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MAKING YOUR WORKPLACE SMOKEFREE

A DECISION MAKER'S GUIDE



M A K I N G Y O U R W O R K P L A C E S M O K E F R E E

A D E C I S I O N M A K E R ' S G U I D E

**U.S. Department of Health and Human Services
Centers for Disease Control and Prevention
Office on Smoking and Health**

**Wellness Councils of America
American Cancer Society**

PREFACE

As scientific evidence documenting the health hazards posed by environmental tobacco smoke (ETS) continues to mount, workplace decision makers have more reason than ever to protect employees from exposure to ETS on the job. The U.S. Environmental Protection Agency concluded in January 1993 that each year ETS kills an estimated 3,000 adult nonsmokers from lung cancer and that the workplace is a significant source of ETS. In a recent study, nonsmoking employees exposed to ETS at work but not at home had significantly higher levels of a nicotine metabolite in their blood than did nonsmoking workers with no work or home exposure to ETS. Levels of exposure to ETS are lowest in smokefree workplaces.

Even before these recent studies were available, the U.S. Surgeon General had determined in 1986 that ETS is a cause of disease, including lung cancer, in otherwise healthy nonsmokers. The Surgeon General also reported that the simple separation of smokers and nonsmokers within the same airspace may reduce, but does not eliminate, the exposure of nonsmokers to ETS. In 1991, the National Institute of Occupational Safety and Health of the Centers for Disease Control and Prevention recommended that “all available preventive measures should be used to minimize occupational exposure to ETS.”

Health issues provide ample justification for restricting ETS exposure at the worksite. But there are other good business reasons. Instituting smokefree work environments can reduce costs for cleaning and maintaining facilities and equipment and improve employee morale. “Smokefree” does not mean “anti-smoker.” Policies that restrict or eliminate smoking may provide incentives for employees to stop smoking altogether. You can demonstrate your commitment to employees who smoke by offering to help interested smokers quit.

In brief, that's the “why” for companies and organizations to go smokefree. This handbook, *Making Your Workplace Smokefree: A Decision Maker's Guide*, provides the “how.” This up-to-date guide draws on years of research and first-hand experience, and it offers practical and proven strategies for implementing successful policies in a variety of work settings. As you will see in this guide, with a little planning an effective workplace policy is easy to implement. We hope that workplace decision makers will use this handbook as a blueprint for action in promoting the health and safety of all workers.

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Government agencies, health voluntary organizations, researchers, and others have produced guides on managing issues of ETS in the workplace. This guide relied on the following as basic resources:

Dana-Farber Cancer Institute, American Cancer Society, and Liberty Mutual Insurance Company, with support from the Massachusetts Department of Public Health. *Guide to Workplace Tobacco Control*. 1993.

Office of Disease Prevention and Health Promotion and Office on Smoking and Health, American Cancer Society, American Heart Association, and American Lung Association. *No Smoking: A Decision Maker's Guide to Reducing Smoking at the Worksite*. 1985.

TABLE OF CONTENTS

PREFACE	i
ACKNOWLEDGMENTS	ii
ABOUT THIS GUIDE	vi
CHAPTER ONE. COSTS AND OTHER CONSEQUENCES OF TOBACCO	1
Adverse Effects of Combining Cigarette Smoking and Other Workplace Risks	2
Health Consequences of ETS	3
Costs to Employers	4
Liability	5
Costs to Individuals and to Society	6
CHAPTER TWO. BENEFITS OF A SMOKEFREE WORKPLACE	11
Support for Workplace Policies	12
Employee Morale	12
Productivity and Medical Costs	13
Corporate Image	13
CHAPTER THREE. A DECISION MAKER'S GUIDE TO CHOOSING A WORKPLACE POLICY	17
It's Your Decision	18
Model Smokefree Policy and Procedure	19
CHAPTER FOUR. SUPPORT FOR EMPLOYEES WHO SMOKE	23
The Goal: A Smokefree Workplace, Not Stigmatizing Employees Who Smoke	24
Nicotine Addiction	24
Immediate Benefits of Quitting Smoking	25
Smoking Cessation and the Workplace	25
Types of Support for Employees Who Smoke	26
Match Support to Employee Needs	26
Cessation Support Options	28
Deciding What Types of Support to Offer	31

CHAPTER FIVE. STEP-BY-STEP: YOUR DECISIONS AND HOW TO MAKE THEM WORK	33
Assess the Current Situation	34
Decide on a New ETS Policy and Develop a Plan to Implement It	37
Communicate with Employees and Management	38
Announce and Manage the Policy	39
APPENDICES	43
Appendix A. Resources	44
Appendix B. Sample Policies	46
Appendix C. Organizations with Smokefree Workplaces	49

ABOUT THIS GUIDE

GOAL: *Provide a 100% smokefree workplace environment.*

- *Protect all employees from exposure to environmental tobacco smoke.*
- *Provide cessation support to smokers who want to quit smoking.*

This guide provides you, the worksite decision maker, with information on how to design, implement, and evaluate environmental tobacco smoke (ETS) policies and related activities. Ideally, to protect all persons from exposure to ETS, companies will implement policies that require a smokefree environment in company buildings and other enclosed places and provide significant smoking cessation support for their employees and covered dependents.

Chapter One provides the background information you need to make the decision to implement policies and related activities to eliminate ETS in the workplace.

Chapter Two highlights legal, scientific, human resource, facility, image, and economic reasons to reduce ETS in the workplace.

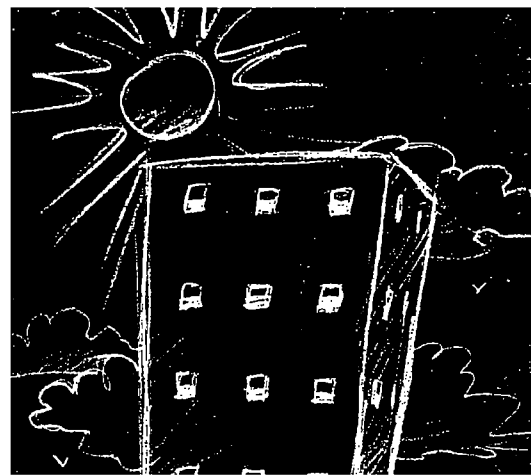
Chapter Three provides a model smoking policy and additional options to help companies and other organizations design policies to fit their needs.

Chapter Four helps the company develop smoking cessation activities to ensure help is available for employees who want to quit smoking.

Chapter Five provides step-by-step instructions in designing policies and related programs to meet the needs of the company.

Additional resources, sample policies, and a list of organizations with smokefree workplaces are included in the Appendices.

C h a p t e r **1**



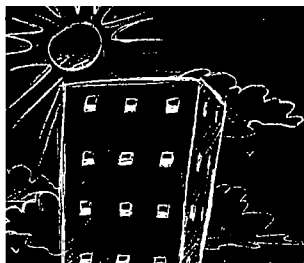
**C O S T S
A N D O T H E R
C O N S E Q U E N C E S
O F T O B A C C O**

Tobacco causes more deaths than AIDS, alcohol abuse, automobile accidents, illegal drugs, fires, homicide, and suicide combined.¹

Examples of hazardous materials that when combined with smoking present a serious health risk⁵:

- Coal
- Grain
- Silica
- Welding materials
- Asbestos
- Petrochemicals
- Aromatic amines
- Pesticides
- Cotton dust
- Ionizing radiation

Asbestos workers who smoke have ten times the risk of developing lung cancer as asbestos workers who do not smoke.⁵



Cigarette smoking is the leading preventable cause of death in this country: it is responsible for one in every five American deaths.² Smoking claims the lives of an estimated 1,100 people each day—over 400,000 smokers die from smoking-related diseases and 3,000 nonsmokers die from lung cancer each year.^{2,3} In addition, nonsmokers exposed to environmental tobacco smoke (ETS) have higher death rates from cardiovascular disease than nonsmokers who are not exposed to ETS.⁴

Since the first Surgeon General's report on smoking and health in 1964, tobacco use has been increasingly linked to disease, disability, and premature death. That is, tobacco users die sooner than people who don't use tobacco. Figure 1-1 lists known effects of tobacco use and ETS.

ADVERSE EFFECTS OF COMBINING CIGARETTE SMOKING AND OTHER WORKPLACE RISKS

According to the 1985 Surgeon General's report on *The Health Consequences of Smoking: Cancer and Chronic Lung Disease in the Workplace*, the combination of smoking with exposure to hazardous substances at the workplace presents a serious health risk.⁵ As explained in the 1979 Surgeon General's report on smoking and health, cigarette smoking can

- transform existing chemicals into more harmful ones,
- increase exposure to existing toxic chemicals,
- add to the biological effects caused by certain chemicals, and
- interact synergistically with existing chemicals.⁶

For example, the health effects of smoking and workplace exposure to asbestos are greater than the sum of the risks of separate exposures. For most workers who smoke, however, cigarette smoking is a greater cause of death and disability than any hazard in the workplace.⁵

HEALTH

CONSEQUENCES OF ETS

ETS is a proven health hazard. For example, the 1986 Surgeon General's report on involuntary smoking concluded that exposure to ETS can cause lung cancer: nonsmoking spouses have nearly a doubled risk of developing lung cancer if their spouses are heavy smokers.⁷ Research reviewed in the reports of the Surgeon General, the National Academy of Sciences,⁸ and the National Institute of Occupational Safety and Health (NIOSH)⁹ found that secondhand tobacco smoke was harmful, and the U.S. Environmental Protection Agency (EPA)³ estimated that ETS causes 3,000 lung cancer deaths each year in the United States. In addition, scientific studies published in peer-reviewed journals on both animals and human subjects indicate that nonsmokers exposed to secondhand tobacco smoke have higher death rates from heart disease.^{4,10-12} Figure 1-2 highlights statements from important reports on ETS.

ETS has been classified as a Group A (known human) carcinogen, as have asbestos and benzene.³ Nonsmokers subjected to ETS are exposed to nicotine, carbon monoxide, and cancer-causing agents. A recent study found that nonsmokers exposed to ETS only at work had significantly higher levels of a

FIGURE 1-1. HEALTH CONSEQUENCES OF TOBACCO USE AND ETS

Health Consequences of Tobacco Use¹⁴⁻¹⁷

Mortality and Morbidity

- Results in premature death
- Causes significant disease and disability

Cardiovascular Effects

- A cause of coronary heart disease
- A cause of cerebrovascular disease (stroke)
- A cause of atherosclerotic peripheral vascular disease

Cancer

- A cause of lung cancer
- A contributing factor for pancreatic cancer
- A cause of laryngeal cancer
- A contributing factor for renal cancer
- A cause of cancer of the oral cavity (lip, tongue, mouth, and pharynx); smokeless tobacco is also a cause of oral cancer
- Associated with gastric cancer
- A cause of esophageal cancer
- A cause of bladder cancer

Lung Diseases

- A cause of chronic bronchitis
- A cause of emphysema

Women's Health Effects

- A cause of intrauterine growth retardation, leading to low birth weight babies
- A contributing factor for cervical cancer
- A probable cause of unsuccessful pregnancies

Other Health Effects

- Addiction to nicotine
- Adverse interactions with occupational hazards that increase the risk of cancer
- Alteration of the actions and effects of prescription and nonprescription medications
- A probable cause of peptic ulcer disease

Health Consequences of ETS^{3, 4, 7, 10-12, 18-20}

- A cause of lung cancer in nonsmokers
- Associated with higher death rates from cardiovascular disease in nonsmokers
- In children, associated with respiratory tract infections, increased prevalence of fluid in the middle ear, additional episodes of asthma, and increased severity of symptoms in children with asthma, and a risk factor for new onset of asthma in children who have not previously displayed symptoms
- Associated with increased risk of sudden infant death syndrome (SIDS)
- Associated with increased irritant effects, particularly eye irritation, among allergic persons

FIGURE 1-2. STATEMENTS FROM REPORTS ON ETS

In 1986, the Surgeon General made these conclusions⁷:

- Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers.
- Simple separation of smokers and nonsmokers within the same airspace may reduce but does not eliminate exposure of nonsmokers to ETS.

In 1991, NIOSH strengthened the findings on ETS in the workplace⁹:

- ETS is a potential occupational carcinogen.
- ETS poses an increased risk of lung cancer and possibly heart disease to occupationally exposed workers.
- Exposure to ETS should be reduced to the lowest feasible level.
- Employers should minimize occupational exposure to ETS by using all available preventive measures.

In 1993, the EPA released a report on the respiratory health effects associated with passive smoking with this conclusion³:

- ETS is a human lung carcinogen responsible for 3,000 lung cancer deaths annually in U.S. nonsmokers.

Evidence that ETS is a risk factor for cardiovascular disease and other diseases continues to accumulate.^{3,4,10-12,18-20}

ETS is a combination of smoke exhaled by the smoker and the smoke that comes from the burning end of a cigarette, cigar, or pipe.

nicotine metabolite in their blood than nonsmokers reporting no workplace exposure.¹³

The EPA concluded that children with asthma have their condition worsened by exposure to ETS.³ Yet children often spend considerable amounts of time in the worksites of adults (e.g., schools, restaurants).

More people die from ETS than all other regulated occupational substances combined.³ Even one of these deaths due to ETS is unnecessary.

COSTS TO EMPLOYERS

The costs of employee smoking to the employer are significant. Direct costs to the employer include health care costs associated with smoking. Indirect costs include lost productivity, absenteeism, and recruitment and retraining costs resulting from death and disability related to smoking. Following is a list of some of the factors that contribute to smokers costing employers more than nonsmokers.²¹

- Absenteeism
- Health insurance and life insurance costs and claims
- Worker's compensation payments and occupational health awards
- Accidents and fires (plus related insurance costs)
- Property damage (plus related insurance costs)
- Smoke pollution (increased cleaning and maintenance costs)
- Illness and discomfort among nonsmokers exposed to passive smoke

The cost to employers of employees who smoke is not a simple number; many factors and variables need to be consid-

ered. However, the most frequently cited estimate for the excess cost (adjusted to 1991 dollars) is \$1,300 per year per smoking employee.²¹

Other costs arise from ETS. Historically, smoking restrictions were implemented to prevent fires and explosions in the workplace. These rules were established to protect products, machinery, and furniture rather than to protect the health of employees. Today, employers who implement smokefree policies for their offices can save money for the same reason because computer equipment, furniture, and carpets last longer in a smoke-free environment and require less maintenance.²²

Regardless of the size of your company, implementing smokefree policies and providing support to help employees and covered dependents to quit smoking makes good business sense.

LIABILITY

As early as 1972, the Surgeon General warned that cigarette smoking was dangerous to nonsmokers.²³ Since then, court rulings and state, county, and city statutes and regulations have provided protection for the nonsmoker. In March 1994, the Occupational Safety and Health Administration of the U.S. Department of Labor issued proposed national regulations to govern indoor

air quality, including ETS. In workplaces where smoking is not prohibited, the proposed rule calls for designated smoking areas that are enclosed and exhausted directly to the outside. The regulations have not been finalized as of September 1996. Many states and municipalities have enacted legislation restricting smoking in public, and some have moved to restrict smoking at the worksite. As of June 1995, 47 states had restricted smoking at some level in workplaces or public places, and 21 states regulated smoking in private worksites.²⁴ More than 800 local ordinances exist that impose restrictions on tobacco use.²⁵

Because no one has the right to impose a health risk on others and because an employer has a common-law responsibility to provide a safe and healthful workplace,²⁶ liability is a significant issue for employers. Because ETS has been classified as a Group A (known human) carcinogen, it would be difficult to argue that an employer who has not reduced ETS to the lowest possible levels has provided a safe workplace.

