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Smoking Is an Occupational Hazard

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Background *Even though the prevalence of tobacco smoking has declined in the general population and among white-collar workers, the prevalence of tobacco smoking among blue-collar workers remains unacceptably high. Blue-collar workers experience greater exposure to workplace toxins which can add to, or even multiply, their risk of adverse health effects from tobacco smoking. Among blue-collar workers, workers in the restaurant, bar, and gaming industries are exposed to much higher levels of environmental tobacco smoke (ETS) than are office workers, and are at increased risk of cancer and cardiovascular diseases even if they are non-smokers themselves.*
Methods *The literature on health risks, and the disparity between white and blue collar workers in smoking prevalence, and the literature on various tobacco control strategies provide the sources on which this review is based.*
Conclusions *Over the past 20 years, the accumulating scientific evidence about smoking as an occupational hazard has prompted the implementation of various educational, economic, and legal tobacco control strategies.* Am. J. Ind. Med. 46:161–169, 2004. *Published 2004 Wiley-Liss, Inc.*†

KEY WORDS: environmental tobacco smoke; ETS; smoking ban; tobacco control; worksite health promotion; tobacco taxation; tobacco dependence treatments; smoking interactions with occupational toxins; smoking prevalence disparities

INTRODUCTION

Since the discovery of the plant *Nicotina tobacum* in the New World by Columbus over 500 years ago, tobacco use has become widespread throughout the world. Delivery of the active ingredient in tobacco leaves—nicotine—has become more efficient through time. From the use of snuff in the 16th and 17th centuries, to cigars in the 18th and 19th centuries, to cigarettes in the 20th and 21st centuries, the number of people exposed to tobacco and its combustion products has greatly increased. The invention of the manufactured cigarette in the early 20th century has the dubious honor of causing most of the worldwide increase in tobacco smoking [Mackay and Eriksen, 2002].

As the 21st century begins, the World Health Organization reports that one-third of the world's adult population uses tobacco despite evidence that smoking tobacco has significant adverse health effects on both the user and those environmentally exposed to combustion products in the smoke from the user's tobacco [Mackay and Eriksen, 2002]. Evidence that exposure to environmental tobacco smoke (ETS) poses a significant risk to workers, alone or in combination with exposure to other workplace toxins, has also been accumulating over the past 20 years. As a result of this scientific evidence, various tobacco control strategies, affecting both the general public and the working population, have become a focus of increasing attention and action throughout the United States.

TOBACCO SMOKING, HEALTH RISKS, AND OCCUPATIONAL DISPARITIES

Beginning with the first US Surgeon General's Report in 1964 [US DHEW (US Department of Health, Education, and Welfare), 1964], the adverse effects of tobacco smoke on the health of smokers have been well documented. These adverse effects include carcinogenic effects, such as lung, bladder, and oral cavity cancers [US DHHS (US Department of Health and Human Services), 1982] as well as non-

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carcinogenic effects, such as cardiovascular diseases [US DHHS (US Department of Health and Human Services), 1983] and chronic obstructive lung disease [US DHHS (US Department of Health and Human Services), 1984]. Tobacco smoking is, and has been for many years, the leading preventable cause of death in the United States [US DHHS (US Department of Health and Human Services), 2000]. The recent reversal of the century-long trend toward a female advantage in mortality may be due to increased tobacco smoking in women [Pampel, 2002]. And, while chronic disease and premature death from smoking have been problems that have affected developed countries for the past half-century, the problem will only increase, and by 2020 seven of every ten people who die as a result of tobacco smoking will be in developing countries [World Bank, 1999].

Even though the prevalence of tobacco smoking among adults in the United States decreased almost by half from 42.4% in 1965 to 24.1% in 1998, blue-collar workers are more likely to be current smokers than those identified as white-collar workers [CDC (Centers for Disease Control and Prevention), 2000]. This gap in smoking prevalence between blue-collar and white-collar workers is widening. In 1978, 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, service workers were 17% more likely to smoke cigarettes than white-collar workers; by 1997, they were 55% more likely to smoke [NCHS (National Center for Health Statistics), 2000]. Among some blue-collar workers in the construction trades, the prevalence of current smokers can be as high as 40% [Nelson et al., 1994]. Why these occupational disparities exist is uncertain, but a number of factors may be operative. They may be due to early smoking initiation—those who start smoking at a young age are more likely to be employed in blue-collar and service occupations [Giovino et al., 2002]. Stress may be a factor. Job-related stress has been associated with increased smoking intensity and decreased success at quitting [Green and Johnson, 1990]. Workplace culture may also play a role in that blue-collar workplaces may be more supportive of smoking and more tolerant of ETS exposure [Giovino et al., 2002].

