind of effect from two different exposures is positive but greater than the sum of the effects of each separate exposure. This joint effect is called "greater than additive" and may in some cases be described as "multiplicative." The magnitude of the effect is equivalent to multiplying the two individual effects. This kind of effect is also sometimes called "synergistic," suggesting that a combination of factors markedly amplifies the deleterious effects. (The term synergistic, however, is used differently in the fields of genetics and oncology). The 1985 Surgeon General's Report concluded that cigarette smoking and asbestos act "synergistically" to increase the risk of lung cancer [DHHS 1985].

Synergistic effects may occur in chronic, non-malignant respiratory diseases through adverse effects of smoking and smoking-related diseases on lung deposition and lung clearance of occupational dusts. Laboratory studies comparing patients with COPD to those without lung disease have demonstrated a markedly increased fraction of deposition of inhaled, one micron diameter particles in those with COPD [Kim and Kang 1997]. Similarly, mucociliary clearance of inhaled particles may be impaired in smokers due to adverse effects of smoking on the mucociliary clearance system of the bronchi and trachea.

*Antagonism* – A third category of interactions is reserved for more unusual circumstances where exposure to one substance—while harmful—seems to reduce the negative effects of a second exposure, or may even alter the nature of the effect of the second exposure. In toxicologic terms, this may be thought of as an antagonistic interaction.

*Immediate Effects* – Simultaneous cigarette smoking and occupational exposures can also have immediate interactions. Contamination of the surface of cigarettes with the fluoropolymer polytretrafluoroethylene and subsequent heating of the polymer can lead to a potentially severe acute respiratory and systemic response, called polymer fume fever [CDC 1987].

## Non-Malignant Respiratory Diseases: Relationship To Active Cigarette Smoking and Occupational Exposures

### Asthma

*Non-Occupational Asthma* – Asthma is commonly defined as reversible obstructive airway disease. It may have its onset in childhood or adulthood. A subset of asthma in adults, estimated at from 5% to 15%, is caused specifically by workplace exposures and is known as occupational asthma. Work-aggravated asthma describes asthma which is made more symptomatic or severe as a result of workplace exposures. In adults, the differentiation of asthma from COPD or chronic bronchitis and emphysema can be difficult, and COPD is strongly and causally associated with cigarette smoking. The "Dutch Hypothesis" of asthma posits that individuals with persistent asthma develop chronic changes in the airways, over a period of years, that are clinically (although not necessarily pathologically) indistinguishable from COPD.

Studies have shown that active cigarette smoking is surprisingly frequent in adult asthmatics. In most studies, active smoking is associated with indices of disease severity such as increased frequency of airway symptoms and accelerated rate of lung function loss. Adult smokers have greater airway reactivity—a feature of asthma—compared with non-smokers. The linkage of smoking with lung diseases is so common that it may be surprising even to knowledgeable clinicians to learn that active cigarette smoking plays a relatively small role in the primary causation of asthma (Table 4).

A role for active cigarette smoking in causation of asthma has most frequently been examined in cross-sectional surveys of adults, in which they are asked about a physician diagnosis of asthma and about current or ex-smoking status. Unlike the association of smoking with lung cancer and COPD, where the risk of disease among smokers is on the order of ten-fold compared to non-smokers, the risk for asthma associated with smoking is often a relative risk of two or less. There is also a suggestion of gender-related and age-related differences in susceptibility.

In a community sample of women over 55 years of age, 40% of self-reported asthma was attributable to smoking [Forastiere et al. 1998]. Among more than 74,000 U.S. women 34 to 68 years of age, current smokers were at significantly higher risk for chronic bronchitis, but at significantly lower risk for asthma than women who never smoked or women who quit [Troisi et al. 1995]. A recent case-control study of asthma and smoking found no significant difference between the proportion of smokers among asthmatics compared to controls [Ben-Noun 1999]. A similar result was found in a recent random sample of the general population of Perugia, Italy, where current smoking was inversely associated with the prevalence of current asthma [Siracusa et al. 1997]. In a Swedish population-based study of asthma incidence, the risk for female smokers was significant (risk ratio 1.6). However, no excess risk for asthma was found among smoking males [Toren and Hermansson 1999]. Because most of these studies were prevalence surveys, it is not possible to determine whether the presence of asthma was a factor which prevented adults from initiating smoking or motivated them to quit smoking. Regardless of the nature of the relationship, smoking does not appear to be a major risk factor in causation.

*Occupational Asthma* – Studies have shown a relationship between cigarette smoking and occupational asthma, depending on the nature of the substance causing occupational asthma. In individuals exposed to substances causing typical allergic or IgE mediated asthma, there is often a significant relationship between cigarette smoking and increased indices of allergic sensitization, such as skin prick wheal-and-flare and specific serum IgE responses or cutaneous and mucosal allergic responses such as rhinitis and eye itch. In such studies, the association of asthma incidence with smoking may be present but less strong. For example, in a multivariate study of occupational asthma in bakers—one of the most frequently reported occupations in which asthma occurs—skin sensitization to specific flour antigens was significantly more common in smokers, but respiratory symptoms were not [DeZotti et al. 1994]. In occupational asthma caused by substances such as acid anhydrides [Taylor et al. 1997] and platinum salts [Venables et al. 1989], there is a positive effect of active smoking on asthma incidence. While smoking itself does not have a major effect in asthma causation, cigarette smokers in these industries are more likely than non-smokers to develop occupational asthma to the large organic or biologic allergens (Table 5).

For other causative agents of occupational asthma, however, there appears to be no relationship with cigarette smoking. This has been demonstrated more than once in the case of occupational asthma caused by diisocyanates (chemical catalysts used in the manufacture of widely used polyurethane paints and foams). Diisocynates are the most frequently recognized cause of occupational asthma in North America and Britain and for this cause of asthma, cigarette smoking is not associated with an increased risk for asthma [Butcher et al. 1977].

A different effect of active smoking on the development of occupational asthma has been documented by one of the largest national surveys of occupational lung disease, the English SWORD program. Among those workers whose asthma was attributed to high molecular weight agents, active smokers developed asthma earlier than non-smokers [Ross and McDonald 1988]. However, their prognosis after diagnosis was better than for non-smokers. Thus, active cigarette smoking affected the interval between the initial workplace exposure and the onset of asthma symptoms.

## Chronic Bronchitis

A useful clinical definition of bronchitis is the presence of cough and mucous expectoration. When bronchitis is present most days of the week for three or more months of the year for two or more consecutive years, there is usually a measurable increase in the depth of the mucous gland layer of the airways of the lungs. The patient is said to have “chronic bronchitis.” While sometimes dismissed as a minor illness, chronic bronchitis is common among working populations including non-smokers. It is strongly associated around the world with occupational exposures to a wide variety of substances, including rock, mineral and cement dusts in miners and others, welding and cutting torch smoke [Cotes et al. 1990], and several types of grain and vegetable dusts [Morgan 1978].

Chronic bronchitis is both one of the earliest and most frequently occurring lung diseases in regular cigarette smokers. While it may occur as the only effect of smoking, it often occurs in combination with the more disabling condition, emphysema. Epidemiologic studies have repeatedly demonstrated an additive effect of occupational exposures and active smoking in determining the frequency and severity of bronchitis in working groups [Korn et al. 1987]. Both factors contribute significantly to the individual’s degree of symptoms and impairment. For the smoking worker with chronic bronchitis, cessation of exposure to workplace dusts or gases and active cigarette smoking can have a positive effect on health and well being. Chronic bronchitis is a condition that can be entirely cured if the causative substances are removed before it becomes a permanent condition of the bronchial airways.

## Emphysema and Chronic Airflow Limitation

Emphysema is a common and often severely disabling lung disease whose major characteristic is the loss of the fine latticework of alveolar and capillary tissues across which oxygen is transported in breathing. One of the direct results of emphysema is chronic airflow limitation (or COPD). This association is commonly used to detect the presence of emphysema and measure its severity in life.

COPD is one of the most common causes of premature mortality in the United States. In 1995, it was designated as the fourth leading cause of death after coronary heart disease, cancer, and stroke [National Center for Health Statistics 1997]. Population and age-adjusted mortality from COPD has continued to rise in spite of declining cigarette smoking rates. This is due to the irreversible nature of smoking-related emphysema and to the age-related decline in lung function. Approximately 80–90 % of COPD mortality in men, and a smaller percentage in women, is attributable to active cigarette smoking.

Estimates of the proportion of COPD cases attributable to occupational exposures in the United States have ranged from as high as 14–28% [Becklake 1994]. Such estimates are likely to change. Smoking prevalence has declined, and the prevalence of current causative occupational exposures awaits additional study by the National Institute for Occupational Safety and Health. The list of workplace substances known to cause COPD includes cadmium fume (which causes emphysema), respirable crystalline silica, coal dust, cotton dust, grain dust, and toluene diisocyanate (Table 6).

Tuberculosis, an infectious disease transmitted from person to person which may be an occupational disease in some workplaces, can cause bronchiectasis leading to chronic airflow obstruction. Smokers who are susceptible to the effects of cigarette smoke and to the effects of the workplace agents listed above will have more severe lung disease than if they were exposed to cigarettes only or to the occupational agent alone. While it is reasonable to assume that the combined effects of exposure to these substances with active cigarette smoking are additive, further study is needed to better define the relationships.

## Pneumoconiosis

Pneumoconiosis describes chronic interstitial lung disease caused by inhaled mineral dusts.

*Coal Dust* – Those who inhale coal dust may develop bronchitis, chronic airflow limitation, coalworkers' pneumoconiosis, or a combination of these. The lesion of coalworkers' pneumoconiosis includes areas of focal emphysema in a distribution different from that caused by cigarette smoking. The result, however—loss of gas exchanging alveolar surface, and chronic airflow limitation—is similar. In addition, coal dust exposure causes a focal nodular fibrotic interstitial lesion, the coal macule, which may cause clinically significant disability if present in sufficient number. The characteristic pulmonary function effect of cigarette smoking is an obstructive defect with a decline in the ratio of $FEV_1$ (forced expiratory volume) to FVC (forced vital capacity), whereas coal dust appears to cause a decline in forced vital capacity (FVC) with either a smaller proportional loss in forced expiratory volume in the first second ($FEV_1$) or decline in $FEV_1$ to FVC than is seen from smoking [Morgan et al 1974; Attfield and Hodous 1992]. While the relative importance of coal dust and smoking in causing pulmonary disability in coal miners is still the subject of debate, it is clear that an additive effect takes place.

*Silica* – Like coal and other mineral dusts, silica may cause chronic bronchitis, which can be associated with airflow obstruction. The most common interstitial lesion of inhaled crystalline silica is the silicotic nodule, a discrete area of fibrosis, usually surrounded by normal lung tissue. With exposure to very high doses or to the more fibrogenic forms of silica (cristobalite and tridymite), patchy interstitial fibrosis may occur. A third lesion, alveolar proteinosis, causes filling of alveoli with a tenacious exudate which prevents gas exchange in the involved areas of lung. There can be an additive effect of silica dust exposure and active smoking in producing bronchitis. Where smoking causes airflow obstruction, the effects may also be additive. The response of the lungs to silica is often a mixed obstructive and restrictive pathophysiology, which can be made more severe by the separate effects of cigarette smoking. In addition, active smoking and exposure to silica dust results in an increased risk for lung cancer.

## Pulmonary Fibrosis

*Idiopathic Pulmonary Fibrosis* – As the terminology for the different forms of pulmonary fibrosis has become more specific, the term "idiopathic pulmonary fibrosis" is now used as the equivalent for a specific pathologic type, "Usual Interstitial Pneumonia" or UIP. Cigarette smoking in susceptible individuals usually creates emphysema, or

destruction of alveolar tissue, whereas fibrosis is the remodeling of lung architecture and the formation of added scar tissue. The major inciting event or events which trigger the severe fibrotic process leading to UIP are still unknown. However, recent epidemiological case-control studies suggest that both cigarette smoking [Coultas et al. 1994] and occupational exposure to a variety of metal dusts in manufacturing industries [Hubbard et al. 1999] may contribute as minor factors to the risk of developing this life-threatening lung disease. Our current understanding of UIP indicates a combination of factors—some perhaps genetic, some perhaps environmental—which trigger the process that culminates in fibrosis. Cigarette smoking, while a risk factor, is neither sufficient nor necessary to cause UIP.

*Respiratory Bronchiolitis Interstitial Lung Disease* – A second sub-category of interstitial fibrosis is respiratory bronchiolitis interstitial lung disease, an inflammatory process of the submucosa of the membranous and respiratory bronchioles, often associated with fibrous scarring that extends into the surrounding alveolar walls. This lesion is usually found only in heavy cigarette smokers [King 1993]. It is much less commonly detected than bronchitis and emphysema and may represent an extreme form of the mild bronchiolitis seen more frequently in cigarette smokers. Serious acute occupational inhalation injuries can cause a similar lesion called obliterative bronchiolitis. Studies of acute injuries causing obliterative bronchiolitis superimposed on respiratory bronchiolitis interstitial lung disease have not been conducted. However, the low frequency of both conditions makes the co-existence in one individual unlikely.

*Asbestosis* – The pathologic lesion of asbestosis is indistinguishable from that of UIP, except that lung tissue contains a high burden of asbestos fibers. Because occupational exposure to asbestos and cigarette smoking have historically occurred in the same groups of working men, there has been ample opportunity to study their joint effects, and two have been found (Table 7).

First, in asbestos-exposed cigarette smokers, the presence of both emphysema (from smoking) and fibrosis or asbestosis (from asbestos) are quite common. Both diseases occur progressively after many years of asymptomatic, frequent, regular exposure. Their coexistence is clinically significant, because both interfere with the gas-exchanging capability of the alveoli and contribute to a more severe degree of respiratory disability.

Second, comparisons of the chest radiographs of asbestos-exposed smokers and non-smokers has repeatedly demonstrated an enhancing effect of cigarette smoking on the profusion of interstitial abnormalities as determined by application of the International Labour Organization (ILO) system of categorizing radiographs for pneumoconiosis. While this effect is related to the amount of smoking as estimated by pack-years, the average effect of regular smoking is to increase the profusion by one-half category. For example, the radiograph of a smoker whose asbestos exposure was identical to a non-smoker might be ILO category ½ while that of the non-smoker was ILO category 1/1 [Weiss 1984]. This important observation has not been investigated in other forms of occupational interstitial disease, but is at least consistent with the observation of cigarette smoking as a risk factor for idiopathic pulmonary fibrosis. While the interstitial changes noted on radiograph are assumed to be due to increased interstitial fibrosis, direct pathologic correlation of this relationship has not been established.