Recent examples show the validity of liability concerns. In December of 1995, a widower of a Veterans Affairs hospital nurse was awarded death benefits on the grounds that his wife's fatal lung cancer was caused by exposure to secondhand smoke while treating

ETS contains about 4,000 chemical compounds, including formaldehyde, cyanide, carbon monoxide, ammonia, nicotine, and cancer-causing agents such as benzene and N-nitrosamines.³

FIGURE 1-3. ONE-YEAR COSTS OF SMOKING-ATTRIBUTABLE MEDICAL CARE (1993)³¹

Type of Cost	Amount (\$ billion)
Hospital expenditures	26.9
Physician expenditures	15.5
Nursing home expenditures	4.9
Prescription drugs	1.8
Home health care expenditures	0.9
Total	50.0

Eliminating ETS in the workplace and decreasing smoking by employees can reduce health care costs and increase years of productive life. These two factors alone will positively affect the bottom line for companies. While premature death among smokers may offset the increased health care costs with savings in pension benefits, all responsible employers want their employees to live full and productive lives, before and during retirement.

patients.²⁷ In January 1996, a Florida state appeals court ruled that airline flight attendants could proceed with a national class-action lawsuit against cigarette manufacturers for their responsibility in causing health problems related to exposure to secondhand smoke.²⁸

COSTS TO INDIVIDUALS AND TO SOCIETY

The cost savings associated with a heart attack that is prevented or with the delayed onset of cancer are often difficult to calculate. Yet, reducing the prevalence of smoking behavior in a worksite can save money, not only for the employer but also for individuals and society as a whole. Individual costs in time, health, and money arise because smokers tend to

- have more hospital admissions,
- take longer to recover from illness and injury,

- have higher outpatient health care costs,^{29,30} and
- have lower birth weight babies.¹⁶

A 1994 report from the Centers for Disease Control and Prevention (CDC) estimated that the cost of smoking for direct medical care was \$50 billion for 1993 (Figure 1-3).³¹ Indirect costs of smoking to society, such as lost productivity from increased absenteeism and productive years of lives lost, also are enormous. For 1990, the Congressional Office of Technology Assessment estimated that indirect costs from smoking-attributable illness and death totaled \$47.2 billion.³²

ETS is dangerous to employees and costly for employers. To protect all employees from the health hazards associated with ETS exposure, companies should restrict smoking in the workplace. In addition, companies should offer assistance to employees and dependents interested in stopping smoking.

This chapter has described the consequences of tobacco use and ETS exposure. Chapter Two discusses the benefits of a smokefree workplace.

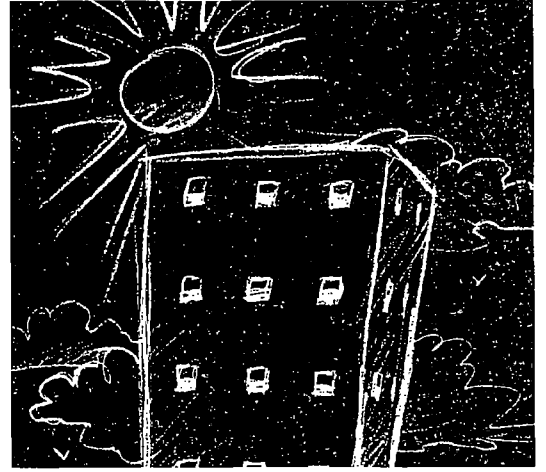
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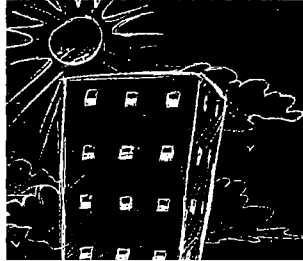
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C h a p t e r **2**



B E N E F I T S O F A S M O K E F R E E W O R K P L A C E

*The primary benefit of a smokefree environment
is the protection of all employees from
the health risks of ETS.*



A ccording to the National Cancer Institute, most employees are nonsmokers, almost half of employees currently work in environments where smoking is prohibited in their work area or in public (shared) areas of their work site, and more than 80% work for companies that have an ETS policy.¹ Clearly, employers are addressing how to deal with smoking in the workplace. Figure 2-1 lists some of the many benefits for both employees and employers that accrue from a smokefree workplace.

SUPPORT FOR WORKPLACE POLICIES

Comprehensive smoking policies, including facility-wide smokefree policies, are not new. However, smokefree policies have received a significant boost over the last 10 years as evidence of the risks associated with exposure to ETS has mounted and the public has become

more aware of these risks. According to Gallup surveys, Americans know about the risks posed by ETS and favor efforts to reduce exposure to it. In 1992, 97% of nonsmokers and 79% of current smokers agreed that exposure to ETS is harmful to healthy adults.³ The percentage of Americans who favor some type of restriction on workplace smoking increased from 81% in 1983 to 94% in 1992.³

Employers have responded by implementing smokefree policies. A 1992 survey showed that 59% of private employers had either smokefree facility policies or permitted smoking only in separately ventilated smoking areas.⁴ A smaller 1994 survey of employers with up to 25,000 employees found that 54% of companies had implemented smokefree policies and that only 7% had no smoking policy at all.⁵

EMPLOYEE MORALE

An employer sends a clear message to employees and the community with a smokefree policy: We care about the health and safety of our employees. Scientific evidence proving that significant health risks are associated with ETS exposure supports employers against any claims of “harassing” employees or visitors who smoke. The employer’s concern for the health of employees is especially clear in the case of employees who have conditions that make them vulner-

able to ETS, including employees who are pregnant or who have heart disease or allergies to tobacco smoke.

Support for employees who smoke goes hand in hand with policies that restrict smoking in the workplace. Helping employees who want to quit sends a straightforward message—the company cares about all employees, including smokers. More information about the kinds of support you may want to provide smokers is in Chapter Four.

PRODUCTIVITY AND MEDICAL COSTS

A smokefree workplace can enhance productivity in two ways: by reducing the effects of ETS on nonsmokers and by reducing excess smoking-related absenteeism among smokers who are motivated to quit as a result of the smokefree policy. For small businesses especially, which have employees who handle a variety of tasks, productivity can be greatly increased by reduced absenteeism.

A smoker who quits smoking could save employers an estimated \$960 in excess illness costs each year.⁶ Persons who quit smoking before age 65 were estimated to save from 40% to 67% of the lifetime excess medical costs of persons who continue to smoke.⁷

FIGURE 2-1. BENEFITS OF A SMOKEFREE WORKPLACE²

For the employees

- A smokefree environment helps create a safe, healthful workplace.
- A well planned and carefully implemented effort by the employer to address the effects of smoking on employees' health and the health of their families shows the company cares.
- Workers who are bothered by smoke will not be exposed to it at the worksite.
- Smokers appreciate a clear company policy about smoking at work.
- Managers are relieved when a process for dealing with smoking in the workplace is clearly defined.

For the employer

- A smokefree environment helps create a safe, healthful workplace.
- Direct health care costs to the company may be reduced.
- Maintenance costs go down when smoke, matches, and cigarette butts are eliminated in facilities.
- Office equipment, carpets, and furniture last longer.
- It may be possible to negotiate lower health, life, and disability coverage as employee smoking is reduced.
- The risk of fires is lower.

CORPORATE IMAGE

Corporate image is important for many businesses. With nonsmokers accounting for about 75% of adult American consumers of goods and services, it is easy to see why many companies and organizations implement smokefree sites to influence consumers' opinions of the company. For example, to demonstrate

its commitment to providing a pleasant and safe dining environment, in 1994 McDonald's implemented smokefree environments in its 1,400 corporate-owned restaurants. Another service industry, Delta Airlines, banned smoking on all flights beginning January 1, 1995. See Appendix C for examples of organizations that have gone smokefree.

Such actions can improve a corporate image not only in the marketplace but also in hiring. Companies that demonstrate concern for the health and well-being of their workforce are more likely to be able to recruit and retain high-quality employees.

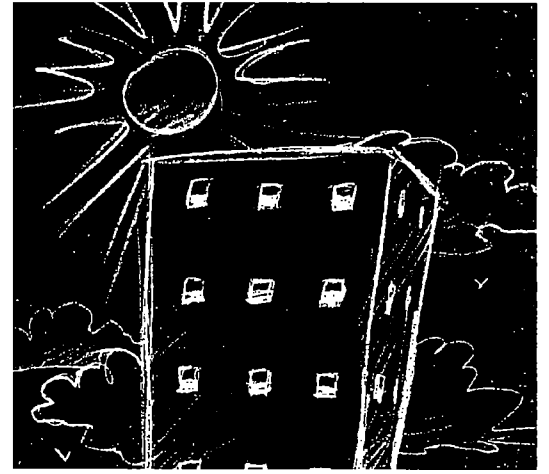
Because so many worksites are already smokefree, employers who have not instituted smokefree policies need to consider complying with community standards and expectations.

This chapter has described the benefits of developing and implementing comprehensive smoking policies—especially a smokefree policy. Chapter Three provides information to help you determine the best policy for your worksite.

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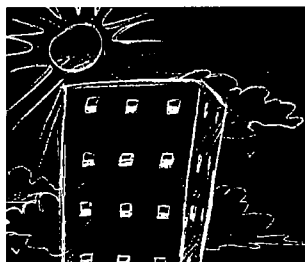
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Chapter 3



A DECISION MAKER'S GUIDE TO CHOOSING A WORKPLACE POLICY

The Surgeon General has reported that the simple separation of smokers and nonsmokers within the same airspace may reduce, but does not eliminate, the exposure of nonsmokers to ETS.¹



While many possible smoking policies exist, only two are viable in today's social and scientific environment:

(1) Smokefree

Smokefree environment in company facilities and vehicles; can be extended to include the property or grounds of the employer.

(2) Separately ventilated areas

Smoking limited to separately ventilated smoking rooms.

Table 3-1 compares the two types of smoking policies. When deciding which to select, consider any relevant laws and ordinances as well as the consequences of the policies. For example, health care costs are not likely to be affected by policies that allow employees to smoke in separately ventilated areas. A 1995 study found that separately ventilated smoking rooms might increase the lung

cancer mortality of smokers whereas a smokefree policy could decrease lung cancer mortality among smokers.²

Cost savings may not result unless the company implements a comprehensive ETS policy and provides cessation activities that result in a reduction in smoking among employees and covered dependents. You need to decide what benefits are most important for your company, keeping in mind that protecting the health of both smokers and nonsmokers requires a smokefree workplace.

According to one study, in workplaces where smoking is allowed, 59% of nonsmokers reported that exposure to ETS caused them discomfort.³ A recent study demonstrated the clear relationship between the level of a smokefree policy and the actual exposure of nonsmokers to hazardous components of ETS: the more comprehensive the policy, the lower the exposure.⁴

IT'S YOUR DECISION

Some questions to consider in making your decision include the following:

- Given the employees' interests, health, and work environment, what policy will provide them the most protection?

TABLE 3-1. SMOKING POLICIES: 100% SMOKEFREE AND SEPARATELY VENTILATED AREAS

Policy	Pros	Cons
Smokefree <i>Smoking is not allowed inside any building or company vehicle. Smoking occurs only at designated outdoor smoking locations. Policy can be extended to prohibit smoking on company grounds. Then, employees who smoke will need to refrain from smoking throughout the workday or leave company grounds to smoke.</i>	• Complies with all laws and ordinances • Greatly reduces ETS exposure for all employees • Provides best health and safety benefits for employees • May reduce the number of cigarettes smoked by employees; may encourage employees to quit smoking • Decreases maintenance costs • Sends a clear message to employees • Easier to administer and enforce • Low cost to implement	• Requires smokers to modify their behavior • Some costs may be incurred if outside smoking shelters are constructed • Employee smoking directly outside building impacts image • Inconvenience to employees who smoke • If not properly managed, smokers may be disproportionately absent from their work stations
Separately Ventilated Areas <i>Smoking is allowed only in dedicated smoking rooms. The rooms have separate ventilation systems designed to prevent ETS from leaking into other areas of the building.</i>	• Complies with most laws and ordinances • Reduces nonsmokers' exposure to ETS • Allows smokers to stay indoors	• May have adverse effects on smokers' health • Building and maintaining separately ventilated lounges is expensive • Requires space • Ventilation systems may not adequately protect nonsmokers from ETS exposure

- What policy will offer the greatest benefits to the company at the lowest cost?
- What policy will management find most supportable?
- What community ordinances exist governing smoking in public places or workplaces?
- What are the customers' or the community's expectations regarding ETS, given the policies in other similar workplaces and public sentiments regarding ETS?

There are a number of ways to gather information to answer these questions and to support your decision about the most appropriate smoking policy.

Chapter Five offers a step-by-step guide to gathering information and determining what policy option to choose.

MODEL SMOKEFREE POLICY AND PROCEDURE

The model policy in Table 3-2 is a starting point for companies that do not have a smoking policy. It can be modified to suit your needs. Note that you

TABLE 3-2. MODEL SMOKEFREE POLICY**Policy**

Due to the acknowledged hazards arising from exposure to environmental tobacco smoke, it shall be the policy of _____ to provide a smokefree environment for all employees and visitors. This policy covers the smoking of any tobacco product and the use of smokeless or "spit" tobacco and applies to both employees and nonemployee visitors of _____.

Definition

1. There will be no smoking of tobacco products within the facilities at any time.

The decision to provide or not provide designated smoking areas outside the building will be at the discretion of management or other decision-making body.

The designated smoking area will be located at least 20 feet from the main entrance.

All materials used for smoking, including cigarette butts and matches, will be extinguished and disposed of in appropriate containers. Supervisors will ensure periodic cleanup of the designated smoking area. If the designated smoking area is not properly maintained (for example, if cigarette butts are found on the ground), it can be eliminated at the discretion of management or other decision-making body.

[For a policy that extends smokefree to include company property, substitute the following: There will be no smoking of tobacco products within the facilities or on the property of _____ at any time.]

2. There will be no smoking in any _____ vehicle.

There will be no smoking in _____ vehicles at any time.

There will be no tobacco use in personal vehicles when transporting persons on _____-authorized business.

3. Breaks

Supervisors will discuss the issue of smoking breaks with their staff. Together they will develop effective solutions that do not interfere with the productivity of the staff.

Procedure

1. Employees will be informed of this policy through signs posted in _____ facilities and vehicles, the policy manual, and orientation and training provided by their supervisors.
2. Visitors will be informed of this policy through signs, and it will be explained by their host.
3. The _____ will assist employees who wish to quit smoking by facilitating access to recommended smoking cessation programs and materials.
4. Any violations of this policy will be handled through the standard disciplinary procedure.

can address issues regarding smokeless or “spit” tobacco in the same policy.

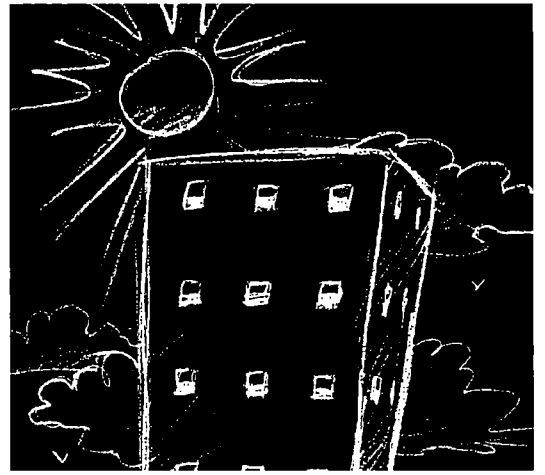
Although ETS is not an issue with smokeless tobacco, aesthetic and health issues do pertain to smokeless tobacco, and it may make sense to include all tobacco products in a single policy.

Deciding which policy to implement is only half of the decision; the other half is deciding what type of support to offer the smokers who will be affected by the change. The next chapter will provide you with useful information to choose support for the employees who smoke.

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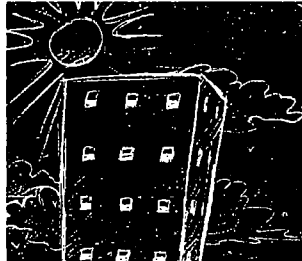
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C h a p t e r **4**



S U P P O R T
F O R
E M P L O Y E E S
W H O
S M O K E

Of current smokers, an estimated 32 million smokers (about 70% of all smokers) report they want to quit smoking completely.¹



What We Know About Nicotine¹:

1. Cigarettes and other forms of tobacco are addicting.
2. Nicotine is the drug in tobacco that causes addiction.
3. The pharmacologic and behavioral processes that determine tobacco addiction are similar to those that determine addiction to drugs such as heroin and cocaine.

The effect of implementing a smokefree policy will be most immediate for employees who smoke. You can help them adjust to changes introduced by your smoking policy.