ETS AND THE WORKING ENVIRONMENT

Aerosol Concentration and Biomonitoring

In the working environment, ETS gaseous and particulate concentrations can be significant. Studies in which short-term aerosol sampling has been performed in high risk ETS workplaces, such as restaurants and bars, indicate that respirable (<2.5 mm) particulate levels can average as high as 242 $\mu g/m^3$ [Repace and Lowery, 1980; First, 1984]. In comparison, non-polluted outdoor air has a concentration of respirable particulates ranging from approximately 10 to 50 $\mu g/m^3$.

Biomonitoring is a more accurate indicator of ETS exposure than is indirect measurement of ETS aerosol concentrations and the primary metabolites of nicotine, 3′-hydroxycotinine, and cotinine, are the candidate biomarkers for ETS exposure [Gorrod, 1993]. Urinary cotinine is the most commonly utilized biomarker in current ETS exposure studies [Benowitz, 1999]. These studies have shown that body fluids (urine, saliva, and serum) from non-smokers who are exposed to ETS can contain measurable concentrations of cotinine. In the home environment, non-smokers who live in the same home environment with tobacco smokers have measurable elevations of cotinine as compared to non-smokers who live with other non-smokers. The cotinine levels ranged from less than 1% to about 8% of the cotinine levels measured in homes with active smokers [Samet, 1987].

Similar findings have also been demonstrated in the working environment. Non-smoking restaurant workers exposed to significant ETS over an 8 hr shift had an average urinary cotinine level of 56 nanograms (ng) per milliliter (ml), compared to a non-smoking group not exposed to ETS who had a cotinine level of 8.3 ng per ml [Husgafvel-Pursiainen et al., 1987]. Based on these types of absorption studies, the National Research Council concluded in 1986 that non-smokers exposed to ETS over long periods of time can absorb the equivalent of 0.1 to 1.0 cigarette per 24 hr period [NAS/NRC (National Academy of Sciences/National Research Council), 1986].

Since 1986, the evidence of significant ETS absorption among non-smokers in the working environment, and the adverse health effects from that exposure, has continued to accumulate [Hammond, 1999]. Casino employees working in gaming areas where patron smoking is freely permitted were found to be exposed to ETS at levels greater than those who reported ETS exposure both at home and at work [Trout et al., 1998]. Food servers in non-smoking restaurants were found to be exposed to ETS at levels twice those found in an office workplace [Siegal, 1993; Johnsson et al., 2003]. Bar and tavern workers were found to have ETS exposure levels that reached levels four to six times higher than other workplaces [Jarvis et al., 1992].

Chronic Health Effects

As far back as 1928, a proposal was made that the cause of lung cancers in non-smoking women was from exposure to their spouse's tobacco smoke [Schnönherr, 1928]. By the 1980s and early 1990s, the US Environmental Protection Agency [US EPA (US Environmental Protection Agency), 1992], the US Surgeon General [US DHHS (US Department of Health and Human Services), 1986], the International Agency for Research on Cancer [O'Neill et al., 1987], and the

National Research Council of the National Academy of Sciences [NAS/NRC (National Academy of Sciences/ National Research Council), 1986] reviewed the health risks associated with ETS exposure and found that ETS exposure increases the risk of developing lung cancer and cardiovascular disease. Evidence of adverse health effects from ETS continues to accumulate for lung cancer [Hackshaw et al., 1997], cardiovascular disease [Law et al., 1997], and stroke [Hankey, 1999]. In fact, there is now evidence that ETS exposure in the working environment may pose a higher risk of lung cancer than ETS exposure in the home environment [Kreuzer et al., 2002].

Even though the weight of scientific evidence demonstrates that ETS is causally associated with lung cancer, cardiovascular disease, and chronic obstructive lung disease, a minority view does exist [Enstrom and Kabat, 2003], but it is not without some controversy [Smith, 2003].