## Hypersensitivity Pneumonitis (HP)

Hypersensitivity pneumonitis, also known as extrinsic allergic alveolitis, is an immunologically mediated inflammatory disease of the pulmonary interstitium. The acute form presents with dyspnea, hypoxemia, and often fever. The chronic form presents with dyspnea, cough, malaise, weight loss, chronic pulmonary function loss, and chronic interstitial disease.

The use of occupational eponyms to describe disease from each of many specific causes (e.g., farmer's lung, pigeon breeder's lung, bathtub-finisher's lung, etc.) has in part obscured the fact that this is a single lung disease with an ever-lengthening list of occupational as well as environmental causes. Most frequently, HP is caused by inhaling a particulate or mixture of substances of biologic origin, including bacteria and fungi. While acute episodes may be self-limiting, continued exposure to the inciting substance can lead to chronic disease which can be fatal. Typically, of many individuals exposed to a causative substance or mixture, only a sub-population develops antibodies to the agent, and a smaller sub-population develops clinical disease. Thus, for example, among dairy farmers exposed to moldy hay, only a small sub-group develop HP to the microorganisms released when hay bales are opened. In regions of highest occurrence, deaths from this disease are reported at between 2.5 and 14.5 per million age-adjusted population [NIOSH 1998].

Cigarette smoking has repeatedly been found to influence the risk for hypersensitivity pneumonitis. Among occupational populations exposed to known causative agents for HP, cigarette smokers are significantly less likely to have a diagnosis of the disease, although smoking is not entirely protective [Schuyler 1999]. HP is an immunologically mediated disease. Thus, it is assumed that inhalation of cigarette smoke alters the susceptibility of the cellular immune system, causing a marked and counter-productive inflammatory response to otherwise benign environmental antigens. Cigarette smoking has also been found to be partially protective against exacerbations of ulcerative colitis, a form of inflammatory bowel disease for which a causative agent or agents have not yet been identified. Whether this similarity points to a link in the pathogenesis of these two, quite different diseases is not known.

## Summary

There are at least three basic effects of active cigarette smoking on non-malignant occupational lung disease. The first is an additive effect from combined exposure to certain occupational agents and active cigarette smoking. An example is that which occurs with the emphysematous lesions of coalworkers' pneumoconiosis. The second is an enhancement of the atopic state resulting in the formation of specific antibodies to inhaled occupational allergens. In some cases, this results in increased incidence of asthma in occupationally exposed smokers, whereas in others, there is no effect on asthma. The third, and perhaps least well understood, is a reduction of the incidence of hypersensitivity pneumonitis among cigarette smokers. While this effect has been well described, the mechanism remains to be determined.

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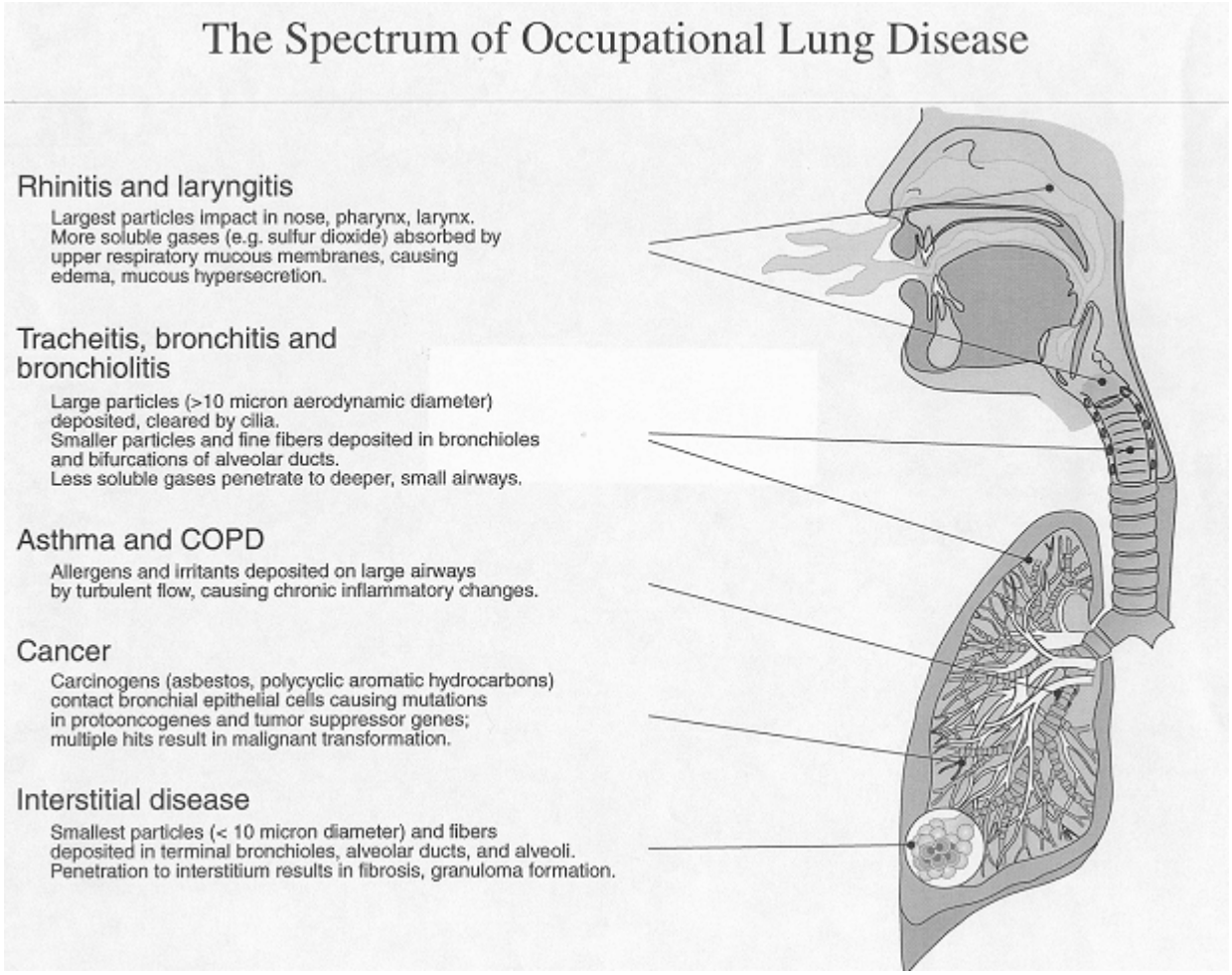
**Figure 1.**

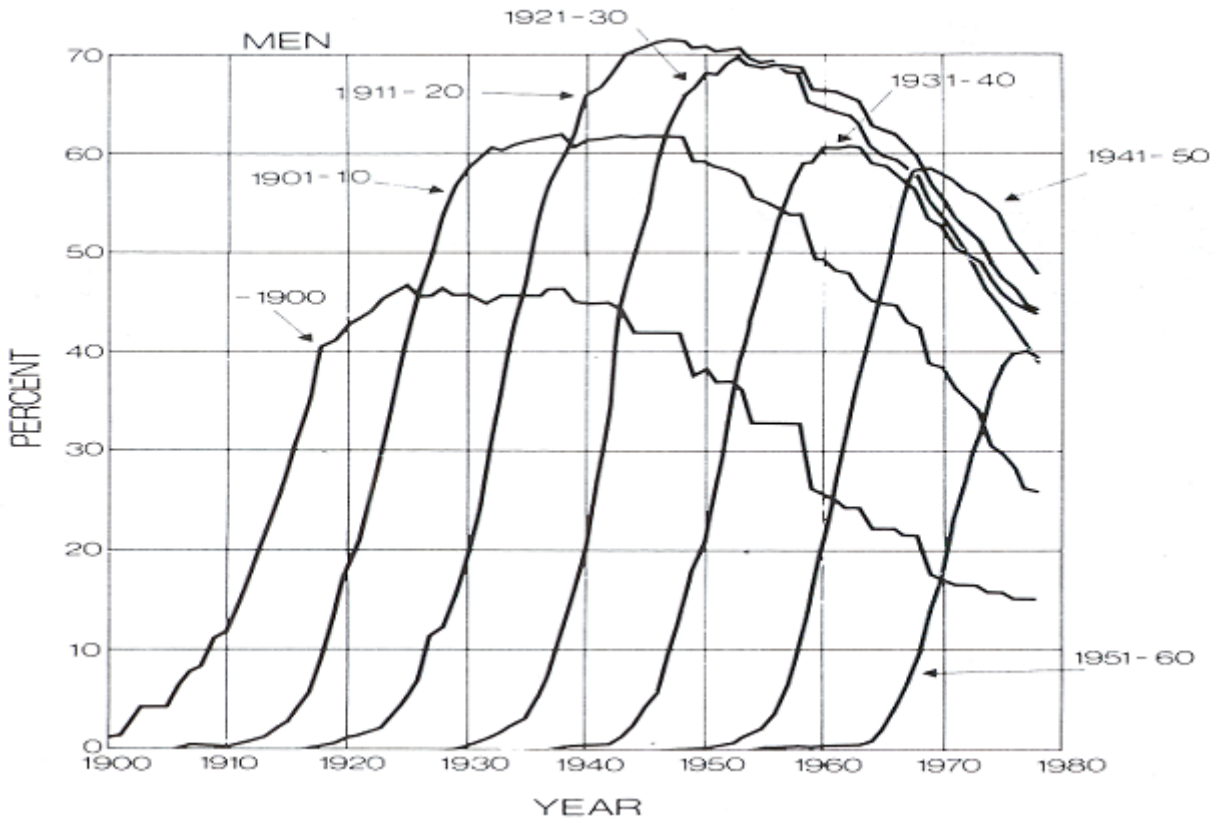



**The spectrum of occupational lung diseases.**

Inhaled workplaces substances may be selectively deposited at different levels of the airways depending on substance size and solubility. Workplace substances may cause all the major categories of respiratory tract disease, from rhinosinusitis to interstitial lung disease.

## Figure 2.

**Prevalence of cigarette smoking among successive birth cohorts of men, 1900 to 1980** (derived from smoking histories in the National Health Interview Survey).



# Tables

Table 1.
**Non-malignant respiratory diseases**

Caused by cigarette smoking:
- Chronic bronchitis
- Emphysema
- Bronchiolitis

Affected by cigarette smoking:
- Asthma
- Fibrosis

Table 2.
**Outcome measures for combined effects of occupational exposures and cigarette smoking**

A. Disease incidence
B. Disease severity
   1. Symptoms
   2. Pulmonary Function ($FEV_1$)
   3. X-ray changes
C. Premature mortality

Table 3.
**Types of combined effects of occupational exposures and active cigarette smoking**

- Additive: the removal of one agent eliminates only the excess due to that agent
- Multiplicative (synergistic): removal of one agent removes (some of the) excess due to both
- Antagonistic: one factor reduces the excess disease caused by another
- Other kinds of interactions

Table 4.
**Asthma: associations with cigarette smoking**

- More than 200 workplace substances are known to cause asthma
- Smoking increases asthma severity, but the association with causation is inconsistent
- Smoking increases the risk for occupational asthma caused by some substances; no effect on risk for other substances
- Smoking accelerates lung function loss in asthmatics

Table 5.

**Selected occupational substances for which there is evidence of a positive association between cigarette smoking and subsequent allergic sensitization, as measured by skin tests, IgE, and/or the occurrence of asthma.***

Acid anhydrides
Colophony (solder core, from pine resin)
Flour antigens
Laboratory Animals
Platinum salts

* This association has *not* been demonstrated for the majority of known causes of occupational asthma, and for some there appears to be a negative association.

Table 6.

**Chronic airflow limitation: selected occupational causes**

- Coal Dust: emphysema and nodular fibrosis
- Crystalline silica: chronic airflow limitation
- Cotton dust: asthma, bronchitis, and chronic airflow obstruction
- Cadmium: emphysema
- Toluene Diisocyanate: chronic airflow obstruction

Table 7.

**Asbestos and active smoking: joint effects**

- On lung function
  - Additive, independent effects of asbestos and cigarette smoking were seen on decreased forced vital capacity and on single-breath diffusing capacity
- On Chest X-Ray ( International Labor Organization Category)
  - Asbestos exposure causes pulmonary fibrosis, while cigarette smoking usually causes emphysema (destruction of alveolar surface)
  - In those with asbestosis who have also been heavy smokers, there is (on average) an increase in the profusion of small linear opacities rated on chest x-ray
  - A smoker may have ½ category higher profusion than a non-smoker with equivalent asbestos exposure

# Tobacco Smoking and Workplace Hazards: Cancer, Heart Disease, and Other Occupational Risks

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## Introduction

The smoking habits of U.S. workers vary greatly by gender, education, and occupation. Using data from National Health Interview Surveys (NHIS), Nelson et al. [1994] found that the prevalence of current smokers declined from 31.7% to 24.2 % for white collar workers, and from 43.7% to 39.2% for blue-collar workers from 1978 to 1990. However, the rate of decline has been considerably less for some occupations such as construction and craft workers where the prevalence of current smokers remained greater than 40% among plumbers, pipefitters, carpenters, laborers, roofers, painters, brick masons and drywall installers. Likewise, the prevalence of current smokers was found to be higher than 40% for many industrial occupations.

Exposure to environmental tobacco smoke (ETS) also has been shown to vary substantially by occupation with some workplace concentrations being ten times higher than home concentrations [Hammond 1999]. Studies reviewed by Repace and Lowrey [1990] suggested a 62% probability of exposure to ETS for a nonsmoker in the workplace. Mean exposures to respirable suspended particles (< 2.5 µm diameter) varied from 18.5 $\mu g/m^3$ to 45.5 $\mu g/m^3$ depending on the work environment.

Active smoking and exposure to ETS provide an opportunity for an interaction to occur between smoking and workplace exposures to physical, chemical, and biological agents. Blue-collar workers, who have the highest prevalence of smoking, also have the highest prevalence of workplace exposures to hazardous physical, chemical agents, and biological agents. In addition, some racial and ethnic worker groups, such as African American workers, have a higher historical prevalence of smoking, as well as higher occupational exposures to hazardous agents.

There is increasing recognition that both smoking and workplace exposures are significant contributing factors in the etiology of many diseases and health conditions including certain cancers, cardiovascular diseases, musculoskeletal disorders, occupational injuries, and occupational fatalities. The adverse health conditions resulting from combined exposure to tobacco smoke and occupational hazards and possible mechanisms of interaction are reviewed in this report.