- Inform employees in advance that a new policy is being developed.
- After the policy is implemented, let smokers know that you appreciate their efforts to comply with the policy.
- Offer smoking cessation assistance.
- Ask nonsmoking employees to support and encourage smokers.
- Plan for continuing support of smokers who want to quit.

Providing cessation support is important because a new policy may encourage smokers to try to quit smoking. At a

minimum, smoking policies tend to result in a reduction in the number of cigarettes smoked.²

THE GOAL: A SMOKEFREE WORKPLACE, NOT STIGMATIZING EMPLOYEES WHO SMOKE

The goal of a smokefree policy is to provide a safe and healthful workplace for all employees. Unfortunately, the simple message of “smokefree” can sometimes be misinterpreted to mean “smoker-free” or “anti-smoker.” Communication is important to allay any misconceptions. Also, a company can demonstrate its commitment to employees who smoke by offering help: Cessation support may help employees quit successfully and reassures smokers that the company is not trying to stigmatize them.

NICOTINE ADDICTION

With over three decades of research documenting evidence that quitting smoking can increase life expectancy through avoiding disease, why don't smokers quit? Usually, because they are addicted to nicotine.³ When nicotine is withheld, withdrawal symptoms can occur in less than 24 hours. Craving for nicotine, anxiety, frustration, irritability, anger, loss of concentration, increased heart rate, fatigue, light-headedness, and

tightness in the chest have all been related to nicotine withdrawal. Although many symptoms disappear within one to three weeks, the craving and physical urge to smoke can remain for months and even years.⁴

IMMEDIATE BENEFITS OF QUITTING SMOKING

Despite the physical discomforts that may accompany cessation, benefits accrue almost immediately. These benefits include the following⁵:

- Carbon monoxide level in blood declines within eight hours.
- Sense of taste and smell quickly improves.
- Oral health improves (cessation helps to prevent or stabilize dental disease and eliminates “smoker’s breath”).
- Stamina and vigor improve as a result of increased oxygen in the system and improved circulation.
- Enhanced self-image is brought on by a sense of accomplishment.
- Acute effects of nicotine on pulse rate, blood pressure, and body temperature are eliminated within 20 minutes of the last cigarette.

SMOKING CESSATION AND THE WORKPLACE

The workplace is an ideal environment in which to encourage smokers to quit. Employees spend so much time at work that smokefree policies can provide the incentive they need to succeed.

However, quitting is not easy for most smokers. In fact, many smokers try to quit repeatedly before they succeed; others may go through longer term “cycles” of not smoking and then smoking again. Although more than 90% of smokers who quit “for good” do so without a structured smoking cessation program, they may have gained valuable practice in how to quit through previous experience with formal methods, such as those offered at the workplace.

Smoking cessation support at worksites ideally includes a variety of methods and materials to meet the diverse needs of employees who smoke. Although 70% of smokers indicate that they would like to quit smoking,¹ not all smokers will make a serious attempt to quit at the same time, and not all smokers will respond to the same program or “prescription” for quitting. Smokers vary in their readiness to quit. Some may have quit and need support to stay away from cigarettes, some may be ready to try to quit, and others may still be just thinking about it. Still others may not

Smokers quit smoking using many different methods.

Smoking cessation support should include a variety of methods and materials to meet the diverse needs of your employees who smoke.

be ready even to contemplate a cessation attempt. Thus, it is important to consider providing both different types of support and ongoing support (not just when a new policy is announced).

TYPES OF SUPPORT FOR EMPLOYEES WHO SMOKE

A number of choices are available. Voluntary agencies, health departments, and national organizations have developed self-help materials (such as booklets, audiotapes, and videotapes) and group behavior-modification programs (either on-site or in a community setting). In some communities they also sponsor presentations and health fairs at worksites. These resources are listed in Appendix A.

Employees who smoke can be offered a variety of assistance, from comprehensive programs to more limited referrals. Table 4-1 lists some of these types of programs. To tailor a program to fit your company, you can mix and match from these options.

MATCH SUPPORT TO EMPLOYEE NEEDS

One way to decide what kinds of support to offer is to look at the options most helpful for smokers at different stages of readiness to quit, then make sure there is at least one supportive option available for each type of smoker.

NEW EX-SMOKERS

When smokers quit smoking, they may face physical discomfort, weight gain, and stress. The following steps help minimize these obstacles.

- A smokefree work environment may help these ex-smokers by eliminating cues to smoke (e.g., seeing others smoke, ash trays).
- Nicotine replacement therapy reduces withdrawal symptoms.
- Most smoking cessation programs (self-help or formal) include nutritional information and exercise to help manage weight.
- Most cessation programs include stress management techniques such as relaxation training, positive imagery, and deep breathing to decrease the anxiety that surrounds quitting smoking.

TABLE 4-1. OPTIONS TO SUPPORT EMPLOYEES WHO SMOKE

Level of Support	Pros	Cons
Comprehensive • Offer and pay for smoking cessation programs for smoking employees and covered dependents • Provide communication to all employees about changes in smoking policies and support to be offered	• May enhance health status of employees • May help contain health care costs • Allows employer to assess impact of smoking program • More likely to yield changes in smoking behavior • Demonstrates employer's commitment to helping employees who smoke	• More expensive than other options • Requires a significant effort by the employer
Facilitation • Work with health care providers (insurers and Health Maintenance Organizations) to provide smoking cessation to employees • Provide self-help cessation materials • Provide communication to all employees about changes in smoking policies and support to be offered	• May enhance health status of employees • Takes advantage of existing resources • Does not require continuing effort or monitoring by employer	• Requires significant start-up effort • Health care providers may be unwilling to provide support
Referral • Provide employees with information on community smoking cessation programs • Provide self-help cessation materials • Provide communication to all employees about changes in smoking policies and support to be offered	• Takes advantage of existing resources • Less expensive than comprehensive support • Easier to implement than comprehensive support or facilitation	• Less effect on smoking behavior and health care costs

- Social support options include periodic aftercare sessions offered by formal programs, 1-800 numbers, and supportive messages from the company's management.

Many of the people who quit smoking relapse: effective cessation programs (formal or self-help) also should provide

employees with knowledge and skills to help prevent a return to smoking.

EMPLOYEES WHO ARE THINKING ABOUT QUITTING AND THOSE WHO WANT HELP TO QUIT

Employers can support employees who want to quit by offering (or offering

referral to) a variety of kinds of help, including self-help programs, formal cessation programs, counseling from a health care provider, and pharmacological aides. Incentive programs also support smokers' attempts to quit.

EMPLOYEES WHO ARE NOT THINKING ABOUT QUITTING

Communication may help smokers who are not thinking about quitting consider the benefits of quitting. Options include

- providing information through occupational health staff,
- encouraging use of 1-800 number health information lines (1-800-4-CANCER, for example),
- placing articles in company newsletters on the benefits of cessation,
- showing posters and billboards that encourage quit attempts,
- offering health risk appraisals or other health assessments,
- participating in national and international campaigns such as the Great American Smokeout (the Thursday before Thanksgiving), National Employee Health and Fitness Day (the third Wednesday in May), Freedom from Smoking Day

(July 5), and World No-Tobacco Day (May 31), and

- offering incentives to quit.

Social support also can encourage smokers to consider quitting. Such support includes the development of environments in both the worksite and the home that support not smoking. Companies should consider offering and paying for smoking cessation programs for both employees and dependents as a means to foster social support.

CESSATION SUPPORT OPTIONS

Many types of cessation support are available in most communities. Some options are described here. Businesses that do not employ enough smokers to warrant on-site cessation activities can turn to existing community resources.

SELF-HELP PROGRAMS

Self-help programs are attractive to many smokers because they offer privacy and flexibility. Good self-help materials should provide employees with information to

- understand their smoking patterns,
- set quit dates,

- identify and resist smoking cues,
- explore alternatives to smoking,
- control weight gain,
- manage stress, and
- prevent relapse to smoking.

Many self-help materials (e.g., booklets, videotapes, and quit kits) are appropriate for worksites. Voluntary health agencies such as the American Cancer Society and the American Lung Association offer excellent self-help materials. Appendix A provides a listing of some of the many resources available. Review a variety of materials to decide which are most appropriate for your employees.

SMOKING CESSATION GROUP PROGRAMS

Although most smokers quit without formal assistance, some employees need the guidance and support provided by structured programs. Members of a group often provide support and counsel one another. Smoking cessation programs can be contracted to outside providers, or employees can be given a list of programs in the community. You should carefully screen providers before contracting for their services or referring employees. Figure 4-1 provides a checklist for screening such services.

FIGURE 4-1. CHECKLIST: ASSESSING A GROUP CESSATION PROGRAM

1. How long has the organization been in existence? How long has it been providing smoking cessation programs?
2. How many people have gone through the program?
3. Will the approach be appropriate for the employees?
 - a. What methods are used to help smokers quit?
 - b. How is maintaining abstinence from smoking addressed?
 - c. What resources are provided to help promote the program among company employees and stimulate participation?
4. Have others been satisfied with the program?
 - a. Will they provide a list of clients, specifically other employers?
 - b. Will they provide references so you can check for satisfaction and success rates?
5. What are the qualifications of the instructors? What training have they received? What is their cessation counseling experience?
6. Are printed materials appropriate for the educational level of the employees? Are they attractive and motivational?
7. Will the structure of the program accommodate the needs of the employees? Can they
 - a. accommodate all shifts?
 - b. provide on-site and off-site programs?
 - c. structure flexible program formats?
 - d. provide audio or visual equipment?
8. Is the program provider willing to provide ongoing assistance and follow-up once the formal program ends?
9. Does the program incorporate participants' support systems? For example, peers and family members?
10. Does the program offer any form of guarantee? For example, can employees repeat the program for free or at a lower cost?
11. Can the program provider provide evidence of six-month and one-year success rates of previous clients? (A range of 20–40% is realistic.) Remember: if it sounds too good to be true, it probably is.
12. How much does the program cost per employee? Are group discounts available?

PHARMACOLOGICAL AIDS

One important reason why it may be difficult for smokers to quit is nicotine addiction. Some smokers find it helpful to use pharmacological aids such as nicotine gum or the nicotine transdermal patch to help overcome their addiction. Nicotine gum releases the nicotine into the mouth, and the nicotine patch is applied directly to the skin, where it releases a continuous flow of nicotine into the bloodstream.

Nicotine replacement has been found to significantly increase the chances of successful cessation for smokers.⁶ In the spring of 1996, the Food and Drug Administration (FDA) approved the nicotine gum to be marketed as an over-the-counter product, so smokers can now purchase it without a physician's prescription. As of September 1996, the FDA approved two nicotine patches for over-the-counter sale.

Check the company's health plan regarding coverage of nicotine replacement products. Often, if the products are not available through a health plan, employers provide nicotine replacement products directly as a self-funded benefit or will reimburse employees who use the products in an effort to quit smoking.

INCENTIVES

Incentives are most effective in increasing interest in quitting. Even small rewards or recognition, such as in a company newsletter, can help smokers succeed at cessation by providing a concrete goal. Incentives

- give employees a positive focus,
- reinforce motivation,
- encourage cessation program participation, and
- reinforce employees' not smoking.

Advantages of incentives are numerous. Incentives

- are easy to set up and operate,
- are very flexible and adaptable,
- can have significant behavioral impact,
- can be designed for different departments and different levels, and
- can be linked to organizational goals and objectives.

Some disadvantages of incentives also exist. For example,

-
- determining the best reward may be difficult,
 - employers can be outwitted by employees (“false” cessation), and
 - nonsmokers might feel slighted.
- The advantages and disadvantages of incentives highlight the need to carefully plan and implement incentive programs.
- How strongly does the company want to support employees who smoke?
 - What resources are available in the company, community, and elsewhere?
 - How do the employees who smoke feel about pending policies? What kind of support would they like? What kind of support do they expect? How many might be interested in taking advantage of what the company offers?

DECIDING WHAT TYPES OF SUPPORT TO OFFER

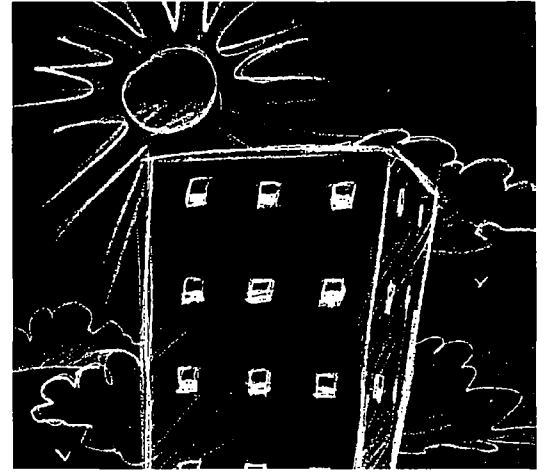
Each company must decide what type of support fits the work situation best. Questions to ask to help decide include the following:

Chapters Three and Four described different kinds of policies and supportive activities from which companies may choose. The next chapter provides a step-by-step guide to planning your policies and activities and making them work.

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C h a p t e r **5**



S T E P - B Y - S T E P :

Y O U R

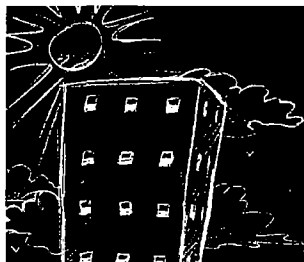
D E C I S I O N S

A N D H O W

T O M A K E

T H E M W O R K

"If you don't know where you are going . . . you might wind up someplace else." Yogi Berra



Gather information, and use this information to guide the development and implementation of the policy.

Although you are not putting the policy "to a vote," most workers do support smokefree policies. Allowing employees to express their opinion will facilitate and guide implementation of the policy.

More and more employers and employees in communities across the nation are working in smoke-free workplaces. Thoughtful planning and a sequenced introduction of a new policy can help ensure the success of your policy. The four steps described in this chapter (Figure 5-1) are based on the experiences of many successful worksite decision makers. Following these steps—with the modifications you make to fit your workplace situation—can smooth the transition to a healthier and safer workplace.

ASSESS THE CURRENT SITUATION

Your first step is to assess the current situation so you know where you are. Review your company's current policies, practices, and employee attitudes in regard to smoking policies. Ask the following questions:

- What are the restrictions on smoking, if any?
- Who is covered by the policy?
- When was the policy established, and why?
- What are the employees' attitudes about the current policy?
- Do employees comply with the policy?
- Who is satisfied or dissatisfied? Why?

Use the checklist (Figure 5-2) to help assess the current situation. Also, review any state or local regulations on smoking in the workplace and get a sense of how other business in your area approach this issue.

FIGURE 5-1. STEPS TO EFFECTIVE WORKPLACE TOBACCO POLICIES

1. Assess the current situation
2. Decide on a new ETS policy and develop a plan to implement it
3. Communicate with employees and management
4. Announce and manage the policy

GATHERING INFORMATION FROM MANAGEMENT AND EMPLOYEES

In addition to your observations about current policies and practices of your company and of other companies, consider the attitudes, beliefs, and behaviors of management and employees before developing new ETS policies and smoking cessation activities. Collect this information in the same way you normally pursue information, such as through meetings, work groups, task forces, or surveys. When forming either formal or informal working groups, consider the following:

- If possible, make sure the group is supported by both management and labor.
- Include representation from (if appropriate)
 - Human Resources,
 - Employee Benefits,
 - Facilities and Operations,
 - Labor Unions,
 - Safety and Health,
 - Corporate Medical,
 - Corporate Training, and
 - Corporate Communications.
- Include smokers, former smokers, and persons who have never smoked on the committee.

FIGURE 5-2. CURRENT SMOKING POLICIES AND PRACTICES

Our current smoking policy allows smokers, including employees and visitors to smoke

- ☐ In offices
- ☐ In designated smoking rooms
- ☐ Other places inside (list: _____)
- ☐ Just outside the front door
- ☐ In the parking lot
- ☐ In designated smoking areas outside
- ☐ In vehicles
- ☐ Other places outside (list: _____)

Smokers, including employees and visitors, currently smoke

- ☐ In offices
- ☐ In designated smoking rooms
- ☐ Other places inside (list: _____)
- ☐ Just outside the front door
- ☐ In the parking lot
- ☐ In designated smoking areas outside
- ☐ In vehicles
- ☐ Other places outside (list: _____)

- Select a group chairperson.
- Establish the mission and goals of the group.
- Communicate the purpose and membership to all employees and managers.