In addition to causing lung cancer and ischemic heart disease in adults, ETS exposure has been implicated in causing sudden infant death syndrome, low-birth-weight infants, and new asthma cases in children [CA EPA (California Environmental Protection Agency), 1997]. However, only a limited literature exists on the role of ETS exposure in exacerbating adult asthma and further epidemiologic research needs to be done to further assess this important non-carcinogenic effect [Jaakkola and Samet, 1999].

Acute Health Effects

In addition to evidence of chronic adverse cardiovascular health effects, there is growing evidence that even short-term ETS exposure can compromise the endothelial function of the coronary arteries of non-smokers and lead to myocardial ischemia [Otsuka et al., 2001]. These acute effect findings may explain why ETS exposure increases the overall risk of cardiac mortality and morbidity in non-smokers exposed to ETS by 25% [He et al., 1999].

INTERACTION BETWEEN SMOKING, ETS, AND OCCUPATIONAL HAZARDS

For the majority of American workers who smoke, tobacco smoking represents a greater cause of mortality than exposure to occupational toxins. However, the presence of ETS exposure in the workplace does present an opportunity for adverse biological interactions to occur between tobacco smoke and occupational toxins that can augment the harm that tobacco smoke causes. Thus, the Surgeon General has recognized that efforts to achieve reductions in both smoking and exposure to hazardous workplace agents are more effective than either strategy alone in decreasing the adverse health risks associated with tobacco smoke in American workplaces [US DHHS (US Department of Health and Human Services), 1985].

Interactions between tobacco smoking and occupational exposures can occur in several different ways to produce both carcinogenic and non-carcinogenic health effects [Dement, 2002]. First, the same chemical constituents in tobacco smoke (e.g., carbon monoxide, aromatic amines, benzene, acetone, formaldehyde, and polycyclic aromatic compounds) can also be associated with work processes, thus increasing a workers' cumulative exposure to the particular chemical. Second, some workplace chemical toxins can produce an adverse health effect in the presence of tobacco smoking that neither can produce alone, for example, polytetrafluoroethylene, in the presence of tobacco smoking, can cause polymer fume fever [Wegman and Peters, 1974].

Third, the surface of a cigarette can become contaminated with an occupational agent such as lead, and in doing so, facilitate entry of the lead into the body via ingestion, inhalation, or cutaneous absorption. Fourth, tobacco smoke together with a workplace toxin can produce an additive adverse effect on a target organ, for example, chronic bronchitis in smoking workers exposed to both coal mine dust and silica. Fifth, tobacco smoke may interact in a multiplicative manner with occupational toxins, for example, the risk of lung cancer in cigarette-smoking asbestos workers is greater than the sum of the risk of the independent exposures to asbestos fibers and to the chemical constituents of tobacco smoke, and is approximated by multiplying the risks of the two separate exposures. Although similar types of additive or synergistic effects could be expected from ETS exposure in combination with other occupational hazards, there has been less research with respect to the biological interaction of ETS and occupational hazards than there has with active smoking.

In addition to the interactions between smoking and workplace chemical and physical agents, which can cause or exacerbate certain diseases, an association between the risk of traumatic occupational injury and tobacco smoking has also been noted. Possible explanations of this association may include: (1) direct toxicity of tobacco smoke, for example, performance decrements due to the physiological effects of carbon monoxide and nicotine; (2) distractibility due to preoccupation of the hands with cigarettes or eye irritation from tobacco smoke; (3) effect of medical conditions associated with smoking such as decrements in cardiovascular fitness; or (4) confounding factors such as the greater use of drugs or alcohol among workers who are tobacco smokers [Sacks and Nelson, 1994].

TOBACCO CONTROL STRATEGIES

The accumulation of research findings about the adverse health effects from ETS exposure in the working environment has had a significant effect on the national debate about the risks of tobacco smoking. The social debate is no longer about what harm the smoker it doing to his or her own health

and longevity, but now the debate is about what harm the smoker is doing to the non-smoker's health. From the days when tobacco smoke was merely a ''nuisance'' to the present day when tobacco smoke is a health ''threat,'' ETS exposure has spurred greater public health and occupational health efforts to (1) improve educational outreach about the risks of smoking and ETS; (2) provide more widespread treatment for tobacco dependence; (3) include increasing access to nicotine replacement products (NRTs); (4) increase the use of economic incentives to decrease user demand; and (5) enact statutes and promulgate regulations to restrict or ban ETS exposure in the working environment.