## Toxic and Carcinogenic Components of Cigarette Smoke

Mainstream (MS) and environmental tobacco smoke (ETS) contain at least 250 chemicals known to be toxic or carcinogenic [DHHS 1986; IARC 1986; NTP 2000]. The major source of ETS is sidestream (SS) smoke, and to a lesser extent exhaled MS smoke. Undiluted side stream tobacco smoke contains higher amounts of some toxic and carcinogenic agents, such as volatile N-nitrosamines and aromatic amines, than MS smoke [DHHS 1989; NRC 1986]. Peak temperatures in the burning cone of a cigarette reach 800° to 900°C during puffing, but only 600°C between puffs, resulting in less complete combustion of tobacco during generation of SS smoke. Also, most of the burning cone is oxygen deficient during smoldering, thus a strongly
reducing environment is present [NRC 1986]. ETS is diluted in the air before it is inhaled and is less concentrated than MS. Sidestream smoke is a significant source of highly respirable particles (< 2.5 µm diameter)[Hudgins and Karetzky 1994].

Table 1 lists examples of toxic and carcinogenic agents and classes of agents identified in SS and MS tobacco smoke [NIOSH 1991]. OSHA [1994] identified 43 chemical compounds in tobacco smoke (Table 2) for which there is "sufficient evidence" of carcinogenicity in humans or animals.

## Diseases Caused by Active and Passive Smoking

In 1964 the Surgeon General concluded that cigarette smoke causes lung cancer. Since that time, additional research on the toxicity and carcinogenicity of tobacco smoke has demonstrated that the health risks from inhaling tobacco smoke are not limited to smokers but also include those who inhale ETS. The 1989 Surgeon General's Report [DHHS 1989] lists specific diseases and their associated ICD-9 codes where epidemiologic data have shown increased risk of smoking attributable mortality. Risk estimates for neoplasms, cardiovascular diseases and respiratory diseases in the Surgeon General's Report are based on the Cancer Prevention Study (CPS-II) conducted by the American Cancer Society. The CPS-II study began in 1982 and included more than 1.2 million persons in all

50 states. Age standardized relative risk (RR) estimates for smoking attributable diseases are shown in Table 3 for males and females, respectively.

Additional epidemiologic data have shown increased risks among smokers for hematopoietic cancers including leukemia and non-Hodgkin's lymphoma [Linet et al. 1991; Brown et al. 1992; Franceschi et al.1989; Brownson et al. 1992; Friedman 1993]. Benzene is a known hematopoietic system carcinogen and is present at concentrations of approximately 45 µg/cigarette in MS smoke; however, the benzene content of SS smoke is much higher [Korte et al. 2000]. In addition to cancers of the hematopoietic system, a recent case-control study suggests that smoking is a risk factor for myelodysplastic syndromes [Bjork et al. 2000].

Given the components found in ETS as compared to MS smoke, a comparable spectrum of disease risks would be anticipated, although at a reduced magnitude due to lower exposure levels. NIOSH [1991] determined that the collective weight of evidence was sufficient to conclude that ETS poses an increased risk of lung cancer and possibly heart disease to occupationally exposed workers. OSHA [1994] also concluded that ETS increased the risk of lung cancer and cardiovascular disease. The National Toxicology Program lists ETS as a *known human carcinogen* in the 9th Report on Carcinogens [NTP 2000].

In addition to the diseases described above, smoking may contribute to injuries and deaths in the workplace. Smokers have been shown to have higher rates of non-intentional injuries. In a meta-analysis of smoking and injury death, Leistikow [1998] found that smoking increased the risk of injury death by 61% after controlling for many potential confounders. Cigarette smokers are approximately twice as likely as non-smokers to be injured at work and have approximately a 50% increased risk of motor vehicle accidents compared to nonsmokers [Sacks and Nelson 1994]. This difference remains after stratification by age, driving experience, level of education, and alcohol consumption; therefore, confounding does not fully explain the association.

Cigarette smoking has been shown to have a number of orthopedic consequences including impeding bone metabolism and fracture repair, increased rates of postoperative infections, and increased incidence of nonunion [Kwiatkowski et al. 1996]. Although the available studies are not conclusive, a body of data supports smoking as a risk factor for low back pain.

## Concepts of Synergy and Interaction

Interactions between cigarette smoke exposures and occupational exposures may occur in the context of a biological process, as a statistical phenomenon, or as a problem in public health and individual decision making [DHHS 1985]. Biological interaction occurs when the presence of one agent (e.g., cigarette smoke) influences the form, availability, or effect of a second agent (e.g., occupational exposures). Biological interaction includes: 1) physical interactions such as the adsorption of carcinogens onto respirable particles in inspired air; 2) pharmacological interactions such as enzyme induction by one agent, which enhances toxicity of a second agent through metabolic activation; and 3) outcome interactions such as the number of cancers produced by separate or combined exposures. Statistical interaction refers to the mathematical model used to assess the effects of combined exposures and can be additive, multiplicative, or some other mathematical form. Finally public health interaction refers to the presence or level of exposure to one agent influencing the incidence, prevalence, or extent of disease produced by a second agent.

NIOSH [1979] reviewed a number of possible interactions between smoking and occupational exposures. Examples of these interactions include the following:

- *Certain toxic agents in tobacco products and/or smoke may also occur in the workplace thus increasing exposure to the agent.* Employees exposed in the workplace to toxic chemicals can receive additional exposures from the presence of those same toxic chemicals found in tobacco smoke. Examples include carbon monoxide, aromatic amines, benzene, acetone, acrolein, aldehydes (e.g., formaldehyde), arsenic, cadmium, hydrogen cyanide, hydrogen sulfide, ketones, lead, methyl nitrite, nicotine, nitrogen dioxide, phenol, and polycyclic aromatic compounds.

- *Workplace chemicals may be transformed into more harmful agents by smoking.* An example is polymer fume fever caused by inhalation of degradation product fumes from heated Teflon® (polytetrafluoroethylene).

- *Tobacco products may serve as vectors by becoming contaminated with toxic agents found in the workplace, thus facilitating entry of the agent into the body by inhalation, ingestion, and/or skin absorption.* Examples of

potential contaminants of tobacco products include boron trifluoride, carbaryl dinitro-ortho-creosol, inorganic fluorides, formaldehyde, lead, inorganic mercury, methyl parathion, and organotin.

- *Smoking may contribute to an effect (i.e., same target organ and health outcome) comparable to that which can result from exposure to toxic agents found in the workplace, thus causing an additive biological effect.* Examples include chlorine, cotton dust, coal dust, and beta radiation. Other additive effects include exposures to occupational bladder carcinogens such as 2-naphthylamine, 4-aminobiphenyl, and ortho-toluidine [Johansson and Cohen 1997].

- *Smoking may act synergistically with toxic agents found in the workplace to cause a much more profound effect than that anticipated simply from the separate influences of the occupational exposure and smoking.* Examples of possible multiplicative interactions between occupational exposures and tobacco smoke include lung cancer risk and exposures to asbestos, silica, radon daughters, arsenic, and chloromethyl ethers [Steenland and Thun 1986]. Occupational exposures to bladder carcinogens (e.g., 2-naphthylamine, 4-aminobiphenyl, benzidine, 4-chloro-o-toluidine, o-toluidine, 4,4' methylene bis (2- chloroaniline), methylene dianiline, and benzidine-derived azo dyes) also have been shown to interact with tobacco smoke in a multiplicative manner in increasing the risk of bladder cancer.

## Biological Mechanisms of Interaction: Tobacco Smoke & Occupational Risk Factors Cancers

Cigarette smoking may interact with occupational carcinogens through the multi-step process of carcinogenesis by altering the dose of carcinogen reaching the critical target cell or by altering the host vulnerability [Burns et al. 1988]. The multi-stage conceptual model of the carcinogenic process considers agents to be tumor initiators, tumor promotors, and cocarcinogens. A tumor initiator acts on the early stage of carcinogenesis and may exert effects after only a brief exposure of target cells; however, a tumor promoter requires prolonged contact with initiated cells to cancers. A cocarcinogen produces tumors only in the presence of other agents.

One mechanism of possible interaction of workplace exposures and tobacco smoke is through the tumor promoting action of cigarette smoke components on cells initiated by either occupational carcinogens or other carcinogens found in tobacco smoke. In this manner, exposure to an occupational tumor promotor may result in cancer expression only among workers also exposed to tobacco smoke.

Exposure to tobacco smoke can interact with occupational exposures to increase the carcinogen dose to target cells by 1) altering the fraction of inhaled carcinogen deposited or retained in the lung, 2) altering the rate of activation of a procarcinogen into a carcinogenic metabolite, or 3) increased transfer of agents across mucosal and cellular membranes [Burns et al.1988]. Smoking impairs mucociliary clearance of carcinogenic particles such as asbestos. In addition, carcinogenic materials such as PAHs in tobacco smoke can be adsorbed onto asbestos and other particles resulting in greater dose of carcinogen delivered to critical cells. The small particles produced by tobacco smoke can also serve as a vehicle for delivery of other carcinogens such as formaldehyde.

Exposure to tobacco smoke may alter host vulnerability to other carcinogens through a number of mechanisms. One possible mechanism is the greater presence of metabolically activated inflammatory cells in the lungs of smokers. Also, tobacco smoke components and occupational exposures might interact by inducing microsomal and other enzyme systems that serve in metabolic activation of carcinogens. Examples of inducible enzyme systems affected by tobacco smoke are pulmonary arylhydrocarbon hydroxylase (AAH) and the mixed function oxidase system. The carcinogenic effects of some occupational carcinogens such as aromatic amines, butadiene, acrylonitrile, and nitrosamines could be altered by induction of the mixed function oxidase system by tobacco smoke.

Rapid developments in molecular biology have established the role of oncogenes and tumor suppressor genes, especially for lung cancer [Vallyathan et al. 1998]. Activation of K-ras oncogenes is found in a large portion of lung cancers as well as mutations of tumor suppressor gene p53. Nelson et. al. [1998] have shown that alterations in chromosome 3p14 (FAIT ebon deletion) in lung cancer patients is a target of both tobacco carcinogens and asbestos. More research is needed to identify interactions of tobacco with other carcinogens to produce specific gene mutations and alternations in gene repair.

## Cardiovascular Diseases

Cardiovascular diseases such as myocardial infarction, sudden death, and arterial thrombosis occur more frequently in cigarette smokers as opposed to nonsmokers, and the same chemicals which produce these effects in active smokers are present in ETS including nicotine, carbon monoxide, polycyclic aromatic hydrocarbons (PAHs), and tobacco glycoprotein [OSHA 1994]. Carbon monoxide and PAHs are common workplace exposures; therefore, interaction with tobacco smoke is likely to be at least additive.

Possible biological mechanisms for cardiovascular effects associated with exposure to tobacco smoke include thrombus formation and vascular wall injury. Exposure to MS smoke and ETS can cause placeless to become more easily activated thus predisposing the placeless to become involved in forming clots and atherosclerotic plaques. Acute exposure to tobacco smoke also results in increased platelet aggregation, which is thought to be an initial stage of the development of coronary thrombosis or vasoconstriction.

Atherosclerotic plaque formation can lead to constriction of the lumen of the blood vessels, resulting in reduced blood supply to the myocardial tissues. Injury to the endothelial lining of the arterial wall is thought to be an essential step in plaque formation, and MS smoke and ETS have been implicated in causing injury to the endothelial cells which line the arterial walls. Exposure to tobacco smoke also has been implicated in stimulating smooth muscle cell proliferation and in altering blood lipids, both of which can contribute to plaque formation.
Exposure to tobacco smoke may place stress on the heart by increasing myocardial oxygen demand, decreasing myocardial oxygen supply, or interfering with the cell's ability to utilize oxygen for energy production. MS smoke and ETS have been shown to increase myocardial oxygen demand by the direct effect of nicotine increasing the resting heart rate and blood pressure. MS smoke and ETS reduce the oxygen supply through the formation of carboxyhemoglobin. Direct or indirect exposure to tobacco smoke has been shown to increase the hemodynamic determinants of myocardial oxygen demand. Smoking also has been shown to increase resting heart rate and decrease heart rate variability thus placing smokers at a greater vulnerability to the development of arrhythmias and sudden death [Levin 1992; Yotsukura et. al. 1998].

While few studies have investigated the interactions between tobacco smoke and occupational exposures as combined risk factors for cardiovascular disease, interactions are biologically plausible in that many common cardiovascular risk pathways and risk factors are affected. The following are examples of mechanisms of action of common occupational exposures:

- Chronic job strain has been shown to increase ambulatory blood pressure [Belkic et al. 2000], thus elevation of the arterial blood pressure and hypertension are reasonable biological mechanisms whereby job strain increases the risk of cardiovascular diseases.

- Occupational exposures to lead are thought to increase the cardiovascular disease risk through a similar hypertension mechanism.

- Other agents such as carbon monoxide and methylene chloride affect cardiovascular risks by reducing the oxygen supply through the formation of carboxyhemoglobin, thus additive effects with tobacco smoke are expected.

- Carbon disulfide exerts direct effects on the cardiovascular system and increases the risk of coronary artery disease through a number of mechanisms including ECG abnormalities, negative effects on smooth muscle, increasing LDL cholesterol, and increasing diastolic blood pressure [Steenland et al. 2000].

- Nitrate esters (e.g., nitroglycerin and ethylene glycol dinitrate) cause angina and occasional cardiac sudden death following withdrawal from exposure. The mechanism thought responsible for this effect is coronary artery spasm; however, nitrates may also increase diastolic blood pressure.

- Emerging data have shown reduced heart rate variability to be associated with exposures to fine particles (PM 10 & PM 2.5) and ozone [Gold et al. 2000; Schwartz 1999]. This same effect has been shown with smoking; thus, interactive effects are possible.

## Injuries and Injury Deaths

Several possible mechanisms have been hypothesized to explain the association between cigarette smoking and the risk of injury [Sacks and Nelson 1996]. These include: 1) direct toxicity of cigarette smoke and components, 2) distractiblity, 3) medical conditions associated with smoking, and 4) confounding factors including personality or behavioral characteristics. Direct effects of tobacco smoke could include performance decrements due the effects of carbon monoxide and nicotine whereas distractiblity could include loss of attention, preoccupation of the hand for smoking, and irritation of the eyes. Confounding factors might include more prevalent use of drugs and alcohol among smokers compared to nonsmokers. However, studies which have controlled for drug and alcohol use have still shown increased injury risk among smokers. Smoking may also serve as a marker for other personality and behavior characteristics such as risk taking-behavior.

### Smoking & Low Back Pain

Low back pain is a major cause of lost work time and productivity. While occupational exposures to musculoskeletal stresses are the primary risk factors for low back pain, smoking also has been implicated. Hypothesized mechanisms whereby smoking could increase the risk or severity of low back pain include: 1) microfractures in the trabeculae of the lumbar vertebral bodies due to osteoporosis caused by smoking, 2) secondary effects of smoking related cough which increases the intra-abdominal pressure, and 3) alteration of metabolism of the intervertebral disc, causing greater susceptibility to mechanical fracture. Smoking also has been shown to impair blood flow in the systemic microcirculation [Kwiatkowski et al. 1996].