Management expectations often drive corporate policies and programs. It is important to know how senior management feels about certain issues before the plans for the ETS policy and supportive cessation activities are finalized. You may want to share information about the benefits of smokefree policies (see Chapter Two) with management. Find

out what management's opinions are on the following:

- What are the advantages and disadvantages of the company becoming an entirely smokefree facility or of offering separately ventilated areas?
- Should incentives be used to encourage employees not to smoke?
- Should the company offer smoking cessation programs? If so, who should pay for these programs?
- What are four characteristics of the company that will ease or support the modification of ETS policies and cessation activities?
- What are four obstacles within the company that need to be considered before the new ETS policies and cessation activities are implemented?

WORKING WITH LABOR UNIONS

Working with labor unions can be an essential part of gathering information. Three major, relevant labor issues should be addressed:

- whether an employee has the right to smoke at the workplace,

- the relationship of smoking restrictions to collective bargaining, and
- whether a focus on ETS is perceived as a diversion from addressing other occupational hazards.

As discussed in Chapter One, no one has the right to impose a health risk on others, and because an employer has the common-law responsibility to provide a safe work environment, an employer has the right to restrict smoking on company premises and even refuse to hire someone because he or she would smoke on the job.¹ In union-represented work environments, however, the employer may be obligated to subject the policy to collective bargaining, depending on contract provisions. Employers should attempt to work collaboratively with unions rather than act independently to restrict smoking, because management and unions share a fundamental common goal: providing a safe and healthful working environment. Issues such as smoking breaks and provision of smoking cessation support may arise, but these can be addressed within the context of providing a smokefree workplace.

To address the concern that focus on ETS is shielding the issue of other occupational hazards, you should turn to the scientific evidence that ETS is a major workplace hazard and needs to be reduced to the lowest possible levels (Chapter One).

Specific questions to address regarding unions include the following:

- What unions are involved?
- Do the unions have a stated position on smoking restrictions?
- Is there contract language or past practice that addresses smoking on the job by union members?
- What is the opinion of labor relations regarding whether a policy on smoking must be negotiated?
- Can unions be involved in a cooperative policy development effort?
- Is the current state of labor-management relations conducive to a change in smoking policy?

DECIDE ON A NEW ETS POLICY AND DEVELOP A PLAN TO IMPLEMENT IT

Use the information you have gathered to help you decide which ETS policy you will implement. Compare the current situation with the options for workplace policies in Chapter Three. Use the model policy as a reference as well as the sample policies provided in Appendix B. You should also consider how to handle other tobacco products, such as spit tobacco or snuff, if these

FIGURE 5-3. ITEMS TO INCLUDE IN THE SMOKING POLICY

- Purpose for policy (harmful effects of ETS on health)
- A tie between the ETS policy and cessation support and the recognizable corporate values (e.g., performance or employees as an asset)
- Clear statement of where smoking is prohibited
- Clear statement of where smoking is permitted (if anywhere)
- Clear statement on enforcement and consequences of noncompliance
- Clear statement of support to be provided for employees who smoke (e.g., cessation assistance)
- Name and phone number of person who can answer questions about the policy.

products are used by the employees. It is reasonable and consistent to handle all tobacco products at the same time and in the same manner.

Figure 5-3 lists items that the company's policy should include. Circulate this policy to formal or informal working groups for suggestions and modifications to identify any potential problems (such as enforcement issues or choice of outside smoking areas) before you finalize it.

Next, plan how to make the transition smooth and the policy work. Figure 5-4 lists key principles of successfully implementing a smokefree policy. This implementation plan will guide the rest of your actions. You may want to tie significant events (such as the effective date of the policy) to existing events, such as the Great American Smokeout (November) or the season (if your smokers will need to go outside to comply,

FIGURE 5-4. KEY PRINCIPLES OF SUCCESSFUL SMOKEFREE POLICY IMPLEMENTATION²

- Focus on smoke, not the smoker.
- Focus on health and safety regarding ETS, not individual rights.
- Obtain management commitment and support. This support can be made visible to employees through the use of paycheck stuffers, posters, newsletters, and other company communication channels.
- Provide training for middle managers and supervisors on policy communication and enforcement.
- Provide real and visible opportunities for employee participation in policy planning and implementation.
- Educate the workplace community about the hazards of combining ETS and materials used in work processes.
- Allow four to six months from the time of the announcement to implementation, depending on the size of the organization and the magnitude of the change from the old to the new policy.
- To maximize motivation, plan to implement the policy in conjunction with national events such as the American Cancer Society's Great American Smokeout in November or around New Year's Day (when people are making New Year's resolutions).
- Ensure that restrictions and enforcement are equitable across job categories.
- Offer smoking cessation programs to all employees and their families before and after the policy change.
- Enforce the ETS policy just as any other policy would be. Provide training in enforcement for supervisors. Do not differentiate between smoking breaks and any other kind of breaks.
- Anticipate unintended effects (e.g., the concentration of smoke in designated areas).
- Continue to provide smoking cessation educational opportunities and programs after the policy has been implemented to support employees in their attempts to quit smoking and to prevent relapse.

begin during a mild season). The plan should include

- when the policy will be announced (at least four months before the effective date; longer for very large organizations),
- when the policy will become effective,

- events that will be tied to the transition,
- supportive activities for smokers,
- role of contact person listed in policy,
- role(s) of work groups or task forces,
- sufficient time for acquiring appropriate signs to communicate the smokefree policy,
- mechanism for allowing employee feedback during the transition period, and
- time to negotiate and work with labor unions, if needed.

COMMUNICATE WITH EMPLOYEES AND MANAGEMENT

Once you have composed your plan, you will need to inform your employees about the upcoming changes. Remember to consider the need to involve—or at least communicate with—management and labor unions (if present).

It is important that all employees understand the policy changes and the implications of these changes. Midlevel man-

agers or supervisors need to understand their responsibilities for implementing and enforcing the policies. You may want to hold meetings to familiarize them with their roles. These meetings can be useful in anticipating and preparing for potential problems, such as the abuse of break time or tension between smokers and nonsmokers. If there are questions about the new policies, it will be important for you to have answers. Figure 5-5 lists issues with suggested responses.

ANNOUNCE AND MANAGE THE POLICY

You are now ready to formally announce the policy to all employees and prepare for implementation. Suggested actions include the following:

- Announce the policy and cessation support by using your company's standard communication channels (examples include letters to employees and their families, paycheck stuffers, posters, and notices in employee newsletters). It is important to demonstrate that management fully supports the policy (e.g., the announcement should come from the chief executive officer or appropriate senior officer).

FIGURE 5-5. POSSIBLE ISSUES AND SAMPLE RESPONSES

Will there be a reduction in health care costs at the end of year one?

Clarify the difficulties in promising savings in health care costs within one year. If you accurately identify who is smoking before the new policy takes effect, a careful assessment of changes in smoking among employees at the end of year one can be used to calculate the potential cost benefit of the smokefree policy.

Does passive smoking really have any adverse health effects on nonsmokers?

Inform about the known health effects of ETS by using the scientifically referenced information in Chapter One. You may want to seek the support of your corporate medical director or a community health professional.

Should employees be allowed to take time away from their job to participate in smoking cessation activities?

You can plan these programs at times that are not part of the work day but are convenient for employees (e.g., before work, during lunch, or after work). Point out that over the long term, time off to attend smoking cessation programs will add up to less time than employees take to smoke.

Will a smokefree policy result in the loss of smoking employees?

Very few employees leave companies because of implementation of smokefree policies: 3.5% of an extensive small business sample and 2% of another sample said employees left due to a smokefree policy.³

Will a smokefree policy be too difficult to enforce?

Enforcement procedures are almost never needed, because most policies are self-enforcing and compliance is very high.³ Compliance is high because both management and employees usually support the smokefree policy.

Will a smokefree policy alienate clients?

In most cases, clearly posted signs are enough to alert clients to your smokefree policy. Some companies hand out a small card explaining the smokefree policy.

Won't a smokefree policy cost too much time and money to implement?

Experience and limited survey data⁴ have demonstrated that developing and implementing a smokefree policy does not need to be expensive or time-consuming. Costs and time can be saved with a well-thought-out implementation.

SMOKEFREE COFFEE AND DOUGHNUTS, PLEASE

Extensive communications to explain your intent are essential to implementing a smokefree policy, according to Kim Dionne, Director of Marketing for Bess Eaton Coffee Shops and Bakeries. Bess Eaton is a regional bakery in New England, and all of their 55 sites (retail, baking, and management) adopted smokefree policies in January 1994. Prior to that, the company had no formal policy.

"The EPA conclusion that secondhand smoke is a Group A carcinogen made us want to eliminate ETS in our workplace," Ms. Dionne explains. "We wanted to offer a clean and healthful environment to both our external customers and our internal customers—our employees."

To achieve their goal of a smokefree workplace, Bess Eaton worked closely with their local Lung Association. The company gathered key managers as a work group to develop implementation suggestions. The suggestions emphasized the need to explain that the policy is not against smoking or smokers but that it promotes the safest work environment possible.

Bess Eaton wasn't shy about the policy change, and they created a full-blown marketing campaign: "We had speakers and did radio and TV spots. We wanted to make a splash and let our customers know it was coming." The policy was made effective on January 1, 1994, and for the next year the company supported its employees who smoked by fully paying for Lung Association smoking cessation classes.

Reactions to the policy have been very positive, with customers, employees, and management expressing satisfaction. Some changes take time, but everyone has been able to adjust. Ms. Dionne believes that productivity has improved along with the attitude of the employees.

"Though we were one of the first coffee shops in New England to do it and that could be seen as somewhat adventurous, we have been very happy with the results."

- Announce a timeline for implementing the ETS policy and cessation activities. The announcement should be made significantly in advance of the policy's effective date to allow smokers to prepare for changes and to permit any facility or material changes that might be necessary (such as removal of cigarette vending machines, placement of out-

door ashtrays and "Smokefree Area/Thank You for Not Smoking" signs, and necessary ventilation modifications). It is recommended that smoking cessation support be offered at the same time as the advance announcement of the pending policy change as well as before and after the effective date. Strong restrictions on smoking may encourage smokers to think about quitting smoking.

- Obtain signs that communicate a positive "smokefree" message.
- Offer to answer employee questions and invite comment about the ETS policy and cessation activities.
- On the effective date of the policy, you should have
 - signs in place,
 - facility changes complete, and
 - smoking cessation and smoker support ready.

Take advantage of the time between the announcement and the policy effective date to anticipate issues that may arise and work to resolve them. Also, don't stop when you flip the calendar to the effective date of the policy. As with other policies, the implementation of the smokefree policy needs to be monitored for effectiveness. Monitoring allows you to tailor implementation and related cessation support, and it allows

you to report to management and employees about the impact of the policy.

Plan to assess your policy in the short term (first one to three months). Figure 5-6 lists what to assess. Find the answers to these questions:

- Are employees and visitors complying with the policy?
- Are smoking employees using the smoking cessation support available to them?
- Are supervisors encountering any problems?

Gather this information through feedback sessions with supervisors, managers, and employees. If you used a survey earlier, you could conduct a brief follow-up survey at this time. For the smoking cessation support, you should be able to monitor how many people take self-help materials, and your smoking cessation group provider (either in-house or outside) should be able to provide you with information about employees' use of their services.

Consider modifying implementation of your policy on the basis of the results of your assessment. Take steps only if there are significant concerns or problems.

FIGURE 5-6. WHAT TO ASSESS

In the short term

- Changes in exposure to ETS in the work environment
- Number of employees attending cessation activities or using self-help materials
- Awareness of ETS policy
- Employee attitudes toward ETS policy and cessation activities
- Improved employee morale
- Less conflict between smokers and nonsmokers
- Enhanced quality of work
- Improved job satisfaction

In the long term

- Changes in number of employees who smoke
- Effect of cessation activities on successful quitting
- Changes in health risks for smokers and nonsmokers
- Enhanced corporate image
- Improved employee attitude toward health
- Reduced absenteeism
- Reduced health care costs
- Lower accident rate
- Decline in turnover
- Fewer sick days
- Improved productivity

You also may want to evaluate your policy over the longer term (e.g., one to three years). This step will require more effort and time, but it can provide valuable information, such as

- awareness of ETS policy and smoking cessation activities,
- participation in smoking cessation activities,
- effectiveness of smoking cessation activities, and
- management support and ETS policy enforcement.

Obviously you'll want to assess whether your ETS policy and cessation support are working. There are some other reasons for evaluation of ETS policies and cessation activities:

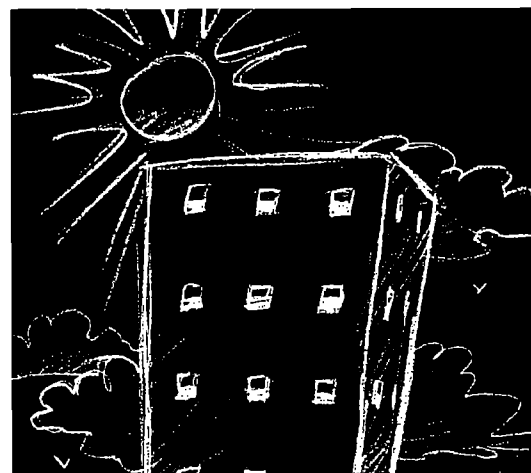
- To identify areas for modification.
- To tell employees and management what happened.
- To provide a structure for the evolution of the policy or activities. All policies and activities need to evolve to meet the changing needs of the organization and employees. Assessment data can help you to justify changes in the policy or activities to meet these changing demands.

This chapter has provided you with detailed suggestions on how to effectively and smoothly implement an ETS policy in your workplace. You will need to tailor the concepts so that they will work in your setting, but the steps have been tested and proven in various environments. The appendices immediately following provides information on organizations to contact for more information, sample policies, and a list of organizations with smokefree workplaces.

REFERENCES

1. Eriksen MP. Workplace smoking control: Rationale and approaches. *Advances in Health Education and Promotion* 1986;1A:65-108.
2. Dana-Farber Cancer Institute, American Cancer Society, and Liberty Mutual Insurance Company, with support from the Massachusetts Department of Public Health. *Guide to Workplace Tobacco Control*. Boston, MA: Dana-Farber Cancer Institute, 1993.
3. Sorensen G, Rosen A, Pinney J, Rudolph J, Doyle N. Work-site smoking policies in small business. *Journal of Occupational Medicine* 1991;33:980-984.
4. Lewit EM, Kerrebrock N, Lewit S, for the COMMIT Research Group. Costs of developing and implementing worksite smoking control policies. Unpublished data.

A p p e n d i c e s



**R E S O U R C E S ,
S A M P L E
P O L I C I E S , A N D
O R G A N I Z A T I O N S
W I T H S M O K E F R E E
W O R K P L A C E S**



APPENDIX A. RESOURCES

This appendix provides information on some of the resources available for use in worksites. The sources of materials are listed rather than all of the available materials. Materials change frequently and there are too many available to adequately catalog them here; the sources, however, remain fairly constant. Included here are non-profit and governmental providers that have developed excellent materials and programs at low or no cost.

These sources can provide information to help you develop your policy (such as fact sheets on the risks from ETS) or smoking cessation support. Ask these sources about materials on policy and cessation at the same time.

Start your search with local and state health departments. Many states have active tobacco control initiatives, and they may have materials and resources that are specifically tailored to your environment. Contact the federal Office on Smoking and Health (listed below) to get the name and number for the appropriate person in your state.

Next, contact your local voluntary health agencies (e.g., the American Cancer Society, the American Heart Association, the local Lung Association). You can find their numbers in the telephone book for your community.

Finally, you can request information from federal government sources.

Office on Smoking and Health

Centers for Disease Control and Prevention

4770 Buford Highway, NE

Mailstop K-50

Atlanta, GA 30341-3724

(770) 488-5705

INTERNET/WEBSITE: <http://www.cdc.gov/nccdphp/osh/tobacco.htm>

National Cancer Institute

9000 Rockville Pike

Building 31, Room 4A-18

Bethesda, MD 20892

(1-800) 4-CANCER

National Institute for Occupational Safety and Health

Centers for Disease Control and Prevention

4676 Columbia Parkway

Cincinnati, OH 45226

(1-800) 356-4674

National Health Information Center

P.O. Box 1133
Washington, DC 20013-1133
(1-800) 336-4797

Agency for Health Care Policy and Research

AHCPR Publications Clearinghouse
P.O. Box 8547
Silver Spring, MD 20907
(1-800) 358-9295 (request materials on the AHCPR Smoking
Cessation Guideline)

Other sources of information include:

Americans for Non-Smokers Rights

Suite J
2530 San Pablo Avenue
Berkeley, CA 94702
(510) 841-3032

Health Enhancement Solutions, Inc.