Information and Worksite Health Promotion

Information measures such as health warning labels on tobacco products, dissemination of research findings on the health consequences of smoking, mass media ''counter-advertising,'' and worksite health promotion programs have all been utilized as tobacco control strategies. Educating current or future smokers how to make better decisions about their own health is the goal of all tobacco health messages, but the addictive nature of nicotine poses a significant physiological obstacle to quitting and may impair fully rational decision-making, especially for long-term smokers [Rigotti, 2002].

The worksite has been a locus of smoking cessation education programs. However, the growing occupational disparity in smoking prevalence between blue and white-collar workers poses significant challenges for worksite programs aimed at increasing awareness of the health dangers of smoking and the health advantages of smoking cessation. Worksites with large number of blue-collar workers report a lower incidence of restrictive smoking policies [Holman et al., 1998] and have fewer health promotion programs available [Grosch et al., 1998]. Moreover, blue-collar workers are less likely to participate in such programs [Morris et al., 1999], and, when they do participate, they are less successful in quitting [Niknian et al., 1991].

Yet, blue-collar workers are the workers with the greatest need for educational interventions. They are the occupational group with the greatest exposure to occupational toxins, which can have an additive adverse health effect with tobacco smoke. Employer-sponsored health promotion programs may be seen by workers as subordinate to management's greater legal responsibilities to provide a safe workplace and to reduce exposures to regulated job-related risks. It is not surprising, then, to note that worksite smoking cessation programs seem to be successful only when they are a part of a comprehensive approach to worker health and safety [Sorenson et al., 1996]. Worksite smoking cessation educational interventions should fully integrate occupational safety and health protection, which is aimed at influencing management workplace hazard reduction decision-making, with health promotion efforts aimed at influencing individual health risk decision-making. On a larger scale, marrying health protection and health promotion programs will require that different professional communities form working collaborations to ensure workforce capability.

Tobacco Use and Dependence Treatments

Educational efforts should be coupled with the availability of medical treatment for tobacco dependence. Even though successful cessation may not be achieved on the first attempt since tobacco dependence is a chronic, relapsing condition, treatment should continued to be offered until sustainable abstinence is achieved, because cessation greatly reduces the risk of tobacco-related diseases, slows the progression of already-established tobacco-related conditions and increases life expectancy [US DHHS (US Department of Health and Human Services), 1990].

The two primary approaches to the treatment of tobacco use and dependence—counseling and pharmacotherapy—have been shown to be effective in achieving long-term abstinence [Fiore et al., 1996, 2000]. Counseling—whether group or individual—can be effective when provided by trained counselors and when it includes regular contacts over 4 weeks [Lancaster et al., 2000; Ringen et al., 2002]. Pharmacotherapies for treating tobacco dependence that have been approved by the US Food and Drug Administration (FDA) include one anti-depressant medication (sustained-release bupropion) and four nicotine-replacement products (gum, transdermal patch, nasal spray, and a vapor inhaler)—all of which have been shown to be effective, doubling the long-term rates of abstinence as compared to a placebo [Hughes et al., 1999].

Health insurance coverage for tobacco use and dependence services is uncommon [Schauffler, 1997]. Yet, greater access to nicotine replacement products and other cessation therapies has been shown to have a positive impact on smoking cessation for members of group health insurance plans [Curry et al., 1998]. Since tobacco dependence treatments have been consistently shown to be cost-effective relative to the medical costs associated with smoking-related diseases [Croghan et al., 1997], insurance reimbursement for tobacco dependence treatments (both counseling and pharmacotherapy) should be a covered benefit for all insurance plans to aid in smoking cessation [Fiore et al., 2000].

Price-Related Strategies

In the United States, tobacco smoking resulted in over $150 billion in annual health-related economic losses from 1995 through 1999, including annual productivity losses of $81.9 billion and medical expenses of $75.5 billion [CDC

(Centers for Disease Control and Prevention), 2002]. Some of these costs are borne by smokers themselves and others by their families, their employers, their health care insurers and state and local governments. Most of the public policy discussion about the social costs of tobacco has revolved around smokers without health insurance and the health care costs borne by states in providing smokers care.

In an era of tightening budgets, states have been vocal in complaining that uninsured smokers impose an unfair cost on their health care budgets. On the other hand, a minority view is that smoking actually has a net positive effect on government budgets because a ''benefit'' of tobacco smoking is a ''health care cost avoided'' from the premature death associated with smoking [Viscusi, 2002].