A summary of possible interactions between tobacco smoke and occupational exposures is given in Table 4. This list is not intended to be all inclusive but is presented as examples of the broad range of common occupational exposures that may interact with exposures to mainstream and environmental tobacco smoke to increase the risk of diseases and adverse health events. In most instances additive effects are to be expected; however, synergistic effects have been established for several agents.

## Research, Prevention, and Intervention Needs

- Active smoking and exposure to ETS at work have been shown to interact with some occupational exposures in an additive and sometimes synergistic manner. Well designed prospective epidemiologic studies are needed, especially for cardiovascular diseases, to better define these interactions and related risks. In addition to cardiovascular diseases as endpoints, studies are needed which investigate the combined effects on intermediate cardiovascular risk factors.

- Studies of occupational cohorts have shown increased risks for a number of common cancers (e.g., pancreas); however, specific occupational risk factors have been identified in only a few instances. Additional research is needed to better understand the role of occupational exposures and/or tobacco smoking in the etiology of these cancers.

- Smoking increases the risk of injuries and injury related death; however, biological mechanisms for these increased risks are not well understood. Also, many studies of injuries and deaths are potentially confounded. Additional studies of high risk blue-collar populations with the careful collection of information on risk factors and potential confounders are needed.

- Innovative prevention programs are needed to reduce both smoking and occupational exposures among high risk worker populations. These programs should be based on sound principles of behavior modification, address the risks of both smoking and occupational exposures, and use appropriate educational materials. Strong evaluation components should be included with all prevention programs.

- Innovative routes of access to high risk worker populations are needed for prevention program implementation and evaluation. Implementation of prevention programs through employers and labor unions has been traditional and should continue. Other community based and health care provider based approaches also should be investigated.

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**Table 1**

**Examples of Toxic And Carcinogenic Agents in Main Stream and Undiluted Side Stream Cigarette Smoke**

| Compound | Type of Toxicity | Amount in SS (per cigarette) | Ratio of SS/MS |
|---|---|---|---|
| **Vapor phase:** | | | |
| Carbon monoxide | T | 26.8-61mg | 2.5-14.9 |
| Carbonyl sulfide | T | 2-3mg | 0.03-0.13 |
| Benzene | C | 400-500µg | 8-10 |
| Formaldehyde | C | 1,500µg | 50 |
| 3-Vinylpyridine | SC | 300-450µg | 24-34 |
| Hydrogen cyanide | T | 14-110µg | 0.06-0.4 |
| Hydrazine | C | 90ng | 3 |
| Nitrogen oxides | T | 500-2,000µg | 3.7-12.8 |
| N-nitrosodimethylamine | C | 200-1,040ng | 20-130 |
| N-nitrosopyrrolidine | C | 30-390ng | 6-120 |
| **Particulate phase:** | | | |
| Tar | C | 14-30mg | 1.1-15.7 |
| Nicotine | T | 2.1-46mg | 1.3-21 |
| Phenol | TP | 70-250µg | 1.3-3.0 |
| Catechol | CoC | 58-290µg | 0.67-12.8 |
| o-Toluidine | C | 3µg | 18.7 |
| 2-Naphthylamine | C | 70ng | 39 |
| 4-Aminobiphenyl | C | 140ng | 31 |
| Benz(a)anthracene | C | 40-200ng | 2-4 |
| Benzo(a)pyrene | C | 40-70ng | 2.5-20 |
| Quinoline | C | 15-20µg | 8-11 |
| N'-nitrosonornicotine | C | 0.15-1.7µg | 0.5-5.0 |
| NNK | C | 0.2-1.4µg | 1.0-22 |
| N-nitrosodiethanolamine | C | 43ng | 1.2 |

| Compound | Type of Toxicity | Amount in SS (per cigarette) | Ratio of SS/MS |
|---|---|---|---|
| Cadmium | C | 0.72µg | 7.2 |
| Nickel | C | 0.2-2.55µg | 13-30 |
| Polonium-210 | C | 0.5-1.6pCi | 1.06-3.7 |

**Sources:** NIOSH [1991]
**Abbreviations:** C - carcinogenic; CoC - cocarcinogenic; MS - mainstream smoke; SC - suspected carcinogen; SS - sidestream smoke; T - toxic; TP - tumor promoter; NNK - 4-(methyl-nitrosamino)-(3-pyridyl)-1-butanone

**Table 2**

**Chemical Compounds in Tobacco Smoke for Which There is "Sufficient Evidence" of Carcinogenicity in Humans or Animals**

| | | |
|---|---|---|
| Acetaldehyde | Dibenzo (a,e)pyrene | N-nitrosodi-n-butylamine |
| Acylonitrile | Dibenzo (a,l)pyrene | ortho-toluidine |
| Arsenic | Dibenzo (a,h)pyrene | Styrene |
| Benz (a)anthracene | Formaldehyde | Urethane |
| Benzene | Hydrazine | Vinyl chloride |
| Benzo (a)pyrene | Lead | 1,1-dimethylhydrazine |
| Benzo(b)fluoranthene | Nickel | 2-nitropropane |
| Benzo (k)fluoranthene | N-nitrosodiethanolamine | 2-napthylamine |
| Cadmium | N-nitrosodiethylamine | 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone |
| Chromium VI | N'-nitrosodimethylamine | 4-aminobiphenyl |
| DDT | N'-nitrosonornicotine | 5-methylchrysene |
| Dibenz(a,h)acridine | N-nitrosopiperidine | 7H-dibenzo(c,g)carbazole |
| Dibenz(a,j)acridine | N-nitrosodi-n-propylamine | Indeno (1,2,3,-cd)pryene |
| Dibenz(a,h)anthracene | N-nitrosopyrrolidine | |
| Dibenzo (a,i)pyrene | | |

Source: OSHA [1994]

**Table 3**

**Smoking Attributable Disease Relative Risk Estimates U.S. Surgeon General's Report, 1989**

| Smoking Related Diseases (ICD-9 Codes) | Age Group | Males | | Females | |
|---|---|---|---|---|---|
| | | RR Current | RR Former | RR Current | RR Former |
| **Cancers** | | | | | |
| Lip, Oral Cavity, Pharynx (140-149) | ≥35 | 27.48 | 8.80 | 5.59 | 2.88 |
| Esophagus (150) | ≥35 | 7.60 | 5.83 | 10.25 | 3.16 |
| Pancreas (157) | ≥35 | 2.14 | 1.12 | 2.33 | 1.78 |
| Larynx (161) | ≥35 | 10.48 | 5.24 | 17.78 | 11.88 |
| Trachea, Bronchus, & Lung (162) | ≥35 | 22.36 | 9.36 | 11.94 | 4.69 |
| Cervix, Uteri (180) | ≥35 | N/A | N/A | 2.14 | 1.94 |
| Urinary Bladder (188) | ≥35 | 2.86 | 1.90 | 2.58 | 1.85 |
| Kidney, Other Urinary (189) | ≥35 | 2.95 | 1.95 | 1.41 | 1.16 |
| **Cardiovascular Diseases** | | | | | |
| Hypertension (401-404) | ≥35 | 1.85 | 1.32 | 1.69 | 1.16 |
| Ischemic Heart Disease (410-414) | ≥35-64 | 2.81 | 1.75 | 3.00 | 1.43 |
| | ≥65 | 1.62 | 1.29 | 1.60 | 1.29 |
| Other Heart Disease (390-398, 415-417, 420-429) | ≥35 | 1.85 | 1.32 | 1.69 | 1.16 |
| Cerebrovascular Diseases (430-438) | ≥35-64 | 3.67 | 1.38 | 4.80 | 1.41 |
| | ≥65 | 1.94 | 1.27 | 1.47 | 1.01 |
| Atherosclerosis (440) | ≥35 | 4.06 | 2.33 | 3.00 | 1.34 |
| Aortic Aneurysm (441) | ≥35 | 4.06 | 2.33 | 3.00 | 1.34 |
| Other Arterial Disease (442-448) | ≥35 | 4.06 | 2.33 | 3.00 | 1.34 |
| **Respiratory Diseases** | | | | | |
| Pneumonia & Influenza (480-487) | ≥35 | 1.99 | 1.56 | 2.18 | 1.38 |
| Bronchitis Emphysema (490-492) | ≥35 | 9.65 | 8.75 | 10.47 | 7.04 |
| Chronic Airway Obstruction (496) | ≥35 | 9.65 | 8.75 | 10.47 | 7.04 |
| Other Respiratory Diseases (010-012, 493) | ≥35 | 1.99 | 1.56 | 2.18 | 1.38 |

**Table 4**

**Possible Interactions of Tobacco Smoke and Workplace Agents**

| Smoking Related Disease or Adverse Health Condition | Proven or Suspected Occupational Risk Factors | Comments on Possible Interaction |
|---|---|---|
| Cancers | | |
| Lip, Oral Cavity, Pharynx | formaldehyde, wood dust | |
| Pancreas | possibly petroleum hydrocarbons, metal working fluids, nitrosamines, benzidine, 2-naphthylamine | |
| Larynx | asbestos, mustard gas | possible interaction of asbestos & smoking |
| Trachea, Bronchus, & Lung | asbestos, arsenic, bis(chloromethyl)ether, chromium VI, nickel, nickel compounds, PAHs, radon, vinyl chloride, acrylonitrile, beryllium, cadmium, formaldehyde, acetaldehyde, silica, coal tar, coke oven emissions, synthetic vitreous fibers | asbestos multiplicative with smoking<br><br>silica and smoking also likely to be multiplicative |
| Urinary Bladder | 2-naphthylamine, 4-aminobiphenyl, benzidine, 4-chloro-o-toluidine, o-toluidine, 4,4 methylene bis (2-chloroaniline), methylene dianiline, benzidine-derived azo dyes | multiplicative interactions shown in case-control studies |
| Kidney, Other Urinary | asbestos, gasoline, petroleum hydrocarbons | |
| Hematopoietic Cancers | benzene, pesticides, herbicides | benzene in tobacco smoke additive with occupational exposures |
| **Cardiovascular Diseases** | | |
| All Cardiovascular Diseases | job strain, shift work, excessive cold, excessive heat, carbon monoxide, lead, carbon disulfide, nitrate esters, cadmium, methylene chloride, selected solvents such as fluorocarbons | additive effect for some tobacco agents, such as carbon monoxide and methylene chloride; workers exposed to high job strain and shift work smoke more |
| **Musculoskeletal Disorders** | | |
| Back Disorders | lifting and carrying loads, whole-body vibration, frequent bending and twisting | possible effect of smoking on low-back pain |
| **Injuries and Fatalities** | | |
| Injuries & Fatalities | many workplace risk factors depending on occupation and industry | injury risks and injury death risks higher in smokers after adjustment for confounders |

| Smoking Related Disease or Adverse Health Condition | Proven or Suspected Occupational Risk Factors | Comments on Possible Interaction |
|---|---|---|
| **Sources:** Steenland et al. [2000], Belkic et al. [2000], Cohen and Johansson [1992], Saracci [1987], Cowles [1983], NTP [2000], Vineis and Pirastu [1997], Johanson and Cohen [1997], Bjork et al. [2000], Korte et al. [2000], Kristensen [1989a,b], Petronio [1998], Burdorf and Sorock [1997], Leistikow et al [1998], Calvert et al. [1998], Clavel and Pietri [1991], Goldsmith [1997], Sacks and Nelson [1994] | | |

# Policy-Related Approaches to Reducing Environmental Tobacco Smoke Exposure in the Workplace

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## Introduction

During the past few decades, health hazards caused by environmental tobacco smoke (ETS) exposure have been increasingly recognized. Among the best-established health hazards are lung cancer in healthy adult nonsmokers and childhood disorders (e.g., respiratory tract ailments) [CA EPA 1999; NRC 1986; DHHS 1986; EPA 1992].

As scientific knowledge of health risks caused by ETS exposure has increased, understanding of such risks has grown also. Included in the change in public attitudes toward ETS is the reframing of smoking as a wider social issue beyond a personal behavior. Public policies to eliminate ETS exposure have similarly increased in frequency and scope over the past decade [Brownson et al.1997]. However, knowledge is limited regarding the overall effectiveness of these policies in controlling ETS, particularly the potential benefits of smoking bans beyond elimination of nonsmokers' exposure (e.g., smoking cessation among smokers). There is also sparse research on the effects of enforcement, worker acceptance of bans, and financial costs and benefits.

This paper describes a) workplace policy initiatives that have been designed to reduce ETS exposure, b) effects and effectiveness of such policy measures, and c) areas for future policy development and research.

## Scope of ETS Exposures and Workplace Policies

Numerous reasons exist for restricting smoking in public places.

1. ETS causes acute and chronic diseases in otherwise healthy nonsmokers.
2. The majority of the public experiences annoyance and discomfort from ETS exposure and views ETS as a health hazard.
3. Many nonsmokers do not take personal action to avoid exposure to ETS when smokers light up in their vicinity.
4. Employers could realize lower maintenance and repair costs, insurance costs, and higher nonsmoker productivity when smoking is prohibited in the workplace.
5. Restricting smoking in work settings might increase the likelihood that smokers in these settings smoke fewer cigarettes or quit smoking entirely.

## Assessing Workplace ETS Exposure

Several investigations of ETS exposure in the workplace and other settings have built the foundation for ETS control policies. In a study of 663 nonsmokers attending a cancer screening [Cummings et al. 1990], 76% of participants reported ETS exposure during the four days before the screening. The authors concluded that the workplace and the home were the primary sources of ETS exposure among nonsmokers. The best single predictor of urinary cotinine was the number of friends and family members who smoke, and who are seen regularly by the subject. In a study of 881 nonsmoking volunteers [Marcus et al.1992], employees in workplaces that allowed smoking were > 4 times more likely to have detectable saliva cotinine concentrations than those working in places with smoking bans. Among 186 former and never smokers using a self-reported exposure diary (without biochemical validation), approximately 50% of the daily ETS exposure was attributed to the workplace [Emmons et al. 1992]. However, for persons who lived with a smoker, more exposure occurred in the home than in the workplace.

The number of comprehensive studies of the levels of ETS exposure in the workplace are limited. In a review of existing studies [Siegel 1993], differences were reported in ETS concentrations by location, as measured by mean levels of nicotine in the ambient air of offices (4.1 $\mu g/m^3$), restaurants (6.5 $\mu g/m^3$), bars (19.7 $\mu g/m^3$), and residences (4.3 $\mu g/m^3$) with at least one smoker.

The most recent U.S. data from 1995–1996 show that 64% of indoor workers are covered by a 100% smoking ban in the workplace [Burns et al. in press]. The proportion of workers who work in a smoke-free workplace varies considerably by state, from 84% in Maryland and Utah to 40% in Nevada.