Suite 505
720 Energy Center Boulevard
Northport, AL 35476
(1-800) 285-5293

Liberty Mutual Insurance Company

(1-800) 320-7581 (request Dana-Farber Cancer Institute "Tobacco
Policy Implementation Kit")

Wellness Councils of America (WELCOA)

Community Health Plaza
Suite 311
7101 Newport Avenue
Omaha, NE 68152-2100
(402) 572-3590

APPENDIX B. SAMPLE POLICIES

The following policies were selected from those of Wellness Councils of America member companies.

BLUE CROSS/BLUE SHIELD OF NEBRASKA

As a major health insurer and concerned employer, we discourage smoking because it is a serious health hazard to both smokers and nonsmokers. Smoking is only permitted in designated locations outside of our buildings. This policy includes nonbusiness hours such as weekends and evenings.

It is our policy to respect the rights of both smokers and nonsmokers in company buildings and facilities.

COLUMBIA NATURAL RESOURCES, INC., WEST VIRGINIA

Policy Title: Smoking Policy

It is the policy of this Company to provide a working environment free of passive smoke and to encourage a smokefree workplace.

Procedure Title: Smoking Policy

Procedure

In addition to the areas covered by Safety Policies and to meet the concerns of employees regarding the hazards of smoking and exposure to smoke and to maintain a safe and healthy environment in which to work, smoking will not be permitted in any building areas occupied by CNR/CGC employees. Where CNR/CGC share building space with other companies or other tenants, employees shall not be permitted to go to those areas to smoke. Smoking will be prohibited in all company vehicles, except where all occupants of the vehicle are smokers. It is the responsibility of each employee to ensure that his/her visitor(s) comply with all provisions of this policy. Any exception to this policy will require the written authorization from the CEO.

Summary

All questions relating to this policy should be directed to the Director-Human Resources.

THE PRINCIPAL FINANCIAL GROUP, IOWA

Policy effective April 1, 1987, smoking was prohibited in the facilities of The Principal Corporate Center

1. The ban includes all office buildings in the Corporate Center complex, Mason City, Grand Island, Waterloo, and those departments of the Corporate Center which are located in space leased in Des Moines by the company.
2. Skywalks and tunnels that connect the Corporate Center buildings are off limits to smoking.
3. Because of their open air design and the brief time people must use them smoking will be allowed in the parking garages. Smoking will not be allowed in the elevators.
4. This ban applies to all persons who are in our buildings, whether or not they are employees of the company. This also includes all contracted service employees.
5. The no smoking policy applies to all office employees and outside business guests who may be visiting the building. An exception may be made on a rare occasion to avoid antagonizing an important customer.
6. Company policy doesn't allow for scheduled breaks. So employees are not allowed to leave the facilities to smoke during working hours.

Administering the Policy

It is very important to be consistent in administering this policy. Violations are treated like any performance problem. If an employee doesn't abide by the policy, first issue a written warning. On the second offense, place the employee on formal, open-end probation (that is, next incident). A third offense results in the employee's termination.

VISTAKON, FLORIDA

Purpose of Policy

It is the philosophy of Vistakon to provide its employees with a work environment that offers the opportunity and resources to optimize their personal health and well-being. In accordance with this philosophy and the convincing evidence of the negative effects of sidestream (passive) smoke, it is Vistakon's intent that all facilities maintain a Smoke-Free environment.

Extent of Policy

The no-smoking policy will apply to all physical facilities owned or leased by Vistakon and located within the domestic U.S., including office buildings, research laboratories, manufacturing plants and distribution centers. Also included under this policy are all company-owned/leased vehicles.

Smoking is permitted on company grounds provided that it occurs beyond 50 feet of any building entrance or in the "external designated smoking" areas. Employees who choose to smoke within the permitted areas must do so on their regularly scheduled breaks or meal periods. No additional time from work shall be authorized for this activity. Each employee is expected to abide by the terms of the Smoke-Free Workplace Policy.

Implementation

Vistakon Live for Life [their in-house health promotion/disease prevention program] will provide, upon request, appropriate smoking cessation resources to interested employees.

Supervisory Responsibility

Management and supervisory staff will be responsible for ongoing compliance with the Smoke-Free Workplace Policy within their work areas. They are expected to adhere to standard practices in resolving issues of nonconformance (in addressing employee complaints) and maintaining expected levels of productivity within their work groups. Policy violation will result in disciplinary action.

APPENDIX C.

ORGANIZATIONS WITH SMOKEFREE WORKPLACES

AT&T

Bell Atlantic

Boeing Electronics and Boeing Computer service, all offices

Bristol-Myers Squibb, nationwide

Calgon, worldwide (includes a property-wide ban)

COMSAT, Washington, DC (includes a property-wide ban)

Continental Airlines

Dunkin' Donuts

Federal Express Corp., nationwide

Hoffman-LaRoche

IBM, nationwide

Johnson & Johnson, worldwide

Levelor-Lorentzen

Lucent Technologies

Mayo Clinic, nationwide (includes a property-wide ban)

McDonald's, corporate owned, all franchises are encouraged to be smokefree

MCI

Merck & Company

Nordstrom, stores and offices nationwide

Northwestern Bell (part of U.S. West)

Pacific Northwestern Bell (part of U.S. West)

Proctor and Gamble

Prudential, nationwide; includes all subsidiaries

Ralston Purina Co.

Sara Lee

Scott Paper Co.

Starbucks, nationwide

Taco Bell, nationwide

Texas Instruments

The Travelers

USA Today

Source: *Smokefree Air Everywhere*, 1996, New Jersey Group Against Smoking Pollution (GASP).

SPECIAL THANKS

The Project Director acknowledges the following individuals in the completion of this guide:

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Wellness Council of Northern Michigan

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Bruce Tusher
AT&T Universal Card Services Corporation

Regina Carlson
New Jersey Group Against Smoking Pollution

Leslie Norman
Centers for Disease Control and Prevention

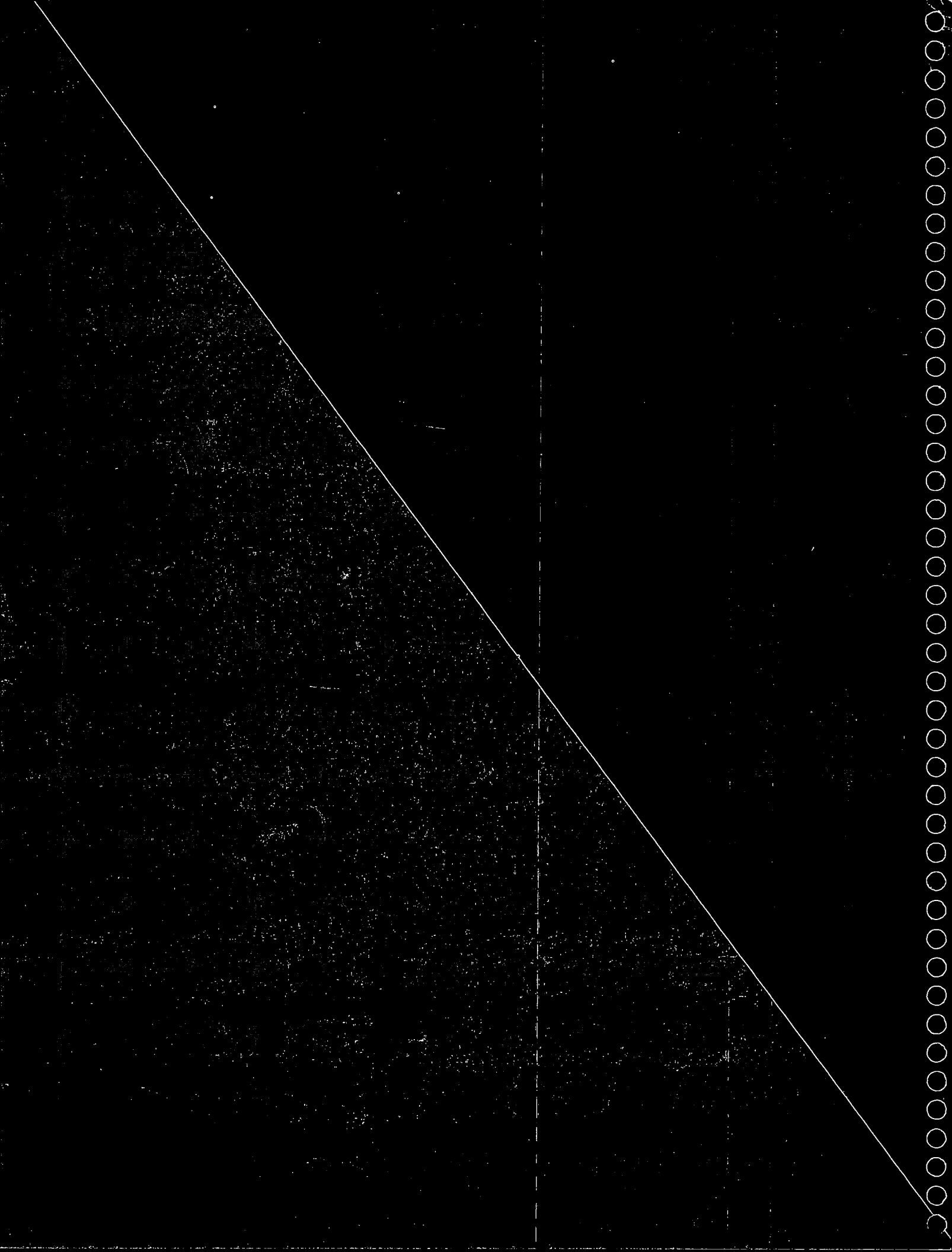
Todd Phillips
Cygnus Corporation

Elizabeth Hess
Cygnus Corporation

Ward Nyholm
Cygnus Corporation

Mary Bedford
Cygnus Corporation

Cheryl Baldwin
High 5 Design



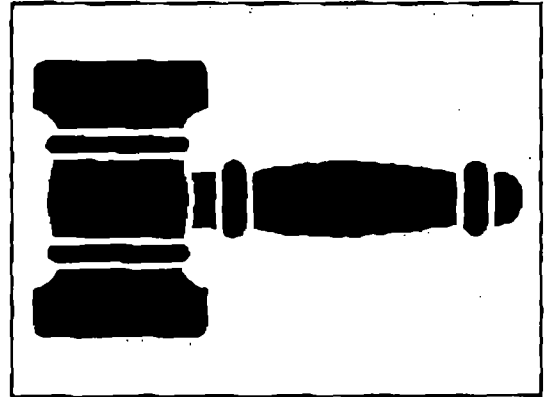
U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES
OFFICE OF THE GENERAL COUNSEL

PHONE: (202) 690-6318

FAX: (202) 690-7998

DATE: June 3, 1997

TO: ELIZABETH DRYE
456-7431



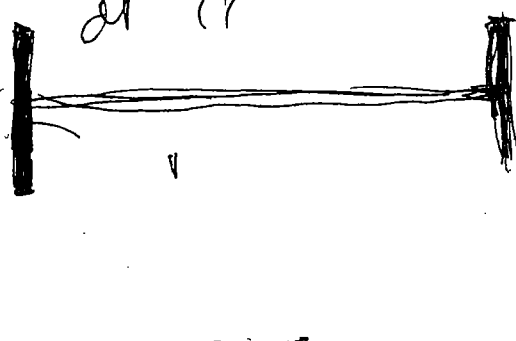
FROM: ANDREW D. HYMAN
Special Assistant to the General Counsel
Office of the General Counsel, Room 707F
U.S. Department of Health and Human Services
200 Independence Ave., SW
Washington, D.C. 20201

COMMENTS: As discussed, attached is background.

No. of Pages (including cover): 2

- The problem of Environmental Tobacco Smoke (ETS) is of great concern, particularly in light of recent studies, such as the one reported a couple weeks ago by the Harvard School of Public Health that found that women who reported being exposed regularly to cigarette smoke at home or work had a 91 percent higher risk of heart attack than those with no exposure.
- Existing GSA regulations that restrict tobacco use were published in 1986 and apply to approximately 10% of Federal domestic facilities. It does not cover Federal buildings under the control of separate management authorities. The GSA regulations permit smoking in designated areas in Federal facilities and do not require that such areas be ventilated separately.
- Federal agencies may prohibit smoking, and some, such as HHS and DOD, have done so. However, most Federal agencies are not smokefree, causing employees and visitors to be exposed to secondhand smoke.

currently looking
at it



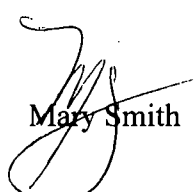
June 3, 1997

Elizabeth Drye:

Attached you will find the materials that you requested:

1. Copy of EPA's risk assessment.
2. Copy of G-7 Declaration plus Implementing Actions. The Declaration is public. I don't think the action items are for public dissemination.
3. List of issues in tobacco suit over risk assessment. This was drafted by an attorney in our General Counsel's office and should be used only for internal consumption. An oral argument is anticipated but a date has not been set.
4. Summary of what EPA is doing on ETS. We can provide more detail if you want it.

I will be out of the office the next few days but in town. You can leave a voice mail at 233-9444. Better yet, Bill Long of my staff can supply you with what you need or answer specific questions. Bill is at 233-9733.



Mary Smith

1997 Declaration of the Environment Leaders of the Eight on Children's Environmental Health

We acknowledge that, throughout the world, children face significant threats to health from an array of environmental hazards. The protection of human health remains a fundamental objective of environmental policies to achieve sustainable development. We increasingly understand that the health and well-being of our families depends upon a clean and healthy environment. Nowhere is this more true than in the case of children, who are particularly vulnerable to pollution. Evidence is growing that pollution at levels or concentrations below existing alert thresholds can cause or contribute to human health problems and our countries' present levels of protection may not, in some cases, provide children with adequate protection.

Among the most important environmental health threats to children worldwide are microbiological and chemical contaminants in drinking water, air pollution that exacerbates illness and death from respiratory problems, polluted waters, toxic substances, pesticides, and ultra-violet radiation. Most of these threats are aggravated for children living in poverty. While not a comprehensive list, we have chosen items for action, enumerated below, because they can benefit most from collective effort by the Eight.

We affirm that prevention of exposure is the single most effective means of protecting children against environmental threats. We seek to improve levels of protection for children, and we reaffirm the priority of children's environmental health in our own countries, as well as in bilateral and multilateral agendas. We agree to cooperate on environmental research, risk assessment, and standard-setting within the jurisdictions of each ministry. We agree to raise public awareness that would enable families to better protect their children's health. We urge our Leaders to make the protection of children's environmental health a high environmental priority and call for international financial institutions, the World Health Organization, the United Nations Environment Programme and other international bodies to continue ongoing activities and give further attention to children's environmental health, in particular the environmental, economic and social dimensions of children's health.

Environmental Risk Assessments & Standard Setting: Historically, due to a lack of comprehensive science, environmental protection programs, standards and testing protocols often have not adequately taken into account nor fully protected infants and children from environmental threats. While our countries have incorporated the precautionary principle or precautionary approaches and safety factors into environmental standard setting, it is important to employ more explicit scientific consideration of children's characteristics and behavior in this process.

We pledge to establish national policies that take into account the specific exposure pathways and dose-response characteristics of children when conducting environmental risk assessments and setting protective standards. We agree there is a need to upgrade testing guidelines to improve our ability to detect risks to children and to assess and evaluate the effects of both single and multiple exposures for children. We urge cooperation through the OECD on adopting revised, harmonized testing guidelines. We will promote research to understand the particular

exposures and sensitivities of infants and children to environmental hazards and exchange research results and information on regulatory decisions. Where there is insufficient information, we agree to pursue the precautionary principle or precautionary approaches to protecting children's health. We call for the consideration of children's environmental health, based on sound science, in the negotiation and implementation of future bilateral, regional and global agreements, such as the negotiations on persistent organic pollutants, long range transboundary air pollution, and trade in particularly dangerous pesticides, chemicals and hazardous wastes.