In 1990, a number of states sued cigarette manufacturers to recoup the uncompensated health care costs of tobacco smoking by trying to hold manufacturers liable for their ''wrongdoing.'' After additional states joined the lawsuit, the four largest cigarette manufacturers signed a financial Master Settlement Agreement with 46 states in 1998 to end the lawsuit [NAAG (National Association of Attorneys General), 2000]. To achieve settlement, the Agreement sidestepped the manufacturers' liability or ''wrongdoing'' issue, and instead set up a price-related system to compensate states for uninsured smoking-related health care costs. Economically speaking, the manufacturers in the Tobacco Settlement were successful in shifting the costs of the long-term, multibillion dollar settlement to smokers through increased cigarette prices.

Using price as a tobacco control strategy is not new; governments have been taxing tobacco since the 1700s. In fact, the father of modern economics, Adam Smith, believed that ''sugar, rum and tobacco are commodities which are nowhere necessaries of life, which are become objects of almost universal consumption, and which are, therefore, extremely proper subjects of taxation'' [Smith, 1776, reprinted 2003]. Of all the economic measures to reduce tobacco demand, the use of price has been shown to be the most effective as well as the most cost-effective [Chaloupka and Warner, 2000]. Higher cigarette taxes promote cessation among current adult smokers, and reduce consumption among adult smokers who continue to smoke, perhaps aiding these users in ultimate cessation. And, since young adult smokers are twice as sensitive to increases in price as adults, and children and adolescents are three times as sensitive, price can be an effective economic strategy to prevent smoking initiation [Fiore et al., 1996].

Historically, critics of tobacco taxation have made several arguments to oppose tobacco taxes. Among the arguments are: higher tobacco taxes will actually decrease government revenues because demand will plummet; tobacco taxes burden the poor disproportionately without providing them any compensating benefit; that taxes will lead to massive job losses in tobacco-associated industries; and that taxes only promote massive smuggling of tobacco products. However, these concerns may be unfounded.

Even though tobacco taxes do reduce tobacco consumption, the demand for cigarettes is relatively inelastic, i.e., cigarette consumption may fall when prices are increased, but consumption falls by a smaller proportion than prices rise [Chaloupka and Warner, 2000]. This may be because addicted consumers respond relatively slowly to price increases, if at all. Tobacco taxes, like other regressive consumption or sales taxes, place a disproportionately heavy financial burden on low-income families, which spend a larger share of their incomes on cigarette taxes than to wealthier households [World Bank, 1999]. However, poor consumers are more sensitive to price increases; their consumption is reduced to a greater extent, and the financial burden on poor consumers imposed by taxation is also reduced. When consumption is reduced, so does health, and out-of-pocket health care expenditures fall, thereby economically advantaging poor consumers [Chaloupka and Warner, 2000].

Another concern is that if demand for tobacco does fall, permanent job losses will result. In most developed and developing countries, money spent by smokers on tobacco would be spent on other goods and services if demand fell, resulting in no permanent job losses to the economy—merely displacement to another industry. However, for a few agrarian countries in sub-Saharan Africa, which are net producers of raw tobacco, sudden and complete cessation of demand could produce significant job losses, but the WHO considers this outcome to be unlikely [World Bank, 1999].

Finally, smuggling can frustrate the purpose of tobacco taxation by keeping consumption high at the same time reducing government revenues. However, tobacco sales that escape taxation—whether over the Internet or through state-by-state transactions—can be prevented by increasing interstate cooperation on tobacco product prices, prohibiting or taxing Internet tobacco sales, treating tobacco companies like hazardous waste producers by imposing strict liability on them for the downstream destiny of their product or by requiring the licensing of tobacco retailers [Leverett et al., 2002].

Legal Strategies

Since 1965, Congress has enacted tobacco-specific statutes, which address directly various aspects of tobacco control. These included requiring (1) health warnings on packaging and on print and outdoor advertising [15 USC (United States Code) Section 1333]; (2) prohibiting advertisements through any electronic communications source regulated by the Federal Trade Commission [15 USC (United States Code) Section 1335]; and (3) making receipt of federal grant monies contingent on prohibiting selling or distribution of tobacco to persons under 18 years of age [42 USC (United States Code) Section 300x-26]. However, Congress had

always stopped short of banning tobacco products whenever it legislated on tobacco.