## Policy Options

For employers and policy makers, certain options exist regarding regulation of ETS in the workplace. Among the options, the least desirable is use of a designated smoking area without separate ventilation. This option provides only minimal protection to nonsmokers; previous studies have reported substantial exposure to ETS in workplaces with smoking areas without separate ventilation [Repace 1994]. The next option is the use of separately ventilated smoking lounges, which protect nonsmokers but are costly and could elevate lung cancer risk among smokers [Siegel et al. 1995]. Third, an option exists for use of separately ventilated smoking lounges with a recommended duration of 30 minutes or less per day, which could minimize health risks to both nonsmokers and smokers [59 Fed. Reg.15968 (1994)]. Finally, the optimal alternative is a totally smoke-free workplace.

Today, nearly all U.S. workplaces regulate smoking. In 1985, approximately 38% of U.S. workers were employed by firms that had policies restricting smoking [Farrelly et al. 1999]. According to the 1999 National Worksite Health Promotion Survey, 79% of workplaces with 50 or more employees had formal smoking policies that prohibited or limited smoking to separately ventilated areas [DHHS 2000a]. The objective for Healthy People 2010 is 100% [DHHS 2000b]. Limited systematic data exist regarding enforcement of existing policies to restrict workplace smoking. National data also suggest that, despite protections, workers in blue-collar and service occupations are much more likely to be exposed to workplace ETS than white-collar workers [Gerlach et al. 1997].

*Government policies.* Presently, the only notable federal regulation of ETS is the smoking ban on airline flights originating or arriving in the United States. This ban was strongly supported by flight attendants. Other critical federal actions have included bans on smoking in federal office buildings, the symbolic ban on smoking in the White House, and bans on smoking in childcare facilities that receive federal funds. The Occupational Safety and Health Administration proposed regulations that would either prohibit smoking or limit it to separately ventilated areas [OSHA 1994]. As of 1998, 20 states and the District of Columbia had limited smoking in private workplaces [CDC 1998]. However, only one state law (California) met the Healthy People 2000 objective of banning indoor smoking or limiting it to areas with separate ventilation. Clean indoor air ordinances at the city and county levels first appeared in the early 1970s, and have been reported to affect workplace ETS exposure [Moskowitzet al. 1999; Pierce et al. 1994]. Currently, approximately 1,000 local ordinances in the United States restrict public smoking. However, governmental laws and regulations often exclude workplaces with fewer than 50 workers from coverage.

Data are limited regarding the effectiveness of enforcement mechanisms. Further, few resources have been dedicated to enforcement of ETS ordinances, and the majority of the regulatory action is assumed to be self-enforcing.

*Private sector restrictions.* In the United States, hospitals have voluntarily implemented a nationwide smoking ban. This ban was announced in November 1991, and full implementation was required by December 31, 1993. Two years after implementation, the policy was successful, with 96% of hospitals complying with the smoking ban standard [Longo et al. 1995]. Corporations in other industries have implemented smoking bans without legislation or regulatory actions. For example, bans in fast-food restaurants such as McDonald's and Taco Bell are a response to concern for children's health and to consumer demand. Another example is the proliferation of nonsmoking rooms in motels and hotels. These changes reflect the hospitality industry's response to market demand. As such, private corporate policies not mandated by law serve as a market barometer of public opinion regarding the desirability of smoke-free indoor air. These changes are intended to protect the health of patrons, but also benefit workers in these service industries.

## Effects and Effectiveness of Workplace Clean Indoor Air Policies

Although workplace clean indoor air regulations influence nonsmokers' ETS exposure and smokers' behavior, evaluation data to quantify these effects are limited. Such changes are inherently difficult to evaluate because of the complex interaction of social forces that shape behavior, and the overlapping effects of concomitant regulatory policies (e.g., a new clean indoor air law and an increase in the cigarette excise tax). In recent years, researchers have increasingly recognized the role of the legal, social, economic, and physical environment in influencing individual smoking behavior (e.g., the smoking policy in a workplace) [Brownson et al. 1997; NCI 1991].

Regulatory interventions within the workplace environment are based on the premise that individuals are strongly influenced by the social environment in which they act. Smokers frequently respond to environmental cues when deciding whether or not to smoke [NCI 1991]. A cue to smoke can come after a work break, whereas a cue not to smoke can come after entering a smoke-free workplace. Many cues have their origins in rules regarding acceptable behaviors, such as social norms.

## Studies of Workplace Bans

*Attitudes and social norms.* Studies of awareness and attitudes toward workplace smoking restrictions and bans have been conducted in cross-sectional samples of the general population, and among employees affected by bans. Even a decade ago, in a survey of 10 U.S. communities [CDC 1991], smoking restrictions or bans were favored by the majority of all respondents in all locations including bars, restaurants, hospitals, workplaces, and government buildings. Although support for smoking restrictions was higher among nonsmokers, 82 to 100% of smokers favored restrictions on smoking in public places. Support was highest for smoking bans in indoor sports arenas, hospitals, and doctors' offices [CDC 1991]. Among city workers in Canada, satisfaction with workplace smoking restrictions was high after implementation of a new smoking law [Pederson et al. 1993].

In studies of hospital smoking bans, patients, employees, and physicians overwhelmingly support the policy; in one study [Becker et al. 1989], a majority of smokers supported a hospital smoking ban. Studies of smoking restrictions and bans in other industries also have reported high satisfaction among nonsmokers who are in favor of workplace bans. In a prospective study of a smoking ban in a large workplace [Borland et al. 1990], attitudes of both nonsmokers and smokers toward the ban were more favorable 6 months after the ban was implemented. Although a majority of smokers was inconvenienced by the ban, they also recognized the overall benefits of the ban. Among city workers in Toronto, who were subject to stringent smoking restrictions in the workplace, 58% were "very satisfied" with the workplace smoking policy [Pederson et al. 1996]. Other data from Canada demonstrate that employees in small workplaces were least knowledgeable regarding smoking restrictions and were less willing to intervene in coworkers' smoking [Ashley et al. 1997]. The literature shows how public agencies, the private sector, and organized labor can work together to implement ETS policies [National Association for Public Health Policy 1997].

*Effects on nonsmokers' exposure.* Workplace smoking bans have been effective in reducing nonsmokers' exposure to ETS. Effectiveness has been measured by the perceived air quality in the workplace after a smoking ban and by active measurement of nicotine vapor. Conversely, workplace policies that allow smoking in designated areas without separate ventilation result in substantial nonsmoker exposure to ETS [Repace 1994]. In a cross-sectional study of 25 Massachusetts workplaces [Hammond et al. 1995], a strong correlation was reported between distributions of nicotine concentrations and smoking policies. Median nicotine concentrations varied from 8.6 $\mu g/m^3$ in open offices that allowed smoking, to 1.3 $\mu g/m^3$ in workplaces that restricted smoking, to 0.3 $\mu g/m^3$ in sites that banned smoking.

Selected studies, regarding the effects of workplace smoking bans on ETS exposure, represent a "best-evidence" subset on the basis of methods developed by the U.S. Task Force on Community Preventive Services (Table 1) [Briss et al. 2000; Truman et al. 2000; Zaza et al. 2000] (see also www.thecommunityguide.org). This method of categorizing intervention studies evaluates study design and execution. Despite different metrics used among these studies, the overall body of evidence demonstrates that workplace smoking bans are effective in reducing ETS exposure. However, certain studies were conducted > 10 years ago and do not consider approaches, such as separately ventilated smoking areas, that are now being used to reduce ETS exposure in the workplace (e.g., separately ventilated smoking areas).

*Effects on smoking behavior.* Certain studies have assessed the potential effects of workplace smoking bans on employee smoking behavior. These studies have beenconducted in healthcare settings, government agencies, insurance companies, telecommunication companies, and among random samples of the working population. Effects of workplace smoking bans on employee smoking behavior can be considered from different perspectives, including impact on cigarette consumption, smoking cessation, and overall smoking prevalence within the workplace. Although > 70 English-language studies have been published regarding the effects of workplace smoking bans

worldwide, one group of studies represents a "best-evidence" subset (Table 2) [Briss et al. 2000; Truman et al. 2000]. Among these studies, consistent evidence exists that workplace bans result in a reduction of daily cigarette consumption. Based on the U.S. studies, consumption has declined by approximately 3 cigarettes/day in response to a workplace smoking ban. Effects on smoking cessation are less clear. Although studies [Longo et al. 1996] report that smoking bans increase rates of quitting, the body of evidence is limited and inconsistent. A limited number of well-designed and well-executed studies could be found, but whether workplace smoking bans contribute to overall changes in smoking cessation and prevalence in unclear.

*Overall (population wide) effects on consumption.* Two groups of researchers have summarized the overall effects of workplace smoking bans on cigarette consumption on a population basis. One study attributed recent declines in cigarette consumption in the United States and Australia to smoke-free workplaces [Chapman et al. 1999]. In the United States, workplace bans were estimated to be responsible for 12.7% of the 76.5 billion decrease in cigarette consumption during 1988–1994. If workplace bans were universal, the annual number of cigarettes forgone in the United States would increase to 20.9 billion. A related study [Farrelly et al. 1999] reported that smoke-free workplaces reduced average daily cigarette consumption by 14% relative to workers with minimal or no restrictions. That study further estimated that a total workplace smoking ban would reduce smoking prevalence by an absolute amount of 2.6% and a relative value of 10%. Although certain cross-sectional studies did not meet the criteria for "best evidence" (Table 2), overall, these studies report a consistent and substantial effect of workplace smoking policies on cigarette consumption, recent smoking cessation, and overall smoking prevalence.

## Conclusion and Recommendations

During the 1960s–1990s, substantial progress was made in protecting workers from ETS exposure. Despite these gains, health risks remain, and the following recommendations are warranted.

- As reported in other papers presented at this Proceedings, certain subgroups such as service and blue-collar workers are at highest risk for ETS exposure in the workplace and deserve special attention.

- Policies and regulations often exclude workplaces of < 50 persons, yet these employees represent a substantial workforce.

- Although bans and other restrictions have become common, there remains sparse information on the most effective means of enforcing bans at the local level.

- Beyond eliminating ETS exposure among nonsmokers, smoking bans could have additional synergistic benefits, including increased smoking cessation and reductions in the overall smoking prevalence. Well-designed and well-executed studies in this area are needed.

- The cost implications to employers of workplace ETS policies are not clear because sparse information is available regarding the costs and cost-effectiveness of various workplace smoking policies. Better assessments are needed of the effects of smoking bans on workplace productivity.

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# Smoking Cessation at the Worksite: What Works and What is the Role of Occupational Health?

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This work was partially supported by grants from the Liberty Mutual Insurance Group, the National Cancer Institute (Grant numbers 1 PO1 CA75308 and 5 RO1 CA68087), and the National Institute of Occupational Safety and Health (Grant number CDC/NIOSH RO1 OH04012).

## Introduction

In 1984, the National Institute for Occupational Safety and Health (NIOSH) concluded that simultaneous application of both health protection and health promotion would "make possible a 'synergism of prevention' to improve the health of workers through comprehensive risk reduction" [NIOSH 1984]. Health protection efforts are aimed at minimizing workers' exposures to job-related risks, such as exposure to hazardous chemicals. Protection may be maximized through the use of product substitution, engineering controls, job re-design, and, as a supplemental measure, use of personal protective equipment—measures that are generally within the domain of management decisions rather than of individual worker actions. Individual behaviors are the target of health promotion, which aims to reduce risk-related behaviors such as use of tobacco. Worksites provide an important setting for educational efforts to reach large numbers of workers not accessible through other channels. Despite these differing aims, health protection and health promotion clearly share the common goal of promoting worker health. Their complementary functions in protecting and enhancing the health of workers provide an important opportunity for coordinated efforts [Robins and Klitzman 1988; Sorensen et al. 1995].

Coordination between health protection and health promotion in the workplace has not been the norm in the United States, however. The two fields approach their objectives with differing assumptions, set differing priorities, and utilize different methods. Relationships may be strained by competition for resources in the face of scarce dollars devoted to worker health. The result has all too often been a fragmented approach to worker health [Sorensen et al. 1995; Baker et al. 1996].

Nonetheless, there have been increasing calls for a comprehensive approach to worker health, based on multidisciplinary, integrated methods aimed at creating health-promoting workplaces [Robins and Klitzman 1988; Walsh et al. 1991; DeJoy and Southern 1993; Blewett and Shaw 1995; Sorensen et al. 1995; Baker et al. 1996; Chu et al. 1997]. Indeed, there are growing precedents for worksite programs that integrate efforts to reduce behavioral risks, including tobacco use, with health protection initiatives [Marcus et al. 1986; Roter et al. 1987; Schenck et al. 1987; Maes et al. 1998; Sorensen et al. 1998b].

This paper presents a model for worksite smoking cessation that is embedded in a comprehensive approach to worker health. A comprehensive approach to worker health is defined as one which addresses multiple factors influencing worker health, including efforts to reduce exposures to workplace hazards, modify job factors to support healthy outcomes, and promote health-enhancing behaviors, including non-smoking. By definition, a comprehensive approach must target multiple levels of influence, including the levels of the work environment, the workplace organization, interpersonal supports, and the individual worker. This model draws heavily on research conducted in the tobacco control arena, and also extends that research to conceptualize a comprehensive model for worker health that incorporates both tobacco control and occupational health.

This paper recommends promising intervention strategies following a structure for intervening at multiple levels of influence within the worksite, and describes methods and priorities for future research.

### Why Integrate Worksite Tobacco Control and Occupational Health and Safety Programs?

Worksite tobacco control initiatives face a crucial challenge: the growing occupational disparity in smoking prevalence. Blue-collar workers are more likely to be smokers than workers in white-collar jobs [Sorensen and Pechacek 1986; Covey et al. 1992; Nelson et al. 1994]. The prevalence of cigarette smoking in 1987-1990 was 39.2% for blue-collar workers, 34.5% for service workers, and 24.2% for white collar workers [Nelson et al. 1994]. More recent figures by education indicate that this disparity is continuing; in 1995, 35.7% of adults with less than 12 years of education were smokers, compared to 29% among those completing high school, 22.9% among those with 13-15 years of education, and 13.6% among college graduates [National Center for Health Statistics 1999]. In addition, over time, smoking prevalence has declined more slowly among blue-collar workers compared to white collar workers. Blue-collar workers also work in settings less supportive of non- smoking. For example, these workers also report a lower prevalence of restrictive smoking policies in worksites where they are employed,

compared to the reports of other workers [Holman et al. 1998]. Health promotion programs are also less available to workers with low education levels [Grosch et al. 1998]. Evidence also suggests that blue-collar workers are less likely to participate in worksite health promotion programs than are white collar workers [Conrad 1987; Gebhardt and Crump 1990; Glasgow et al. 1993; Erfurt 1993; Sorensen et al. 1996b; Morris et al. 1999]. When they do participate, they may be less successful in changing health behaviors than are their white collar counterparts [Niknian et al. 1991].