Children's Exposure to Lead: Lead poisoning is a major environmental hazard to children and our countries have taken many successful actions to reduce children's exposure to lead. Our countries continue to support the reduction in risks from exposure to lead.

We call for further actions that will result in reducing blood lead levels in children to below 10 micrograms per deciliter. Where this blood lead level is exceeded, further action is required. We acknowledge the importance to child health of maternal exposure to lead and agree to reduce maternal exposure.

We commit to fulfill and promote internationally the OECD Declaration on Lead Risk Reduction. We commit to a phase-out of the use of lead in gasoline, the elimination of exposure to lead in products intended for use by children, the phase-out of the use of lead in paint and rust-proofing agents, the restriction of lead in products that may result in ingestion in food and drinking water and to set schedules and develop strategies for elimination or reduction of lead from these sources. In addition, we agree to conduct public awareness campaigns on the risks to children from lead exposure and to develop scientific protocols and programs to monitor blood lead levels in children to track our progress in this important effort.

Microbiologically Safe Drinking Water: Worldwide, the greatest threat to childhood survival is lack of access to clean water, with more than four million children dying annually from diarrheal disease associated with contaminated water. In recent years, a number of countries have experienced serious waterborne disease outbreaks associated with microbial contaminants, such as cryptosporidium and bacterial and viral pathogens. All countries and relevant international organizations should better incorporate the existing knowledge bases into protecting children from microbiological contaminants in drinking water.

We agree to focus increased attention on drinking water disinfection, source water protection and sanitation, as major instruments of good drinking water quality in our national and regional programs, as well as through existing bilateral foreign assistance programs, international organizations and financial institutions. We will facilitate technology transfer to and capacity building in developing countries where micro biologically safe drinking water is a primary child survival concern.

We strongly support the initiative on sustainable use of freshwater for social and economic purposes, including, inter alia, safe drinking water and sanitation, proposed in the context of the preparations for UNGASS and consider that this initiative should make a major contribution to children's health.

We agree to share information and policies among our countries to improve our drinking water standards and will designate officials from our ministries to exchange monitoring data on microbiological drinking water contaminants and waterborne disease outbreaks on a regular

basis. We agree to collaborate on research to support the development of technologies and methods to control disease outbreaks and will give special emphasis to appropriate technologies for small drinking water treatment systems.

Air Quality: Air quality is of particular importance to infants and children, both indoors and outdoors. Childhood asthma and other pediatric respiratory ailments are increasing dramatically in our countries and are substantially exacerbated by environmental pollutants in the air, including emissions from fossil fuel combustion and other sources. While research on children's exposure to some specific air pollutants has been conducted by some of our countries, further research is needed.

We undertake to reduce air pollution in our respective countries, which will alleviate both domestic and transboundary impacts of air quality and, particularly, children's health. Recognizing that indoor air pollution has been identified as a critical problem affecting children's health worldwide, we agree to exchange information on indoor air health threats and remedial measures.

Environmental Tobacco Smoke: Children exposed to environmental tobacco smoke are more likely to suffer from reduced lung function, lower respiratory tract infections and respiratory irritations. Asthmatic children are especially at risk. Many of these symptoms lead to increased hospitalizations of children.

We affirm that environmental tobacco smoke is a significant public health risk to young children and that parents need to know about the risks of smoking in the home around their young children. We agree to cooperate on education and public awareness efforts aimed at reducing children's exposure to environmental tobacco smoke.

Emerging Threats to Children's Health from Endocrine Disrupting Chemicals: There is growing scientific evidence that a variety of environmental contaminants can exert adverse health effects by their ability to alter the functions of hormones within the body. These effects, which include cancer, reproductive disorders, changes in behavior and immune dysfunction, have been observed in laboratory animals exposed to specific chemicals, wildlife populations in several broadly contaminated ecosystems such as the Great Lakes, and to a more limited extent in humans exposed to some organochlorine compounds. Some of these chemicals also are capable of causing long-term neurological damage. Infants and children may be at particular risk to the potential effects of these contaminants. Children may be exposed to endocrine disrupting chemicals *in utero*, through breast milk and in the environment.

We encourage continuing efforts to compile an international inventory of research activities, develop an international assessment of the state of the science, identify and prioritize research needs and data gaps, and develop a mechanism for coordinating and cooperating on filling the research needs. These activities should complement initiatives that are being pursued in international fora such as the Inter-governmental Forum on Chemical Safety (IFCS) and through the work of agencies such as the United Nations Environment Programme. We pledge to develop cooperatively risk management or pollution prevention strategies, as major sources and environmental fates of endocrine disrupting chemicals are identified and will continue to inform the public as knowledge is gained.

Impacts of Global Climate Change to Children's Health: Decisive international action must be taken to confront the problem of global warming including at Kyoto. Our children and future generations face serious threats to their health and welfare from changes in the Earth's climate due the build-up of greenhouse gases in the atmosphere. Overwhelming scientific evidence links human actions to anticipated changes in the global climate system that are likely to result in unacceptable impacts to all nations. In the words of the Intergovernmental Panel on Climate Change: "Climate change is likely to have wide-ranging and mostly adverse impacts on human health, with significant loss of life." Children will be among the most susceptible to more severe heat waves, more intense air pollution, and the spread of infectious diseases and we are only beginning to understand the interactions between these issues and other global trends, such as ozone depletion. Future generations will face many potential impacts of climate change with serious health, environmental and economic consequences.

We must address environmental health threats with a specific focus on children which, for many countries, will require increased coordination between environment, health and other ministries. Countries must increase institutional and other scientific capacities to work on the specific problems of environmental threats to children. We will make the steps agreed upon in this declaration a priority in domestic action plans, report on our progress in carrying out those steps in appropriate international fora and broaden our cooperative efforts on children's environmental health with other countries.

We recognize that environmental threats to children's health must be set in a larger context of poverty alleviation and economic and social development and we urge Leaders to commit to specific results-oriented actions that will accelerate a global transition to sustainable development at The UNGASS and other international fora.

Annex A

Implementation Actions on Protecting Children's Health and Environment Which The Environment Leaders of the Eight Have Agreed to Promote Within Their Governments and Countries

Risk Assessment and Standard Setting

- Urge the OECD to expedite completion of the process of updating and harmonizing developmental and reproductive toxicity testing guidelines.
- Designate officials to work towards enhanced international harmonization of risk assessment approaches that explicitly address environmental risks to children.

Lead

- Each country agrees to develop and share individual country actions to accomplish the goals of the OECD Declaration on lead.
- The Eight will establish principal points of contact and a mechanism for sharing timely information regarding lead hazards in toys and other products to which children might be exposed, including imported products, and will consider other joint actions as appropriate.
- Provide access, on a timely basis, to new technological developments on blood lead level testing.

Microbiologically Safe Drinking Water

- Recommend that foreign assistance programs of the Eight, international organizations, and international financial institutions focus increased attention on drinking water disinfection and source water protection for nations worldwide.
- Designate contact points to exchange monitoring data on microbiological drinking water contaminants and waterborne disease outbreaks.
- Designate contact points to collaborate on research to support the development of technologies and methods, focused on small drinking water systems, to control disease outbreaks.

Endocrine Disrupting Chemicals

- Request that the International Organization on the Management of Chemicals and U.S. EPA complete an international inventory of ongoing research activities.
- Work with UNEP and other appropriate international organizations to complete an international scientific assessment.
- Develop an international research strategy after completion of the inventory and scientific assessment.
- Support an OECD initiative to develop a battery of screening and testing guidelines for endocrine disrupting chemicals that considers the special susceptibilities and exposures to children.

Environmental Tobacco Smoke

- Convene a scientific conference, through WHO or another appropriate scientific organization, to synthesize and share the latest scientific information on risks to infants and children from environmental tobacco smoke and compile information on the most effective educational strategies concerning exposures to children.

Air Quality

- Carry out regional commitments to address transboundary impacts of air pollution.
- Cooperate through existing scientific organizations to enhance the exchange of information on health threats and effective remedial approaches for addressing indoor air quality problems.

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The tobacco industry's arguments are:

A. Procedural challenges

1. EPA is without legal authority to investigate and disseminate information about health hazards of secondhand smoke. Response: The Radon and Indoor Air Quality Research Act (the statutory authority under SARA Title IV under which we acted) specifically authorizes EPA to investigate health effects of indoor air pollutants, and to inform the public of our conclusions. The legislative history to the Radon Act specifically states that we were expected to investigate and publicize risks associated with secondhand smoke

2. EPA failed to comply with procedural requirements of the Radon Act, such as establishing an advisory committee of independent individuals before taking action. Response: this is demonstrably untrue. An advisory committee was formed, and all other requirements were complied with.

B. Challenges to our Study

1. Tobacco industry's main pitch is that we prejudged the issue and manipulated data to validate this prejudged conclusion. This is untrue, and fails to acknowledge that EPA's study and methodology was submitted to the objective Science Advisory Board -- a body of 18 scientists independent of EPA drawn from different ideological bents -- who unanimously concurred with the results of the study and with our scientific methodology.

2. Our study (finding that exposure to secondhand smoke causes lung cancer) is supported by the following key data:

a) secondhand smoke is chemically identical to the known carcinogen mainstream smoke -- it contains the same carcinogens and other pollutants as mainstream smoke (sometimes in higher concentrations). Data show that these carcinogens are absorbed into the blood and other tissues of persons exposed to secondhand smoke, sometimes at fractions as high as 20 % of what an active smoker would absorb. (Jargon for this argument is "bioplausibility"). Tobacco industry has no real answer to any of this data

b) human epidemiologic studies (studies comparing incidence of lung cancer of women who did not smoke and were married to smokers, versus incidence of lung cancer of non-smoking women married to non-smokers) show remarkably consistent evidence of a causal link between exposure to secondhand smoke and lung cancer, a link that cannot be explained by any other causative agent. Particularly compelling evidence is that the studies come from all over the world (so that increased risk can't be attributed to cultural conditions in a particular country or area), and that the likelihood of contracting lung cancer increased as the amount of exposure to secondhand smoke increased (dose-response relation). Tobacco industry has no real answer to these data

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c. EPA's conclusions are consistent with such respected entities as the US Surgeon General and National Academy of Sciences, and (as noted above) were endorsed unanimously by the independent Science Advisory Board

d. Tobacco industry has been saying that we changed our statistical methodology during the course of the study in order that the results of the epidemiologic studies would be statistically significant. This is factually untrue (i.d. we did not change methodology). Also, all of the statistical techniques we used are well recognized, and were endorsed unanimously by the objective Science Advisory Board

EPA Activities on ETS

I. Health Effects:

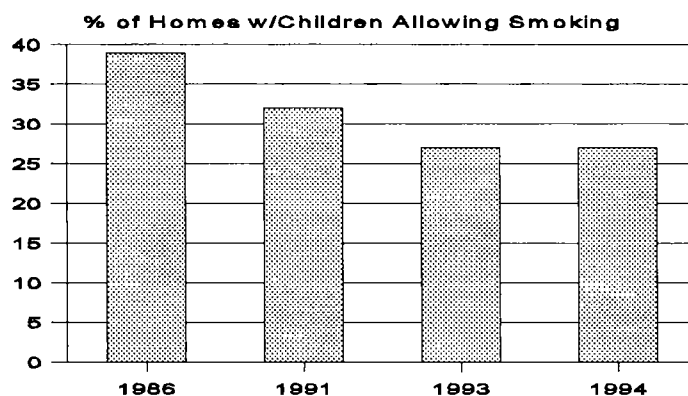
- 3,000 Lung Cancer Deaths
- As many as 300,000 lung infections in Children
- Contributes to Childhood Ear Infections and Asthma (as many as 1 million children have their asthma worsened)
- Non-EPA Research Findings on Heart Disease (40,000 deaths)
- Other Emerging Science on Childhood Effects such as SIDS

II. EPA ETS Goal:

By 2005, The proportion of households in which children 6 and younger are regularly exposed to smoking will be reduced to 15% from over 39% in 1986.

Sources: HHS Healthy People 2000 and EPA Environmental Goals Report

III Recently, National Progress has Slowed.



Note: Each % Point Equals Roughly 250,000 children
Source: National Health Interview Survey (CDC)

IV. Past and Current Projects

- o Development and distribution of Public Information Brochure, including mass-mailing to 200,000 physicians with American Medical Association. 1992-1994
- o Translation of Public Information Brochure into Spanish and Chinese and distribution in collaboration with Consumer Federation of America. 1995

- o Daycare Outreach Pilot project with Pennsylvania Chapter, American Academy of Pediatrics. 1996
- o Expansion of Daycare project to additional states with National Resource center for Health and Safety in Childcare. 1997-2002
- o Culturally-Diverse Church-based outreach program with American Lung Association. 1996-1998
- o Development and promotion of educational materials on the risks of radon and ETS in the African-American community with the National Medical Association. 1995-1997.
- o Development and marketing of speakers kit for pediatricians on ETS with American Academy of Pediatrics. 1997-2000
- o Public Information Campaign to motivate parents to not allow smoking in the home where children are present with the Consumer Research Council.

Conclusions of the 1986 Surgeon General's report on Involuntary Smoking

1. Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers.
2. The children of parents who smoke, compared with the children of nonsmoking parents, have an increased frequency of respiratory infections, increased respiratory symptoms, and slightly smaller rates of increase in lung function as the lung matures.
3. Simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, exposure of nonsmokers to environmental tobacco smoke.

Source: *The Health Consequences of Involuntary Smoking: A Report of the Surgeon General*. 1986. US Dept. Of Health and Human Services

Introduction

Development and Organization of the 1986 Report

The 1986 Report was developed by the Office on Smoking and Health of the U.S. Department of Health and Human Services as part of the Department's responsibility, under Public Law 91-222, to report new and current information on smoking and health to the United States Congress.

The scientific content of this Report reflects the contributions of more than 60 scientists representing a variety of disciplines. Individual manuscripts were written by experts known for their understanding of and work in specific content areas. These manuscripts were refined through a series of meetings attended by the authors, Office on Smoking Health staff and consultants, and the Surgeon General.

Upon receipt of the final manuscripts from the authors, the Office and its consultants edited and consolidated the individual manuscripts into appropriate chapters. These draft chapters were subjected to an extensive outside peer review (see Acknowledgments for individuals and their affiliations) whereby each was reviewed by up to seven experts. Their comments were integrated and the entire volume was assembled. This revised edition of the Report was resubjected to review by 17 distinguished scientists outside the Federal Government, both in this country and abroad. Parallel to this review, the entire Report was also submitted to various institutes and agencies within the U.S. Public Health Service for review and comment.

The 1986 Report contains a Foreword by the Assistant Secretary for Health, a Preface by the Surgeon General of the U.S. Public Health Service, and the following chapters:

- Chapter 1. Introduction, Overview, and Summary and Conclusions
- Chapter 2. Health Effects of Environmental Tobacco Smoke Exposure
- Chapter 3. Environmental Tobacco Smoke Chemistry and Exposures of Nonsmokers
- Chapter 4. Deposition and Absorption of Tobacco Smoke Constituents
- Chapter 5. Toxicity, Acute Irritant Effects, and Carcinogenicity of Environmental Tobacco Smoke
- Chapter 6. Policies Restricting Smoking in Public Places and the Workplace

Overview

Inhalation of tobacco smoke during active cigarette smoking remains the largest single preventable cause of death and disability

for the U.S. population. The health consequences of cigarette smoking and of the use of other tobacco products have been extensively documented in the 17 previous Reports in the health consequences of smoking series issued by the U.S. Public Health Service. Cigarette smoking is a major cause of cancer; it is most strongly associated with cancers of the lung and respiratory tract, but also causes cancers at other sites, including the pancreas and urinary bladder. It is the single greatest cause of chronic obstructive lung diseases. It causes cardiovascular diseases, including coronary heart disease, aortic aneurysm, and atherosclerotic peripheral vascular disease. Maternal cigarette smoking endangers fetal and neonatal health; it contributes to perinatal mortality, low birth weight, and complications during pregnancy. More than 300,000 premature deaths occur in the United States each year that are directly attributable to tobacco use, particularly cigarette smoking.