Regulation of nicotine as a drug

Historically, the FDA had always denied that it had the statutory authority to regulate tobacco. In the early 1990s, the FDA began to consider whether nicotine was a drug consistent with the definition of drug found in FDA's enabling statute, the Food, Drug, and Cosmetic Act [21 United States Code, Section 301 et seq. (Food, Drug, and Cosmetic Act)] and whether a cigarette was drug-delivery device [Kessler et al., 1996]. In the Act, Congress had delegated to FDA the power to regulate the safety and efficacy of drugs that were introduced into interstate commerce.

After completing an extensive investigation, the FDA decided that nicotine was an addictive drug that posed significant threat to children, and that it had the authority to regulate tobacco products [Kessler, 2001]. FDA issued regulations, which were promptly challenged by the tobacco manufacturers [US DHHS (US Department of Health and Human Services), 1996]. The US Supreme Court decided that the Congress did not intend to grant the FDA the jurisdiction to regulate tobacco products [FDA (Food and Drug Administration), 2000]. The FDA's efforts to regulate nicotine as a drug ended. Now, only an act of Congress can confer on FDA the power to regulate tobacco as a drug [Myers, 2000].

Workplace smoking bans

Although various local governments had been successful in enacting restrictions on tobacco smoking in public places by the early 1990s, no state legislature had been successful in enacting a statewide workplace smoking ban. As the medical evidence accumulated that restaurant and bar workers were at higher risk of the adverse carcinogenic and cardiovascular health effects of ETS, the focus of the public policy debate on smoking shifted from the general public to worker safety and health protection as the basis for a possible ban on tobacco smoking in the working environment. The public health and occupational health communities joined together to advocate for smoke free workplaces and, as the reason for such workplaces, articulated a worker-based fairness rationale—why should workers have to choose between their job and their health?

In California, the first successful statewide tobacco smoking ban was finally enacted in 1994 after several unsuccessful attempts [California Assembly Bill 13 (AB 13), 1994]. As a result of this ground-breaking legislation in 1994, California law now provides that ''no employer shall knowingly or intentionally permit, and no person shall engage in, the smoking of tobacco products in an enclosed space at a place of employment'' [CA Lab. Code (State of California Labor Code), 1994]. In California, restaurants and all other workplaces except bars and taverns became smoke-free in 1994, and bars and taverns became smoke free in 1998. Enforcement of the statewide smoking ban on employers is two-tiered—through local law enforcement or public health departments for the initial three violations, and then through the California Division of Occupational Safety and Health for any subsequent violations.

Contrary to fears by restaurant and bar owners, evidence from taxable sales data indicate that patronage and sales actually increased after the smoking ban went into effect and that bar owners and employees now prefer to work in smoke-free environments [CDHS (California Department of Health Services), 2003]. This is undoubtedly due to the influx of non-smokers into restaurants and bars that had avoided such smoking establishments prior to the smoking ban. Tourism does not seem to have been adversely affected either [Glantz and Charlesworth, 1999]. Even though it will take years to assess improvements in health effects from the smoking ban, very initial symptom and function studies show that the smoking ban has had positive effects on the respiratory health of affected workers [Eisner et al., 1998].

At this writing, four other states—Delaware, New York, Connecticut, and Maine—have enacted statewide workplace tobacco smoking bans like California's. Florida has a ban on indoor smoking, but it does not apply to stand-alone bars, i.e., bars that serve only customary bar snacks and generate no more than 10% of gross revenue from food sales [Florida Department of State, Division of Elections, 2003]. In the future, it is expected that other states will consider adoption of California-style statewide smoking bans based on a concern for the health and safety of their workers.

CONCLUSIONS

Smoking is an occupational hazard, both for the worker who smokes and for the non-smoker who is exposed to ETS in his or her workplace. Additional research is needed to better characterize smoking prevalence among blue-collar workers and what factors can influence cessation among these workers, to improve the understanding of interactions between tobacco smoke and occupational injuries, and to evaluate the impact of state tobacco smoking bans on worker health and safety. Even though more research is needed to further refine our understanding about the health effects of ETS, this need should not stop further efforts to eliminate ETS from the workplace, to make tobacco dependence treatment more available and reimbursable by health care insurance providers, and to integrate health promotion into state and federal worker safety and health protection programs. Smoking as an occupational hazard should be completely eliminated for the sake of the health and safety of American workforce.

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