Blue-collar workers also experience a high prevalence of hazardous exposures on the job [Walsh et al. 1991]. Among blue-collar workers, those exposed to hazards on the job are more likely to be smokers, even when gender, race and education are controlled [Sorensen et al. 1996a]. Exposures to hazards on the job and to cigarettes are of concern for several reasons [NIOSH 1979]. Toxic agents in tobacco smoke may occur in the worksite, thereby increasing exposure to the agent among smokers. In addition, smoking may act synergistically with toxic agents found in the worksite, resulting in more a profound effect than that which might be expected from the separate influences of either the occupational hazard or smoking alone. Workplace chemicals may also be transformed into more harmful agents by smoking, through the heat generated by burning tobacco; thus, as a smoker inhales a cigarette, other chemical agents are also being inhaled, becoming more toxic as they pass through the burning cigarette. Tobacco products also may serve as vectors for ingestion of workplace chemicals. The cigarette may become contaminated with hazardous agents found in the workplace, for example, when toxins on workers' fingertips are transferred to the cigarette and then to the mouth.

These dual exposures are associated with a range of short-term adverse outcomes. Walsh and her colleagues [Walsh et al. 1991] surveyed workers and managers from a large manufacturing firm about their occupational risks and health behaviors. Workers with high levels of risk on the job and in their health behaviors missed an average of three additional days per year, and reported five times as much psychological distress, including depression, anxiety and sleep disturbances, as workers in the low-risk group. In addition, they reported more symptoms of physical pain, poorer general health, and lower job satisfaction than the sample overall.

Given these multiple risks, it is imperative that successful comprehensive programs be developed to promote and protect the health of blue-collar workers. To be effective, programs need to be responsive to the priorities and concerns of these workers. Workers may view tobacco use as within a "zone of nonacceptability" for management actions, while job-related health and safety issues may be seen as a too-often ignored responsibility of management [Barnard 1968; Green 1988]. Indeed, the risks that matter the most to them may be those that have been identified as key priorities in risk communication research: risks that are involuntary, outside personal control, undetectable, and that seem unfair [Bradbury 1989; Baker 1990; Fischoff et al. 1993]. These risk features often characterize occupational hazards.

Management actions to reduce worker exposure to hazardous substances may be a higher priority to workers than more personal health behavior changes such as smoking cessation. Skepticism about management's commitment to improve worker health may reduce workers' interest in participating in health promotion programs at work [Sorensen et al. 1995; Warshaw and Messite 1998; Morris et al. 1999]. Furthermore, reduction of job risks may be required to gain credibility with this audience, and to increase its receptivity to health education messages about individual health behaviors [Green 1988; Sorensen et al. 1998b]. One study of blue-collar workers found that workers who reported that their employers had made changes to reduce exposures on the job were significantly more likely to have participated in smoking control and nutrition programs than workers not reporting management changes [Sorensen et al. 1996b].

Indeed, wellness programs that fail to address the hazards of work miss significant sources of health-related problems and costs, both to individual workers and employers. Yet health protection programs that ignore personal risk factors may be underestimating workers' understanding of the complexities of health and well-being [Walsh et al. 1991]. Results of a study of craftspersons and laborers in 22 worksites in Massachusetts indicated that workers are concerned about their dual risks [Sorensen et al. 1996a]. They found that smokers exposed on the job to chemical hazards were more than three times more likely than those unexposed to be thinking of quitting smoking or taking action to quit, controlling for gender, race and education. Among men, concern about chemical hazards was further associated with an increased interest in quitting. Awareness of the interactive and synergistic effects of tobacco smoking and exposure to hazardous materials may raise smokers' motivations to quit smoking. In addition to the importance of the direct interactions between tobacco use and exposures to occupational hazards, workers' perceptions of job exposures have the potential to influence interest in quitting.

Although there is increasing discussion of the importance of comprehensive programs integrating health promotion and occupational health efforts in the worksite, few studies have been conducted to date to assess their

impact. One study that provides promising preliminary evidence was the WellWorks study, conducted as part of the National Cancer Institute's national worksite initiative known as the Working Well Trial. Four research intervention sites tested the effects of a comprehensive worksite cancer prevention model aimed at nutrition and smoking, using a randomized controlled design in 114 worksites nationwide, including 24 at the WellWorks site [Abrams et al. 1994b]. Only the WellWorks Project tested the effectiveness of a model integrating health promotion and health protection [Sorensen et al. 1996c]. This intervention aimed to integrate messages on tobacco control, nutrition, and occupational health in programs targeting both workers and management. This site was the only study center in which a significant result for smoking cessation was observed; the six-month quit rate in the intervention worksites was 17.3%, compared to 12.7% in the control sites ($p=0.037$) [Sorensen et al. 1996c]. Analyses of the cohort of workers participating in both the baseline and final surveys indicate that this intervention was especially effective for skilled and unskilled workers, for whom there were significantly greater improvements in fiber consumption than other workers ($p<.01$), and a trend toward higher smoking cessation rates [Sorensen et al. 1998b]. A follow-up of this study currently being completed addresses the question, does the addition of worksite health protection increase the effectiveness of worksite health promotion only? These results provide promise that a comprehensive approach to worker health may be particularly effective in promoting the health of blue-collar workers.

## Smoking Cessation Programs at the Worksite

A comprehensive approach integrating health promotion and health protection has not typically characterized tobacco control at the worksite, although in some cases tobacco control has been incorporated into a broader health promotion program also addressing other health behaviors [O'Donnell 1994; Wilson et al. 1996; Heaney and Goetzel 1997]. State-of-the-art tobacco control programs at the worksite focus on two levels of influence. At the individual level, worksite smoking cessation initiatives aim to help smokers quit smoking. At the level of the worksite environment, tobacco control policies serve the dual purpose of protecting non-smokers from the hazardous effects of environmental tobacco smoke, and promoting an environment supportive of non-smoking.

Worksite tobacco control policies are not a central focus of this paper; another paper by Ross Brownson in this Proceedings addresses the topic of "Policy-related approaches to reducing environmental tobacco smoke exposure in the workplace." However, tobacco policies are a key component of an overall workplace tobacco control effort, and are central to supporting smoking cessation among workers. Worksite policies on tobacco have been shown to decrease worker exposure to environmental tobacco smoke [Stillman et al. 1990; Marcus et al. 1992; Hammond et al. 1995] and contribute to worker reductions in smoking, including quitting [Paulozzi et al. 1992; Kinne et al. 1993; Woodruff et al. 1993; Brigham et al. 1994; Pierce et al. 1994; Eriksen and Gottlieb 1998]. Employer efforts to promote compliance with smoking policies can contribute to an overall climate supportive of nonsmoking [Sorensen et al. 1992].

In 1992, 40% of private sector worksites employing more than 50 workers offered smoking cessation programs, and 86% reported having a formal policy that prohibits or restricts smoking, including 34% that banned smoking within the worksite [USDHHS 1992]. The 1994 National Health Interview Survey assessed availability of worksite health promotion programs among respondents employed in worksites with at least 50 employees, including both public and private worksites. Forty-three percent reported that smoking cessation programs were available in their worksite, the highest mean availability of worksite health promotion programs. However, only 4.6% of respondents overall reported participating in these programs [Grosch et al. 1998].

Smoking cessation programs at work have been evaluated using a range of study designs; numerous excellent reviews of this literature are available [Bibeau et al. 1988; Klesges and Cigrant 1988; Fielding 1991; Strasser 1991; Eriksen and Gottlieb 1998]. Over time, the number of studies has increased along with the rigor of the study designs employed. Studies of worksite smoking cessation programs might be categorized into two groups. The first set is designed to assess the effects of intensive programs for individual workers who smoke. Smokers are recruited to the study and generally randomly assigned to treatment condition. These studies are designed to test the effectiveness of different smoking cessation methods among smokers interested in quitting. In general, more intensive programs, with multiple sessions and multiple components, yield higher quit rates than shorter term, less intensive interventions [Fielding 1991; Eriksen and Gottlieb 1998]. Because these programs include highly motivated volunteers who are ready to commit to a quit-smoking program, they may miss important segments of the working population who are not interested in participating in intensive programs.

A second set of studies uses the worksite as the unit of randomization in order to assess the effects of worksite-wide programs designed to reach this broader audience. Using a range of intervention strategies, worksite-wide programs promote non-smoking among smokers at all stages of readiness to quit. Although worksite-wide programs are likely to result in lower quit rates than those targeting smokers motivated to quit smoking, their overall impact may be greater. From a public health perspective, the "impact" of an intervention is a product of both its efficacy in changing behavior and its reach, meaning the proportion of the population reached either through their direct participation, or indirectly through diffusion of intervention messages throughout the worksite [Abrams et al. 1996; Glasgow et al. 1999]. Worksite-wide programs aim to reach a broad audience within the worksite, creating an overall climate supportive of non-smoking. The literature increasingly reports the results of studies that focus on worksite-wide smoking cessation initiatives.

Smoking cessation programs thus include a range of initiatives, including smoking cessation clinics or classes, medical interventions, minimal intervention programs, incentives and competitions, and social and environmental supports. The full range of programs is important to a worksite-wide tobacco control program, because the combination of strategies increases the chances of influencing smokers at varying stages of readiness to quit smoking.

*Smoking cessation group programs:* Many worksites offer the same types of smoking cessation programs originally developed and offered in clinical settings, or, in some cases, provide referrals to clinic- or community-based programs, such as through the American Cancer Society or the American Lung Association, for-profit programs (e.g., Smoke Enders), or health care organizations [Fielding 1991]. One meta analysis of 20 controlled worksite-based studies found a weighted average follow up quit rate from all interventions of 13% [Fisher et al. 1990]. Eriksen and Gottlieb [1998] reviewed 25 studies, with 6- to 24-month quit rates of 37 cessation groups; quit rates ranged from 0-91%, with the median cessation rate of 23%. Several of the more rigorous studies showed markedly lower rates. Interpretation of the results of many of these studies is thus limited by the weaknesses in the designs employed, including failure to define the characteristics of the population offered participation in the study, failure to include dropouts in the calculation of quit rates, use of very small samples, inadequate follow-up of cessation maintenance, and use of non-randomized designs [Fielding 1991; Eriksen and Gottlieb 1998].

*Medical interventions:* Increasingly, worksite smoking cessation programs have been supplemented by medical interventions, including physician advice for high risk smokers to quit and the use of nicotine replacement therapy with brief voluntary counseling [Rose and Hamilton 1978; Li et al. 1984; Whitney and Harris 1994; Eriksen and Gottlieb 1998].

*Minimal-contact intervention programs*: Minimal-contact interventions often are used to promote smoking cessation among those not yet ready to quit, or to provide help with quitting for those not willing to invest time and energy into a group cessation program. Minimal-contact interventions may include promotion of a telephone help line or the Great American Smoke-out, self-help interventions such as written materials and short videos, and assessments with feedback, such as that carbon monoxide assessments. Within the context of a worksite-wide tobacco control program, minimal-contact intervention strategies may serve to engage smokers in thinking about quitting, increase participation in group programs, and support worksite norms supportive of non-smoking [Eriksen and Gottleib 1998].

*Incentives and competitions:* Incentives may be either monetary or nonmonetary. For example, a ban on smoking at the worksite may provide an incentive for smokers to quit. Employers have often provided monetary incentives for quitting and maintenance of cessation, including reduction in the cost of participation in a smoking cessation program, bonuses or payments for smoking cessation, or differential premiums for health or life insurance benefits [Fielding 1991]. Smoking cessation competitions have frequently been used in combination with incentives. Competitions have been initiated between different worksites, between departments or other groups within a worksite, or among individual workers. Competitions have the potential to increase recruitment rates to smoking cessation programs. Eriksen and Gottleib concluded that competitions also may increase cessation rates [Eriksen and Gottlieb 1998]. Combining competition and incentive approaches has also been associated with a higher percentage of the total smoking employee population quitting compared to behavioral approaches alone [Fielding 1991].

*Social and environmental supports:* The success of smoking cessation programs is clearly influenced by the supportiveness of the worksite environment. A supportive environment may be reflected in the existence of a formal health promotion program and a formal smoking policy that is uniformly enforced [Fielding 1991]. Worksite tobacco control policies are central to building a worksite climate supportive on non-smoking. In addition, some worksite smoking cessation programs systematically incorporate strategies for building social support for quitting. Eriksen and Gottleib [1998] reviewed four randomized trials that examined the addition of social support; only one of these studies yielded a consistent, significant incremental effect of social support. Environmental support may also be provided by labor unions, which may negotiate for non-smoking benefits or institute their own tobacco control policies and thereby contribute to an overall climate supportive of non-smoking [Sorensen et al. 2000].

There are numerous advantages to locating smoking cessation programs in worksites. As this brief review indicates, worksites offer the potential for support of long-term cessation; mobilization of peer support for cessation; use of environmental supports, including smoking policies; incorporation of incentives and competitions and policies; and the possibility of offering multi-component interventions repeatedly over time as a means of building and sustaining interest in quitting. In general, corporate interest in and support for tobacco control has been considerable [Fielding 1991; Stokols et al. 1996]. Worksites also provide a convenient setting for conducting outcome evaluations, although there has been considerable variability in the rigor of the studies conducted to date. Although the findings indicate that intensive, multi-component programs may result in the highest quit rates, participation in such intensive programs remains low, particularly on the part of some high risk groups, including Blue-collar workers. These results also indicate that less intensive interventions, when combined with high participation rates, can have an impact on the total population of smokers at a worksite [Stokols et al. 1996; Eriksen and Gottlieb 1998]. This body of literature provides a firm basis for the next generation of worksite tobacco control initiatives. A comprehensive approach integrating tobacco control into a broader worker health program provides particular promise for influencing the smoking habits of Blue-collar workers, among whom smoking prevalence remains high.

## Promising Worksite Interventions Promoting Tobacco Control: A Comprehensive Approach to Promoting Worker Health within Healthy Worksites

Worker health is influenced by a range of workplace factors, including potential exposures to workplace hazards and job design factors, such as those contributing to job strain. Employers exert primary control over these workplace factors, and have the responsibility to provide a safe and healthy work environment. Worker health is also influenced by workers' decisions about their health behaviors, including tobacco use. A comprehensive approach to worker health addresses these multiple factors influencing worker health. In contrast to the often-observed piecemeal approach, a comprehensive program for worker health benefits from offering multiple and coordinated interventions targeting both workers and management [DeJoy and Southern 1993; Chu et al. 1997]. The effectiveness of tobacco control efforts is, therefore, likely to be enhanced when these efforts are conceptualized as part of an integrated, comprehensive program promoting worker health within a healthy workplace.