This Report examines in detail the scientific evidence on involuntary smoking as a potential cause of disease in nonsmokers. Nonsmokers' exposure to environmental tobacco smoke is termed involuntary smoking in this Report because the exposure generally occurs as an unavoidable consequence of being in proximity to smokers, particularly in enclosed indoor environments. The term "passive smoking" is also used throughout the scientific literature to describe this exposure.

The magnitude of the disease risks for active smokers secondary to their "high dose" exposure to tobacco smoke suggests that the "lower dose" exposure to tobacco smoke received by involuntary smokers may also have risks. Although the risks of involuntary smoking are smaller than the risks of active smoking, the number of individuals injured by involuntary smoking is large both in absolute terms and in comparison with the number injured by some other agents in the general environment that are regulated to curtail their potential to cause human illness.

This Report reviews the evidence on the characteristics of mainstream tobacco smoke and of environmental tobacco smoke, on the levels of exposure to environmental tobacco smoke that occur, and on the health effects of involuntary exposure to tobacco smoke. The composition of the tobacco smoke inhaled by active smokers and by involuntary smokers is examined for similarities and differences, and the concentrations of tobacco smoke components that can be measured in a variety of settings are explored, as is smoke deposition and absorption in the respiratory tract. The studies that describe the risks of environmental tobacco smoke exposure for humans are carefully reviewed for their findings and their validity. The evidence on the health effects of involuntary smoking is reviewed for biologic plausibility, and compared with extrapolations of the risks of active

smoking to the lower dose of exposure to tobacco smoke found in nonsmokers. This review leads to three major conclusions:

1. **Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers.**
2. **The children of parents who smoke compared with the children of nonsmoking parents have an increased frequency of respiratory infections, increased respiratory symptoms, and slightly smaller rates of increase in lung function as the lung matures.**
3. **The simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, the exposure of nonsmokers to environmental tobacco smoke.**

The subsequent chapters of this volume describe in detail the evidence that supports these conclusions; the evidence is briefly summarized here.

Environmental Tobacco Smoke Constituents

Important considerations in examining the risks of involuntary smoking are the composition of environmental tobacco smoke (ETS) and its toxicity and carcinogenicity relative to the tobacco smoke inhaled by active smokers. Mainstream cigarette smoke is the smoke drawn through the tobacco into the smoker's mouth. Sidestream smoke is the smoke emitted by the burning tobacco between puffs. Environmental tobacco smoke results from the combination of sidestream smoke and the fraction of exhaled mainstream smoke not retained by the smoker. In contrast with mainstream smoke, ETS is diluted into a larger volume of air, and it ages prior to inhalation.

The comparison of the chemical composition of the smoke inhaled by active smokers with that inhaled by involuntary smokers suggests that the toxic and carcinogenic effects are qualitatively similar, a similarity that is not too surprising because both mainstream smoke and environmental tobacco smoke result from the combustion of tobacco. Individual mainstream smoke constituents, with appropriate testing, have usually been found in sidestream smoke as well. However, differences between sidestream smoke and mainstream smoke have been well documented. The temperature of combustion during sidestream smoke formation is lower than during mainstream smoke formation. As a result, greater amounts of many of the organic constituents of smoke, including some carcinogens, are generated when tobacco burns and forms sidestream smoke than when mainstream smoke is produced. For example, in contrast with mainstream smoke, sidestream smoke contains greater amounts of ammonia, benzene, carbon monoxide, nicotine, and the carcinogens

1. SUMMARY AND CONCLUSIONS

1.1. MAJOR CONCLUSIONS

Based on the weight of the available scientific evidence, the U.S. Environmental Protection Agency (EPA) has concluded that the widespread exposure to environmental tobacco smoke (ETS) in the United States presents a serious and substantial public health impact.

In adults:

- ETS is a human lung carcinogen, responsible for approximately 3,000 lung cancer deaths annually in U.S. nonsmokers.

In children:

- ETS exposure is causally associated with an increased risk of lower respiratory tract infections (LRIs) such as bronchitis and pneumonia. This report estimates that 150,000 to 300,000 cases annually in infants and young children up to 18 months of age are attributable to ETS.
- ETS exposure is causally associated with increased prevalence of fluid in the middle ear, symptoms of upper respiratory tract irritation, and a small but significant reduction in lung function.
- ETS exposure is causally associated with additional episodes and increased severity of symptoms in children with asthma. This report estimates that 200,000 to 1,000,000 asthmatic children have their condition worsened by exposure to ETS.
- ETS exposure is a risk factor for new cases of asthma in children who have not previously displayed symptoms.

1.2. BACKGROUND

Tobacco smoking has long been recognized (e.g., U.S. Department of Health, Education, and Welfare [U.S. DHEW], 1964) as a major cause of mortality and morbidity, responsible for an estimated 434,000 deaths per year in the United States (Centers for Disease Control [CDC], 1991a). Tobacco use is known to cause cancer at various sites, in particular the lung (U.S. Department of Health and Human Services [U.S. DHHS], 1982; International Agency for Research on Cancer [IARC], 1986). Smoking can also cause respiratory diseases (U.S. DHHS, 1984, 1989) and is a major risk factor for heart disease (U.S. DHHS, 1983). In recent years, there has been concern that nonsmokers may also be at risk for some of these health effects as a result of their exposure ("passive smoking") to the tobacco smoke that occurs in various environments occupied by smokers. Although this ETS is dilute compared with the mainstream smoke (MS) inhaled by active smokers, it is chemically similar, containing many of the same carcinogenic and toxic agents.

In 1986, the National Research Council (NRC) and the Surgeon General of the U.S. Public Health Service independently assessed the health effects of exposure to ETS (NRC, 1986; U.S. DHHS, 1986). Both of the 1986 reports conclude that ETS can cause lung cancer in adult nonsmokers and that children of parents who smoke have increased frequency of respiratory symptoms and acute lower respiratory tract infections, as well as evidence of reduced lung function.

More recent epidemiologic studies of the potential associations between ETS and lung cancer in nonsmoking adults and between ETS and noncancer respiratory effects more than double the size of the database available for analysis from that of the 1986 reports. This EPA report critically reviews the current database on the respiratory health effects of passive smoking; these data are utilized to develop a hazard identification for ETS and to make quantitative estimates of the public health impacts of ETS for lung cancer and various other respiratory diseases.

The weight-of-evidence analysis for the lung cancer hazard identification is developed in accordance with U.S. EPA's *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 1986a) and established principles for evaluating epidemiologic studies. The analysis considers animal bioassays and genotoxicity studies, as well as biological measurements of human uptake of tobacco smoke components and epidemiologic data on active and passive smoking. The availability of abundant and consistent human data, especially human data at actual environmental levels of exposure to the specific agent (mixture) of concern, allows a hazard identification to be made with a high degree of certainty. The conclusive evidence of the dose-related lung carcinogenicity of

MS in active smokers (Chapter 4), coupled with information on the chemical similarities of MS and ETS and evidence of ETS uptake in nonsmokers (Chapter 3), is sufficient by itself to establish ETS as a known human lung carcinogen, or "Group A" carcinogen under U.S. EPA's carcinogen classification system. In addition, this document concludes that the overall results of 30 epidemiologic studies on lung cancer and passive smoking (Chapter 5), using spousal smoking as a surrogate of ETS exposure for female never-smokers, similarly justify a Group A classification.

The weight-of-evidence analyses for the noncancer respiratory effects are based primarily on a review of epidemiologic studies (Chapter 7). Most of the endpoints examined are respiratory disorders in children, where parental smoking is used as a surrogate of ETS exposure. For the noncancer respiratory effects in nonsmoking adults, most studies used spousal smoking as an exposure surrogate. A causal association was concluded to exist for a number of respiratory disorders where there was sufficient consistent evidence for a biologically plausible association with ETS that could not be explained by bias, confounding, or chance. The fact that the database consists of human evidence from actual environmental exposure levels gives a high degree of confidence in this conclusion. Where there was suggestive but inconclusive evidence of causality, as was the case for asthma induction in children, ETS was concluded to be a risk factor for that endpoint. Where data were inconsistent or inadequate for evaluation of an association, as for acute upper respiratory tract infections and acute middle ear infections in children, no conclusions were drawn.

This report also has attempted to provide estimates of the extent of the public health impact, where appropriate, in terms of numbers of ETS-attributable cases in nonsmoking subpopulations. Unlike for qualitative hazard identification assessments, where information from many sources adds to the confidence in a weight-of-evidence conclusion, for quantitative risk assessments, the usefulness of studies usually depends on how closely the study population resembles nonsmoking segments of the general population. For lung cancer estimates among U.S. nonsmokers, the substantial epidemiology database of ETS and lung cancer among U.S. female never-smokers was considered to provide the most appropriate information. From these U.S. epidemiology studies, a pooled relative risk estimate was calculated and used in the derivation of the population risk estimates. The large number of studies available, the generally consistent results, and the condition of actual environmental levels of exposure increase the confidence in these estimates. Even under these circumstances, however, uncertainties remain, such as in the use of questionnaires and current biomarker measurements to estimate past exposure, assumptions of exposure-response linearity, and extrapolation to male never-smokers and to ex-smokers. Still, given the strength of the evidence for the lung carcinogenicity of tobacco smoke and the extensive human database from actual environmental exposure levels, fewer assumptions are necessary than

is usual in EPA quantitative risk assessments, and confidence in these estimates is rated medium to high.

Population estimates of ETS health impacts are also made for certain noncancer respiratory endpoints in children, specifically lower respiratory tract infections (i.e., pneumonia, bronchitis, and bronchiolitis) and episodes and severity of attacks of asthma. Estimates of ETS-attributable cases of LRI in infants and young children are thought to have a high degree of confidence because of the consistent study findings and the appropriateness of parental smoking as a surrogate measure of exposure in very young children. Estimates of the number of asthmatic children whose condition is aggravated by exposure to ETS are less certain than those for LRIs because of different measures of outcome in various studies and because of increased extraparental exposure to ETS in older children. Estimates of the number of new cases of asthma in previously asymptomatic children also have less confidence because at this time the weight of evidence for asthma induction, while suggestive of a causal association, is not conclusive.

Most of the ETS population impact estimates are presented in terms of ranges, which are thought to reflect reasonable assumptions about the estimates of parameters and variables required for the extrapolation models. The validity of the ranges is also dependent on the appropriateness of the extrapolation models themselves.

While this report focuses only on the respiratory health effects of passive smoking, there also may be other health effects of concern. Recent analyses of more than a dozen epidemiology and toxicology studies (e.g., Steenland, 1992; National Institute for Occupational Safety and Health [NIOSH], 1991) suggest that ETS exposure may be a risk factor for cardiovascular disease. In addition, a few studies in the literature link ETS exposure to cancers of other sites; at this time, that database appears inadequate for any conclusion. This report does not develop an analysis of either the nonrespiratory cancer or the heart disease data and takes no position on whether ETS is a risk factor for these diseases. If it is, the total public health impact from ETS will be greater than that discussed here.

1.3. PRIMARY FINDINGS

A. Lung Cancer in Nonsmoking Adults

1. Passive smoking is causally associated with lung cancer in adults, and ETS, by the total weight of evidence, belongs in the category of compounds classified by EPA as Group A (known human) carcinogens.
2. Approximately 3,000 lung cancer deaths per year among nonsmokers (never-smokers and former smokers) of both sexes are estimated to be attributable to ETS in the United States. While there are statistical and modeling uncertainties

in this estimate, and the true number may be higher or lower, the assumptions used in this analysis would tend to underestimate the actual population risk. The overall confidence in this estimate is medium to high.

B. Noncancer Respiratory Diseases and Disorders

1. Exposure of children to ETS from parental smoking is causally associated with:
 - a. increased prevalence of respiratory symptoms of irritation (cough, sputum, and wheeze),
 - b. increased prevalence of middle ear effusion (a sign of middle ear disease), and
 - c. a small but statistically significant reduction in lung function as tested by objective measures of lung capacity.
2. ETS exposure of young children and particularly infants from parental (and especially mother's) smoking is causally associated with an increased risk of LRIs (pneumonia, bronchitis, and bronchiolitis). This report estimates that exposure to ETS contributes 150,000 to 300,000 LRIs annually in infants and children less than 18 months of age, resulting in 7,500 to 15,000 hospitalizations. The confidence in the estimates of LRIs is high. Increased risks for LRIs continue, but are lower in magnitude, for children until about age 3; however, no estimates are derived for children over 18 months.
3.
 - a. Exposure to ETS is causally associated with additional episodes and increased severity of asthma in children who already have the disease. This report estimates that ETS exposure exacerbates symptoms in approximately 20% of this country's 2 million to 5 million asthmatic children and is a major aggravating factor in approximately 10%.
 - b. In addition, the epidemiologic evidence is suggestive but not conclusive that ETS exposure increases the number of new cases of asthma in children who have not previously exhibited symptoms. Based on this evidence and the known ETS effects on both the immune system and lungs (e.g., atopy and airway hyperresponsiveness), this report concludes that ETS is a risk factor for the induction of asthma in previously asymptomatic children. Data suggest that relatively high levels of exposure are required to induce new cases of asthma in children. This report calculates that previously asymptomatic children exposed to ETS from mothers who smoke at least 10 cigarettes per day will exhibit an estimated 8,000 to 26,000 new cases of

asthma annually. The confidence in this range is medium and is dependent on the conclusion that ETS is a risk factor for asthma induction.

4. Passive smoking has subtle but significant effects on the respiratory health of nonsmoking adults, including coughing, phlegm production, chest discomfort, and reduced lung function.

This report also has reviewed data on the relationship of maternal smoking and sudden infant death syndrome (SIDS), which is thought to involve some unknown respiratory pathogenesis. The report concludes that while there is strong evidence that infants whose mothers smoke are at an increased risk of dying from SIDS, available studies do not allow us to differentiate whether and to what extent this increase is related to in utero versus postnatal exposure to tobacco smoke products. Consequently, this report is unable to assert whether or not ETS exposure by itself is a risk factor for SIDS independent of smoking during pregnancy.

Regarding an association of parental smoking with either upper respiratory tract infections (colds and sore throats) or acute middle ear infections in children, this report finds the evidence inconclusive.

1.3.1. ETS and Lung Cancer

1.3.1.1. Hazard Identification

The Surgeon General (U.S. DHHS, 1989) estimated that smoking was responsible for more than one of every six deaths in the United States and that it accounted for about 90% of the lung cancer deaths in males and about 80% in females in 1985. Smokers, however, are not the only ones exposed to tobacco smoke. The sidestream smoke (SS) emitted from a smoldering cigarette between puffs (the main component of ETS) has been documented to contain virtually all of the same carcinogenic compounds (known and suspected human and animal carcinogens) that have been identified in the mainstream smoke (MS) inhaled by smokers (Chapter 3). Exposure concentrations of these carcinogens to passive smokers are variable but much lower than for active smokers. An excess cancer risk from passive smoking, however, is biologically plausible.

Based on the firmly established causal association of lung cancer with active smoking with a dose-response relationship down to low doses (Chapter 4), passive smoking is considered likely to affect the lung similarly. The widespread presence of ETS in both home and workplace and its absorption by nonsmokers in the general population have been well documented by air sampling and by body measurement of biomarkers such as nicotine and cotinine (Chapter 3). This raises the question of whether any direct evidence exists for the relationship between ETS exposure and lung cancer in the general population and what its implications may be for public health. This

report addresses that question by reviewing and analyzing the evidence from 30 epidemiologic studies of effects from normally occurring environmental levels of ETS (Chapter 5). Because there is widespread exposure and it is difficult to construct a truly unexposed subgroup of the general population, these studies attempt to compare individuals with higher ETS exposure to those with lower exposures. Typically, female never-smokers who are married to a smoker are compared with female never-smokers who are married to a nonsmoker. Some studies also consider ETS exposure of other subjects (i.e., male never-smokers and long-term former smokers of either sex) and from other sources (e.g., workplace and home exposure during childhood), but these studies are fewer and represent fewer cases, and they are generally excluded from the analysis presented here. Use of the female never-smoker studies provides the largest, most homogeneous database for analysis to determine whether an ETS effect on lung cancer is present. This report assumes that the results for female never-smokers are generalizable to all nonsmokers.