This comprehensive model has a strong theoretical foundation. Research on health behavior change, including smoking cessation, has been guided by theoretical models developed by the behavioral and social sciences. Although evidence is still accruing about the efficacy of workplace interventions integrating tobacco control and occupational health, it is possible to identify promising intervention strategies by drawing on the preliminary evidence on effective worksite interventions and basing conclusions on a theoretical framework for efficacy.

A range of theoretical frameworks have suggested that worker health is the result of a complex interplay of factors involving the individual worker and the immediate work environment, as well as characteristics of the larger contexts in which both the individual worker and the worksite are embedded [Robins and Klitzman 1988; Stokols 1992; Sorensen et al. 1995; Baker et al. 1996]. The social ecological model provides a structure for understanding these multiple levels of influence, including at the individual, interpersonal, organizational, community, and public policy levels [Bandura 1986; McElroy et al. 1988]. This model offers a framework for recognizing the importance of underlying factors associated with worker health, including exposures to hazards in the worksite environment. Accordingly, the effectiveness of worksite tobacco control interventions will be enhanced when coordinated interventions aim to promote cessation among individual smokers, build social support for quitting and social norms that support non-smoking, engage management in assuring a healthy work environment; involve workers' families in non-smoking initiatives, and provide links to community and public policy initiatives that support tobacco control as

well as broader efforts promoting worker health. This model also provides a framework for moving beyond the individual as the locus of intervention and responsibility for health, in recognition of management's central role in worker health. Intervention strategies promising to improve the effectiveness of tobacco control require coordinated efforts at the organizational, interpersonal, and individual levels.

## Intervening at the Organizational Level

To be effective, interventions at the organizational level must involve key stakeholders, including management, workers, and unions. Policies supporting worker health include those influencing the work environment and the organization of work.

*Assure management commitment and support*. Management commitment to a comprehensive worker health program provides a key foundation for success. Legislation on occupational health and safety and workers' compensation has consistently placed responsibility on employers for providing safe and healthy working conditions [DeJoy and Southern 1993]. Management sets the directions for worker health, either through clear statements of priorities or through tacit understandings transmitted through administrative hierarchies. Support may be reflected in corporate plans or mission statements, providing written direction that enables workers to act in the interests of their health, for example, through participation in health and safety committees [Emmons 2000]. Such documentation also serves as the "basic blueprint" for bringing various groups together within the organization, including those representing benefits/employee relations, employee assistance, health promotion, medical services, and occupational safety and health [DeJoy and Southern 1993]. The vision provided by management for an integrated worker health program can help to overcome the boundaries of the traditional, professional domains of those responsible for worker health. This coordination may be made possible through a committee structure which provides opportunities for worker participation. Top management commitment also provides the basis for training of direct supervisory personnel to assure their support for and participation in a comprehensive worker health program [Robins and Klitzman 1988].

The WellWorks study observed that while many companies were interested in participating in health promotion programming, some expressed reservations about committing to the occupational health component [Sorensen et al. 1995]. Management may similarly be reluctant to devote worker time for health and safety programs. Further research is needed to identify strategies to elicit management support for comprehensive programs.

Management support also serves to sustain and institutionalize programs over the long term. Assigning a person responsibility for health programs and committing budgetary support to these endeavors are strong indicators of management commitment likely to help sustain programs over time. To observe change in health outcomes, programs must be of sufficient duration to provide on-going, persistent messages supporting health, including non-smoking [Heaney and Goetzel 1997; Sorensen et al 1995]. Programs sustained over time and interwoven into the human resource strategy of the organization are likely to be accepted as the norm for the worksite [Heaney and Goetzel 1997].

*Involve workers in program planning and implementation*: Worker participation in program planning can assure that programs respond to worker needs and priorities. Programs are likely to be more effective when they are based on an understanding of workers' concerns about health risks on the job. By identifying these priorities, smoking can be addressed in the broader context of the worksite. Workers can also provide direction on ways to promote smoking cessation that work best in their worksite.

Such input may be achieved in a variety of ways. Health and safety committees provide an overall structure for jointly engaging workers and management in efforts to promote a healthy workplace, and their roles can logically be expanded to encompass tobacco control and other health behaviors. Because these committees are an established part of the workplace operations, they provide a means of institutionalization of program efforts over the long term. Alternatively, companies may form wellness committees that assist in planning tobacco control initiatives. Where these committees exist, their roles may similarly be expanded to incorporate health and safety concerns as well. Participation of line workers in committees may be limited, however, by constraints placed on workers' time away from their jobs, and requires the support of line supervisors to assure that they are able to participate consistently. In addition, because of obvious power differentials in joint worker-management committees, workers may not feel free to speak openly about their concerns in the presence of management - again underlining the importance of management commitment.

Additional strategies may be used to give workers a voice in program planning, such as focus group interviews with workers or conversations with workers during break times. In general, these strategies allow program planners to understand the context of workers' lives, in order to develop meaningful programs that are responsive to workers' priorities and concerns. These strategies may also have the side benefit of engaging workers in the intervention efforts. Worker participation in program planning may also contribute to the development of generic skills that may be applied across health issues, such as problem identification, problem solving, and communication skills [Blewett and Shaw 1995].

Participatory methods are also important as a basis for educational strategies. Use of learner-centered models can build a sense of worker control, which goes beyond transmission of information and skills, and may facilitate joint problem-solving in support of tobacco control and other aspects of worker health [Luskin et al. 1992; Wallerstein and Weinger 1992; Baker et al. 1996]. Interventions which actively involve participants in the planning, implementing and evaluating may be health-enhancing in and of themselves [Israel et al. 1996].

*Involve unions*: Unions also provide a voice for workers. Historically, labor unions have played an important role in advocating for occupational health and safety. Protecting the health of their members is a key union mission. Within the area of tobacco control, however, organized labor represents an important underutilized resource, and provides an avenue for accessing blue-collar workers. A recent study of organized labor's positions on worksite tobacco control policies found that nearly half of local unions surveyed supported worksite smoking bans or restrictions, and only 8% actively opposed worksite tobacco control policies. In addition, 35% provided smoking cessation assistance or bargained for reduced insurance rates for their non-smoking members [Sorensen et al. 2000]. Unions may also play a role in obtaining coverage for nicotine replacement therapy, now recommended as a standard component of smoking cessation programs [Fiore et al. 1996].

Planning for tobacco control initiatives within unionized worksites needs union participation. Labor-management relationships are likely to influence workers' response to any program, regardless of its assumed neutrality or benefit [Baker et al. 1996]. For this reason, programs allied too closely with management may be viewed with skeptism by union members. With union input, programs can be effectively crafted to meet the needs of blue-collar workers. Unions may be instrumental in providing workers a voice in program design and implementation, or in balancing overzealous management smoking policies, such as the elimination of break times (in the absence of smoking breaks) or the discriminatory practice of not hiring smokers [National Association of Public Health Policy 1997].

In unionized worksites, concerns may arise that certain efforts may enter into areas that traditionally have been reserved for collective bargaining. Failing to engage unions in such efforts takes power and control away from the collective bargaining process and hence may be viewed as a threat to the union [Green 1988]. Unions' right to negotiate tobacco control policies has been supported by the National Labor Relations Board and by legal precedence [National Association of Public Health Policy 1997]. When unions have grieved worksite smoking policies, the most common reason has been that policies were unilaterally imposed by management, without a voice given to the union [Sorensen et al. 1997]. It is thus imperative that worksite tobacco control policies be negotiated with unions.

*Promote policies supporting a healthy worksite environment*: The work environment clearly plays a crucial role in worker health. The hierarchy of controls model provides a useful framework for promoting changes to reduce exposure to occupational hazards, following a recommended sequence for control of hazards beginning with control as close to the source as possible [Office of Technology Assessment 1985]. The ideal choice is the substitution of safer substances for those that are hazardous, thereby removing the potential hazard. Engineering controls provide a second line of defense for the control of hazards, followed by administrative controls, such as job redesign or job rotation. Personal protective equipment is recommended only as a last line of defense when substitution or engineering controls are not possible; by itself it is not an acceptable methods of control because its effectiveness is highly variable and not reliable. Management actions to assure a safe and healthy work environment are a central component of a comprehensive worker health program. As described above, these actions also may indirectly support workers' interests in quitting smoking.

Restrictions on smoking are an important component of an overall policy on indoor air quality, aimed at reducing workers' exposures to environmental tobacco smoke as well as other air quality hazards [National Association of Public Health Policy 1997]. These policies also contribute to building an overall work climate supportive of non-smoking by shaping social norms supportive of non-smoking and providing external incentives for cessation.

*Address the organization of work and job characteristics influencing worker health:* Job characteristics and the organization of work are also important correlates of smoking behavior. Workers in jobs with the greatest potential for exposure to hazardous substances are also most likely to be smokers [Sorensen et al. 1996a]. Shift workers have particularly high rates of smoking [Knutsson and Nilsson 1998]. Job content and the dynamics of a workplace have ramifications for a worker's health. These effects clearly extend well beyond blue-collar workers to influence workers in a range of jobs. "Job strain" results when workers face high psychological workload demands combined with low control or decision-making latitude in meeting those demands [Karasek and Theorell 1990], and may be compounded by a lack of social support [Johnson and Hall 1988]. Other specific stressful working conditions can include involuntary overtime, piece-rate work, inflexible hours, arbitrary supervision, and deskilled work [Landsbergis et al. 1993]. Strong evidence exists that job strain is a risk factor for heart disease, [Siegrist et al. 1990; Schnall et al. 1998] and is associated with smoking [Green and Johnson 1990; Johansson et al. 1991; Landsbergis et al. 1998], as well as other deleterious health outcomes, including depression and chemical dependencies, sedentary behavior; and stomach ulcers [Johansson et al. 1991]. Landsbergis and colleagues [Landsbergis et al. 1997] found that increases in job decision latitude over three years were associated with decreases in smoking.

These characteristics of work must be considered part of a comprehensive plan for promoting worker health [Karasek and Theorell 1990; Israel et al. 1996; Landsbergis et al. 1998]. Assessment of job content and job design may lead to necessary changes in the organization of work, and have been described as central in a range of worksite interventions [Israel et al. 1996; Maes et al. 1998]. Breaks must be structured to provide fair and adequate rest; in some workplaces, taking a smoking break may be the only valid reason for a worker to take a break from physically or mentally demanding work. Changes in such policies are of paramount importance to a successful tobacco control initiative. Understanding the social meanings associated with smoking in work settings is critical to designing comprehensive interventions.

## Intervening at the Interpersonal Level

Interventions at the interpersonal level include promoting social support and social norms supportive of worker health and linking interventions to workers' social contexts.

*Promote social support and social norms supporting worker health:* Co-worker support is important in quitting smoking [Green and Johnson 1990]. Co-worker discouragement of quitting has been associated with lower confidence in the ability to quit smoking [Sorensen and Pechacek 1986], and the pressure that smokers feel from nonsmokers has been shown to be effective in motivating them to quit [Gottlieb and Nelson 1990]. Blue-collar workers are likely to experience social norms less supportive of tobacco control than are white collar workers [Morris et al. 1999; Sorensen et al., unpublished]. Social norms and social support, from both co-workers and supervisors, are also important in workers' compliance with protective recommendations [de Vries and Lechner 2000]. A study by Morris and colleagues [1999] found that not only did white collar workers feel more support from their supervisors to participate in health promotion programs, but they also reported more support from their coworkers and a stronger employer health orientation than did blue-collar workers.

It is important to incorporate social resources in the job setting to support smoking cessation, as well as other health behaviors and occupational health and safety. For example, buddy systems and support groups have been used to encourage smoking cessation [Naditch 1985; Harris 1986; Baker et al. 1996]. The effectiveness of social support is exemplified in a study by Erfurt, Foote, and Heirich [Erfurt et al. 1991], in which they compared the effects of four interventions involving: (1) a wellness screening; (2) a wellness screening plus health education; (3) the same, plus follow-up counseling; and (4) all of the prior, plus peer support groups, buddy systems, health promotion classes, and plant-wide activities. The highest level of change was seen the fourth group.

Peer-led programs similarly may provide a strategy for dissemination of tobacco control information, a source of role models for effective behavior change, and a means of fostering positive social norms. Peer-led models may fit particularly well into collaborations with labor unions [Allen et al. In Press]. It should be noted, however, that the use of peers is a supplement to health professional involvement; peers need a strong orientation and training program and ongoing support to be effective [Corneil and Yassi 1998].

*Link interventions to workers' social contexts:* The social contexts in which workers live —both on and off the job—clearly influence workers' health behaviors, including tobacco use. These forces are also likely to shape the

effectiveness of interventions. On the job, it is important to understand, for examples, how time on the job is structured, the meaning of smoking within one's work group, and work stressors, as described above. The worker's social context also includes socioeconomic conditions, as well as one's physical, social, and cultural environment influencing access to health information, social support, social networks, social norms, and cultural beliefs and attitudes regarding health [Stokols et al. 1996; Corneil and Yassi 1998; Emmons 2000]. For example, workers' interest in quitting smoking may be influenced by the smoking patterns of family members and friends, the strains presented by balancing multiple roles at home and at work, and cultural norms and beliefs. A classic study by Syme [1978] of people with hypertension illustrates the importance of addressing such underlying social and economic conditions. In a randomized, controlled study, hypertension was most likely to be controlled among those patients who received not only information about hypertension, but also were given the opportunity to discuss family difficulties, financial strain, employment opportunities, and where appropriate, were given support and assistance. In the worksite setting, it is important to recognize and respect boundaries between work and workers' private lives. At the same time, programs may be more effective when they take into account factors outside the work environment that may be influencing tobacco use and success with quit attempts. Programs providing links between work and home may be particularly effective in promoting worker health [Sorensen et al. 1999; de Vries and Lechner 2000].

## Intervening at the Individual Level

Interventions at the individual level must begin by reducing structural barriers influencing workers participation in intervention. For maximum reach, interventions must target workers at varying stages of readiness to make changes. Information about tobacco control can also be incorporated into hazards communications programs or other programs on worker health. Recent advances in tailoring messages to individual workers provide promise for increasing the efficacy of these interventions.

Reduce structural barriers for individual workers to participate in interventions. As discussed above, blue-collar workers are less likely than white collar workers to participate in health promotion programs. It is therefore necessary to address structural barriers to workers' participation. For blue-collar workers, supervisors function as gatekeepers controlling worker access to health promotion activities [Morris et al. 1999]. For instance, to keep production lines moving supervisors may refuse to allow workers to attend programs on company time. Further barriers may include working over-time, shift work, having a second job, car-pooling to work, long distances between the plant and the employee's home, and responsibilities at home [Alexy 1990].

As noted above, management support and commitment can serve to reduce these structural barriers by placing high priority on a comprehensive program supporting worker health, with the same levels of support communicated for different groups of workers. Given the push to keep production moving, it may also be necessary to structure smoking cessation programs around the schedules of line workers, bringing programs to production floors and break rooms, or timing programs to fit within the break times of workers.

An additional structural barrier to quitting smoking is access to nicotine replacement therapy (NRT). Although NRT was previously covered under some prescription policies, since it has become available over the counter such coverage is no longer available. Evidence indicates that use of NRT doubles quit rates; the AHCPR Guidelines on Best Practices in Smoking Cessation recommends that NRT be provided to all smokers [Fiore et al. 1996]. Access to NRT is of particular concern to low income workers.

*Provide interventions for smokers at varying stages of readiness to quit:* As noted above in the discussion of smoking cessation programs, a range of programs have been offered, assuming that smokers are at varying stages of readiness to quit smoking. A comprehensive tobacco control program needs to provide a full spectrum of interventions, ranging from minimal interventions to promote smoking cessation among those not yet ready to quit, to incentives and competitions, to smoking cessation group programs and medical interventions for those seeking support for cessation efforts. As a means of addressing worker concerns about exposure to occupational hazards, worksite tobacco control programs can incorporate messages about occupational health and safety. Information about the interactive and synergistic effects of tobacco smoke and occupational hazards may provide further motivation for smokers to quit.

*Build tobacco control messages into hazard communication programs:* OSHA's Hazard Communication Standard requires training of workers about the hazardous substances they work with, the health and physical hazards

associated with these substances, methods of detection of exposure, and methods of protection of adverse effects [48 Fed. Reg.* 53280 (1983)]. Some hazard communication programs have used train-the-trainer models to assure that those familiar with local plant conditions are involved in training and that these key individuals "buy in" to the program, thus building a system-wide impact [Robins and Klitzman 1988]. While focusing on their central mission of occupational safety and health, it is feasible to incorporate tobacco control
[48 Federal Register 1983] messages into these existing worker training programs. An excellent example of this is provided by the Workplace Hazard and Tobacco Education Project, sponsored by the Labor Occupational Health Program in Berkeley, California [Baker et al. 1988]. This hazard communication program incorporates information on tobacco control, encouraging workers to view tobacco smoke as an occupational hazard. The purposes of the classes are to provide information to workers about toxic hazards they face on the job, including the extra risk from tobacco smoke, and to explain options workers have to protect themselves and reduce their risks on the job. Smoke-free workplaces are promoted as a way to control workers' exposures to tobacco smoke, as part of overall strategies to reduce workplace exposures to other hazardous substances. Such integrated programs acknowledge the complex interplay of factors influencing workers' health.

*Tailoring interventions to individual workers when possible:* Smoking cessation programs are increasingly moving away from the one-size-fits all approach to intervention for individuals, to utilize "tailored" approaches to assist with cessation [Sorensen et al. 1998a]. "Tailoring" provides a means of increasing the intensity of interventions delivered. Tailored interventions typically use print communication [Velicer et al. 1993; King 1994; Rimer et al. 1994] or telephone counseling [Curry et al. 1995] to enhance the relevance of interventions to the daily lives of the target population, thereby increasing the likelihood of achieving short-term or sustained intervention effects [Abrams et al. 1996; Rimer and Glassman 1998]. Studies are beginning to examine the application of tailored interventions to worksite settings [Willemsen et al. 1998]. These tailored programs have not yet examined the option of tailoring messages on workers' occupational exposures, which may offer an additional strategy for motivating workers to consider quitting smoking.

## Methodological Challenges in Conducting Tobacco Control Research in Worksites

In the past two decades, an increasing number of studies have been conducted in
worksites to assess the effectiveness of interventions targeting health behaviors, including tobacco control [Jeffery et al. 1993; Sorensen et al. 1993; Salina et al. 1994; Glasgow et al. 1995; Sorensen et al. 1998b; Sorensen et al. 1996c; Glasgow et al. 1997; Sorensen et al. 1999]. In general, the randomized controlled design is the accepted standard for assessing the efficacy of these interventions, with change being assessed from baseline to follow-up and compared between conditions [Koepsell et al. 1992; Susser 1995; Sorensen et al. 1998a]. There are several important challenges presented by worksite research on tobacco control that must be considered in conducting worksite-based research.

*Unit of intervention and evaluation:* The worksite is the appropriate unit of randomization in these worksite studies, since interventions are aimed at the entire population of workers at the site. The interventions are by definition conducted at the worksite level, given the importance of comprehensive programs linking tobacco control to a range of other factors influencing worker health [Greene et al. 1994; Koepsell et al. 1995]. As a consequence, cost and feasibility often limit the scale of these studies, and also restrict statistical power. The small number of units may also contribute to selection bias, particularly in cases where the allocation of worksites is non-random [Koepsell et al. 1992; Mittelmark et al. 1993; Murray 1995].

Although the unit of assignment is the worksite, or cluster, studies in which smoking cessation is the primary outcome measure change at the individual level. In such cases, it is necessary to control for cluster in analyses [Donnar and Klar 1996; Koepsell 1998]. Cluster members cannot be assumed to be independent, since individual workers are likely to share a variety of characteristics.

*Assessment of the outcomes.* Cost may force many worksite studies to use self-administered surveys for collection of self-reported data. Valid and reliable instruments appropriate for self-administration provide an important basis for

collection of population-level data. Standard measures of tobacco use are available. A review of the factors related to false-reporting of smoking concluded that misreporting rates are relatively low, typically near zero and seldom exceeding 5% [Velicer et al. 1995].

*Timing of the assessments.* Most of the studies reviewed included only post-intervention follow-ups, and therefore have not evaluated long-term or delayed intervention effects. Future studies could contribute to our understanding of the effectiveness of worksite tobacco control initiatives by evaluating their long term effects.

*Sampling issues.* In these trials, sampling is a concern at two levels. Worksites selected for inclusion in the study must be representative of a larger population of worksites, and individuals surveyed as part of the outcome assessment must represent the work force from which they were sampled. The self-selection of worksites into studies may contribute to a response bias at the worksite level. In studies reviewed by Sorensen and her colleagues [1998a], between 25% and 62% of eligible worksites invited to participate actually joined the study. When compared to other worksites, participating worksites have been found to have more health promotion programs [Heimendinger et al. 1995].

In comparison to companies declining participation, recruited worksites have been reported to have more favorable financial outlooks and to employ fewer workers [Biener et al. 1994]. The potential for response bias may contribute to the observed secular trends in comparison worksites, perhaps reflecting a "healthy worksite" effect.

The response rates of workers to surveys have been similarly quite variable, ranging from an overall response rate of 22% to 87% in worksites [Sorensen et al. 1998a]. Differential response rates are of particular concern; in the worksite trial that found no significant effects - Take Heart I [Glasgow et al. 1995] - the response rate was 20% higher in the control group than the intervention group, a factor potentially capitalizing on secular trends, and thus masking intervention effects.

*Environmental assessments.* Environmental outcomes provide necessary measures of the effectiveness of environmental interventions, including those targeting occupational exposures as well as tobacco control policies. In addition, environmental assessments may provide indicators of change preceding individual outcomes, provide alternative measures of change in addition to self-reports, and may identify competing explanations for observed individual outcomes [Sorensen et al. 1998a]. For example, increases in indicators of management support for worker health may precede changes in policies supporting worker health. Assessments of changes in exposures to occupational hazards may also be incorporated into studies of comprehensive worker health interventions [LaMontagne et al. In Review].

*Assessment of intervention implementation:* Recent trials have included rigorous assessments of the implementation of interventions through process tracking systems measuring such indicators as dose, or the amount of intervention delivered; fidelity, or the extent to which the intervention was delivered as planned; and program coverage, including participation [McGraw et al. 1989; Corbett et al. 1991; McGraw et al. 1994; Scheirer et al. 1995; Hunt et al. 2000]. These data provide important information that enhances the ability to interpret outcome assessments, identify competing explanations for observed effects, and measure exposure to the intervention [Jacobs et al. 1988; Scheirer 1988; McKinlay 1993].

*Assessment of mediating mechanisms:* There is a need for improved specification of effective intervention methods through assessment of the pathways through which interventions operate. As noted above, studies of worksite smoking cessation programs have focused either on the evaluation of clinic programs, or have evaluated more comprehensive worksite programs as an intervention "package" with multiple components, with the results generally compared to a non-intervention control. Such a comparison does not permit us to disentangle the impact of individual intervention methodologies on behavior change [Mittelmark et al. 1993; Baranowski et al. 1997].

*Assessment of cost effectiveness:* Despite frequent claims that health promotion and occupational health and safety programs make "good business sense," evidence is needed on the long term cost effectiveness of these efforts [DeJoy and Southern 1993]. Such analyses can make use of new systems that allow for tracking costs via insurance claims and disability claims, with links provided to data on program participation and program costs.

Qualitative research methods may provide further insight into our interpretation of the results of quantitative studies [Fortmann et al. 1995; McKinlay 1995]. In addition, qualitative methods play a role in program development,

including defining the program's goals and objectives, identifying appropriate delivery channels, designing educational materials, tracking audience exposure and reaction, and, finally, refining the program [Gilliam and Hollander 1990].

Qualitative methods can be particularly useful in helping to understand the contexts of workers' lives and how their day-to-day experiences influence their patterns of tobacco use and other factors associated with intervention effectiveness. Further qualitative research may also help us to understand Blue-collar workers' perceptions of risk and the possible interplay they see between occupational hazards and tobacco use. The next phase of worksite research will benefit from the application of an expanded range of research methodologies [Sorensen et al. 1998a; Emmons 2000]. Some have raised concerns that exclusive application of the randomized controlled design may restrict our ability to consider the complexity of social settings such as worksites [Susser 1995]. The required standardization of the intervention in the randomized controlled trial may limit the intervention's effectiveness by failing to tailor to the needs of the site and to provide a vehicle for incorporating worker input [Fisher 1995]. Through the diversification of research methods, including observational studies, qualitative research, and participatory action research, it may be possible to address a broader range of questions that will contribute to improved effectiveness of worksite tobacco control initiatives. Israel and colleagues [1985] and others [Eng and Blanchard 1990-91; Robertson and Minkler 1994] have advocated for the use of action research methods as a means of incorporating participants into the research process, thereby also potentially enhancing intervention effectiveness. It is also necessary that the timeframe of research projects correspond to that of worksites. At their essence, comprehensive programs on worker health are aiming to change the culture of worksites. Such changes cannot be successfully implemented within the short duration of most intervention trials.

## Conclusions

A comprehensive approach that integrates tobacco control initiatives into an overall worksite program for worker health holds considerable promise. Such programs may be of particular importance to blue-collar workers, whose high rates of tobacco use and exposures to hazards on the job place them at excess risk relative to other workers. Given the complexity required for effective interventions, it becomes increasingly important that we build bridges across disciplines interested in worker health.

Worksite tobacco control initiatives generally are offered within the context of health promotion programs, which function independently from occupational health and safety programs. Separate training programs for health educators and occupational health and safety professionals share little in terms of curricula and intervention methodology [Israel et al. 1996; Sorensen et al. 1995]. Health promotion providers seldom are trained to understand the hazards imposed by the worksite environment, and are given little background in the political sensitivities of working with management on a comprehensive approach to worker health. Similarly, occupational health and safety professionals seldom are trained in worker behavior, and are given very little preparation on how to be agents of change in the worksite. An expanded vision for worker health might be offered through joint training for health and safety specialists and health educators planning to base their careers in worksites. An example of one such program offered at the bachelors level is at the University of Wisconsin at Stevens Point. Coordinated systems need to be established within the worksite to promote interdisciplinary teams trained to understand the methods and philosophies of their collaborators. Collaboration across these two disciplines may generate more effective strategies for reaching blue-collar workers, for whom tobacco control efforts have been least effective.

Research to develop effective intervention strategies integrating tobacco control and occupational health and safety also requires an interdisciplinary approach. Multidisciplinary teams of researchers may include representation of occupational health and safety, behavioral and social sciences, organizational change, health promotion, labor education, and quantitative and qualitative methods [Israel et al. 1996]. Experts in these areas read different journals, attend different professional meetings, and employ different research methodologies. Indeed, these diverse backgrounds have contributed to differing ideological perspectives about responsibility for worker health. The belief that worker health begins with individual behavior change sets in motion a different set of intervention strategies from the supposition that management bears primary responsibility for worker health. These future collaborations need to be based on mutual trust, recognizing the history of competition for scarce resources. Overcoming the segmentation of these fields ultimately will require a common model of work and health, providing for resolution—or at least understanding—of our differences, assumptions, vocabulary, research methods, and intervention approaches.

Full implementation of these recommendations regarding intervention and research needed may also require changes in the ways that funders view tobacco control and occupational health. Categorical funding of research initiatives has furthered the segregation of these fields. A comprehensive view of worker health would be supported by systematic funding of interdisciplinary, collaborative research and training. Prior collaborations between the National Institute of Occupational Safety and Health and the National Cancer Institute provide an excellent example of a broadened funding agenda.

Few studies to date have examined the efficacy of a comprehensive approach to worker health, in which tobacco control is systematically integrated into a worksite-wide program addressing multiple levels of influence on worker health, leaving a range of research questions needing to be addressed. Further research can help to refine programs for delivery in a range of settings, from small worksites to multi-national corporations. Strategies to assure management commitment to comprehensive programs need to be developed and refined for application in a range of settings. These programs need to extend beyond the traditional model of the permanent employee in a stationary work setting to consider the work realities of mobile workers, such as construction laborers whose worksite may change from week to week, or transportation workers, whose worksite is consistently on the move; and contingent workers, such as contract and temporary workers. Effective tobacco control programs are also needed for workers in institutional settings, such as prisons or psychiatric hospitals, where smoking policies may apply differently to those who work and live in the setting. To be effective with a range of audiences, programs must take into account the assets and health strengths as well as health risks of workers of low socioeconomic status and from racial and ethnic minority groups.

The social ecological model also compels us to understand broader forces influencing tobacco control in the worksite [Stokols et al. 1996; McKinley 1993]. Important avenues for future research are introduced by the assumption that work settings are situated within a broad structure of community settings, including economic systems influencing corporate health, transportation systems affecting worker commutes, the health care system, and the regulatory environment.

It is imperative that our research address and understand the influence of new models for managing the delivery of medical and preventive services, which are altering the structure of many worksite health promotion programs. For many workers, trends toward worksite downsizing, the spread of technological innovations, and part-time employment are changing the structure of work, and need to shape the development and delivery of future worksite health promotion programs.

Productivity and organizational effectiveness are likely to be closely related to employee health and morale; efforts to understand the cost-benefits of worksite interventions need to incorporate these outcomes as interrelated indicators of program success. Understanding these external forces shaping internal worksite realities is likely to contribute to the effectiveness of comprehensive programs promoting worker health within healthy workplaces.

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