Given that ETS exposures are at actual environmental levels and that the comparison groups are both exposed to appreciable background (i.e., nonspousal) ETS, any excess risk for lung cancer from exposure to spousal smoke would be expected to be small. Furthermore, the risk of lung cancer is relatively low in nonsmokers, and most studies have a small sample size, resulting in a very low statistical power (probability of detecting a real effect if it exists). Besides small sample size and low incremental exposures, other problems inherent in several of the studies may also limit their ability to detect a possible effect. Therefore, this report examines the data in several different ways. After downward adjustment of the relative risks for smoker misclassification bias, the studies are individually assessed for strength of association, both for the overall data and for the highest exposure group when exposure-level data are available, and for exposure-response trend. Then the study results are pooled by country using statistical techniques for combining data, including both positive and nonpositive results, to increase the ability to determine whether or not there is an association between ETS and lung cancer. Finally, in addition to the previous statistical analyses that weight the studies only by size, regardless of design and conduct, the studies are qualitatively evaluated for potential confounding, bias, and likely utility to provide information about any lung carcinogenicity of ETS. Based on these qualitative considerations, the studies are categorized into one of four tiers and then statistically analyzed successively by tier.

Results from all of the analyses described above strongly support a causal association between lung cancer ETS exposure. The overall proportion (9/30) of individual studies found to show an association between lung cancer and spousal ETS exposure at all levels combined is unlikely to occur by chance ($p < 10^{-4}$). When the analysis focuses on higher levels of spousal exposure, every one of the 17 studies with exposure-level data shows increased risk in the highest

exposure group; 9 of these are significant at the $p < 0.05$ level, despite most having low power, another result highly unlikely to occur by chance ($p < 10^{-7}$). Similarly, the proportion (10/14; $p < 10^{-9}$) showing a statistically significant exposure-response trend is highly supportive of a causal association.

Combined results by country showed statistically significant associations for Greece (2 studies), Hong Kong (4 studies), Japan (5 studies), and the United States (11 studies), and in that order of strength of relative risk. Pooled results of the four Western European studies (three countries) actually showed a slightly stronger association than that of the United States, but it was not statistically significant, probably due to the smaller sample size. The combined results of the Chinese studies do not show an association between ETS and lung cancer; however, two of the four Chinese studies were designed mainly to determine the lung cancer effects of high levels of other indoor air pollutants indigenous to those areas, which would obscure a smaller ETS effect. These two Chinese studies do, however, provide very strong evidence on the lung carcinogenicity of these other indoor air pollutants, which contain many of the same components as ETS. When results are combined only for the other two Chinese studies, they demonstrate a statistically significant association for ETS and lung cancer.

The heterogeneity of observed relative risk estimates among countries could result from several factors. For example, the observed differences may reflect true differences in lung cancer rates for never-smokers, in ETS exposure levels from nonspousal sources, or in related lifestyle characteristics in different countries. For the time period in which ETS exposure was of interest for these studies, spousal smoking is considered to be a better surrogate for ETS exposure in more "traditional" societies, such as Japan and Greece, than in the United States. In the United States, other sources of ETS exposure (e.g., work and public places) are generally higher, which obscures the effects of spousal smoking and may explain the lower relative risks observed in the United States. Nevertheless, despite observed differences between countries, all showed evidence of increased risk.

Based on these analyses and following the U.S. EPA's *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 1986a), EPA concludes that environmental tobacco smoke is a Group A (known human) carcinogen. This conclusion is based on a total weight of evidence, principally:

- Biological plausibility. ETS is taken up by the lungs, and components are distributed throughout the body. The presence of the same carcinogens in ETS and MS, along with the established causal relationship between lung cancer and active smoking with the dose-response relationships exhibited down to low doses, establishes the plausibility that ETS is also a lung carcinogen.

- Supporting evidence from animal bioassays and genotoxicity experiments. The carcinogenicity of tobacco smoke has been demonstrated in lifetime inhalation studies in the hamster, intrapulmonary implantations in the rat, and skin painting in the mouse. There are no lifetime animal inhalation studies of ETS; however, the carcinogenicity of SS condensates has been shown in intrapulmonary implantations and skin painting experiments. Positive results of genotoxicity testing for both MS and ETS provide corroborative evidence for their carcinogenic potential.
- Consistency of response. All 4 of the cohort studies and 20 of the 26 case-control studies observed a higher risk of lung cancer among the female never-smokers classified as ever exposed to any level of spousal ETS. Furthermore, every one of the 17 studies with response categorized by exposure level demonstrated increased risk for the highest exposure group. When assessment was restricted to the 19 studies judged to be of higher utility based on study design, execution, and analysis (Appendix A), 17 observed higher risks, and 6 of these increases were statistically significant, despite most having low statistical power. Evaluation of the total study evidence from several perspectives leads to the conclusion that the observed association between ETS exposure and increased lung cancer occurrence is not attributable to chance.
- Broad-based evidence. These 30 studies provide data from 8 different countries, employ a wide variety of study designs and protocols, and are conducted by many different research teams. Results from all countries, with the possible exception of two areas of China where high levels of other indoor air lung carcinogens were present, show small to modest increases in lung cancer associated with spousal ETS exposure. No alternative explanatory variables for the observed association between ETS and lung cancer have been indicated that would be broadly applicable across studies.
- Upward trend in exposure-response. Both the largest of the cohort studies--the Japanese study of Hirayama with 200 lung cancer cases--and the largest of the case-control studies--the U.S. study by Fontham and associates (1991) with 420 lung cancer cases and two sets of controls--demonstrate a strong exposure-related statistical association between passive smoking and lung cancer. This upward trend is well supported by the preponderance of epidemiology studies. Of the 14 studies that provide sufficient data for a trend test by exposure level, 10 were statistically significant despite most having low statistical power.
- Detectable association at environmental exposure levels. Within the population of married women who are lifelong nonsmokers, the excess lung cancer risk from

exposure to their smoking husbands' ETS is large enough to be observed, even for all levels of their spousal exposure combined. Carcinogenic responses are usually detectable only in high-exposure circumstances, such as occupational settings, or in experimental animals receiving very high doses. In addition, effects are harder to observe when there is substantial background exposure in the comparison groups, as is the case here.

- Effects remain after adjustment for potential upward bias. Current and ex-smokers may be misreported as never-smokers, thus inflating the apparent cancer risk for ETS exposure. The evidence remains statistically significant and conclusive, however, after adjustments for smoker misclassification. For the United States, the summary estimate of relative risk from nine case-control plus two cohort studies is 1.19 (90% confidence interval [C.I.] = 1.04, 1.35; $p < 0.05$) after adjustment for smoker misclassification. For Greece, 2.00 (1.42, 2.83), Hong Kong, 1.61 (1.25, 2.06), and Japan, 1.44 (1.13, 1.85), the estimated relative risks are higher than those of the United States and more highly significant after adjusting for the potential bias.
- Strong associations for highest exposure groups. Examining the groups with the highest exposure levels increases the ability to detect an effect, if it exists. Nine of the sixteen studies worldwide for which there are sufficient exposure-level data are statistically significant for the highest exposure group, despite most having low statistical power. The overall pooled estimate of 1.81 for the highest exposure groups is highly statistically significant (90% C.I. = 1.60, 2.05; $p < 10^{-6}$). For the United States, the overall pooled estimate of 1.38 (seven studies, corrected for smoker misclassification bias) is also highly statistically significant (90% C.I. = 1.13, 1.70; $p = 0.005$).
- Confounding cannot explain the association. The broad-based evidence for an association found by independent investigators across several countries, as well as the positive exposure-response trends observed in most of the studies that analyzed for them, make any single confounder highly unlikely as an explanation for the results. In addition, this report examined potential confounding factors (history of lung disease, home heat sources, diet, occupation) and concluded that none of these factors could account for the observed association between lung cancer and ETS.

1.3.1.2. Estimation of Population Risk

The individual risk of lung cancer from exposure to ETS does not have to be very large to translate into a significant health hazard to the U.S. population because of the large number of smokers and the widespread presence of ETS. Current smokers comprise approximately 26% of the U.S. adult population and consume more than one-half trillion cigarettes annually (1.5 packs per day, on average), causing nearly universal exposure to at least some ETS. As a biomarker of tobacco smoke uptake, cotinine, a metabolite of the tobacco-specific compound nicotine, is detectable in the blood, saliva, and urine of persons recently exposed to tobacco smoke. Cotinine has typically been detected in 50% to 75% of reported nonsmokers tested (50% equates to 63 million U.S. nonsmokers age 18 or older).

The best estimate of approximately 3,000 lung cancer deaths per year in U.S. nonsmokers age 35 and over attributable to ETS (Chapter 6) is based on data pooled from all 11 U.S. epidemiologic studies of never-smoking women married to smoking spouses. Use of U.S. studies should increase the confidence in these estimates. Some mathematical modeling is required to adjust for expected bias from misclassification of smoking status and to account for ETS exposure from sources other than spousal smoking. The overall relative risk estimate of 1.19 for the United States, already adjusted for smoker misclassification bias, becomes 1.59 after adjusting for background ETS sources (1.34 for nonspousal exposures only). Assumptions are also needed to relate responses in female never-smokers to those in male never-smokers and ex-smokers of both sexes, and to estimate the proportion of the nonsmoking population exposed to various levels of ETS. Overall, however, the assumptions necessary for estimating risk add far less uncertainty than other EPA quantitative assessments. This is because the extrapolation for ETS is based on a large database of human studies, all at levels actually expected to be encountered by much of the U.S. population.

The components of the 3,000 lung cancer deaths figure include approximately 1,500 female never-smokers, 500 male never-smokers, and 1,000 former smokers of both sexes. More females are estimated to be affected because there are more female than male nonsmokers. These component estimates have varying degrees of confidence; the estimate of 1,500 deaths for female never-smokers has the highest confidence because of the extensive database. The estimate of 500 for male never-smokers is less certain because it is based on the female never-smoker response and is thought to be low because males are generally subject to higher background ETS exposures than females. Adjustment for this higher background exposure would lead to higher risk estimates. The estimate of 1,000 lung cancer deaths for former smokers of both sexes is

considered to have the lowest confidence, and the assumptions used are thought to make this estimate low as well.

Workplace ETS levels are generally comparable with home ETS levels, and studies using body cotinine measures as biomarkers demonstrate that nonspousal exposures to ETS are often greater than exposure from spousal smoking. Thus, this report presents an alternative breakdown of the estimated 3,000 ETS-attributable lung cancer deaths between spousal and nonspousal exposures. By extension of the results from spousal smoking studies, coupled with biological measurements of exposure, more lung cancer deaths are estimated to be attributable to ETS from combined nonspousal exposures--2,200 of both sexes--than from spousal exposure--800 of both sexes. This spouse-versus-other-sources partitioning depends on current exposure estimates that may or may not be applicable to the exposure period of interest. Thus, this breakdown contains this element of uncertainty in addition to those discussed above with respect to the previous breakdown.

An alternative analysis, based on the large Fontham et al. (1991) study, which is the only study that provides biomarker estimates of both relative risk and ETS exposure, yields population risk point estimates of 2,700 and 3,600. These population risk estimates are highly consistent with the estimate of 3,000 based on the combined U.S. studies.

While there is statistical variance around all of the parameters used in the quantitative assessment, the two largest areas of uncertainty are probably associated with the relative risk estimate for spousal ETS exposure and the parameter estimate for the background ETS exposure adjustment. A sensitivity analysis that independently varies these two estimates yields population risk estimates as low as 400 and as high as 7,000. These extremes, however, are considered unlikely; the more probable range is narrower, and the generally conservative assumptions employed suggest that the actual population risk number may be greater than 3,000. Overall, considering the multitude, consistency, and quality of all these studies, the weight-of-evidence conclusion that ETS is a known human lung carcinogen, and the limited amount of extrapolation necessary, the confidence in the estimate of approximately 3,000 lung cancer deaths is medium to high.

1.3.2. ETS and Noncancer Respiratory Disorders

Exposure to ETS from parental smoking has been previously linked with increased respiratory disorders in children, particularly in infants. Several studies have confirmed the exposure and uptake of ETS in children by assaying saliva, serum, or urine for cotinine. These cotinine concentrations were highly correlated with smoking (especially by the mother) in the child's presence. Nine to twelve million American children under 5 years of age, or one-half to

two-thirds of all children in this age group, may be exposed to cigarette smoke in the home (American Academy of Pediatrics, 1986; Overpeck and Moss, 1991).

With regard to the noncancer respiratory effects of passive smoking, this report focuses on epidemiologic evidence appearing since the two major reports of 1986 (NRC and U.S. DHHS) that bears on the potential association of parental smoking with detrimental respiratory effects in their children. These effects include symptoms of respiratory irritation (cough, sputum production, or wheeze); acute diseases of the lower respiratory tract (pneumonia, bronchitis, and bronchiolitis); acute middle ear infections and indications of chronic middle ear infections (predominantly middle ear effusion); reduced lung function (from forced expiratory volume and flow-rate measurements); incidence and prevalence of asthma and exacerbation of symptoms in asthmatics; and acute upper respiratory tract infections (colds and sore throats). The more than 50 recently published studies reviewed here essentially corroborate the previous conclusions of the 1986 reports of the NRC and Surgeon General regarding respiratory symptoms, respiratory illnesses, and pulmonary function, and they strengthen support for those conclusions by the additional weight of evidence (Chapter 7). For example, new data on middle ear effusion strengthen previous evidence to warrant the stronger conclusion in this report of a causal association with parental smoking. Furthermore, recent studies establish associations between parental smoking and increased incidence of childhood asthma. Additional research also supports the hypotheses that in utero exposure to mother's smoke and postnatal exposure to ETS alter lung function and structure, increase bronchial responsiveness, and enhance the process of allergic sensitization, changes that are known to predispose children to early respiratory illness. Early respiratory illness can lead to long-term pulmonary effects (reduced lung function and increased risk of chronic obstructive lung disease).

This report also summarizes the evidence for an association between parental smoking and SIDS, which was not addressed in the 1986 reports of the NRC or Surgeon General. SIDS is the most common cause of death in infants ages 1 month to 1 year. The cause (or causes) of SIDS is unknown; however, it is widely believed that some form of respiratory pathogenesis is generally involved. The current evidence strongly suggests that infants whose mothers smoke are at an increased risk of dying of SIDS, independent of other known risk factors for SIDS, including low birthweight and low gestational age, which are specifically associated with active smoking during pregnancy. However, available studies do not allow this report to conclude whether that increased risk is related to in utero versus postnatal exposure to tobacco smoke products, or to both.

The 1986 reports of the NRC and Surgeon General conclude that both the prevalence of respiratory symptoms of irritation and the incidence of lower respiratory tract infections are higher in children of smoking parents. In the 18 studies of respiratory symptoms subsequent to

the 2 reports, increased symptoms (cough, phlegm production, and wheezing) were observed in a range of ages from birth to midteens, particularly in infants and preschool children. In addition to the studies on symptoms of respiratory irritation, 10 new studies have addressed the topic of parental smoking and acute lower respiratory tract illness in children, and 9 have reported statistically significant associations. The cumulative evidence is conclusive that parental smoking, especially the mother's, causes an increased incidence of respiratory illnesses from birth up to the first 18 months to 3 years of life, particularly for bronchitis, bronchiolitis, and pneumonia. Overall, the evidence confirms and strengthens the previous conclusions of the NRC and Surgeon General.

Recent studies also solidify the evidence for the conclusion of a causal association between parental smoking and increased middle ear effusion in young children. Middle ear effusion is the most common reason for hospitalization of young children for an operation.

At the time of the Surgeon General's report on passive smoking (U.S. DHHS, 1986), data were sufficient to conclude only that maternal smoking may influence the severity of asthma in children. The recent studies reviewed here strengthen and confirm these exacerbation effects. The new evidence is also conclusive that ETS exposure increases the number of episodes of asthma in children who already have the disease. In addition, the evidence is suggestive that ETS exposure increases the number of new cases of asthma in children who have not previously exhibited symptoms, although the results are statistically significant only with children whose mothers smoke 10 or more cigarettes per day. While the evidence for new cases of asthma itself is not conclusive of a causal association, the consistently strong association of ETS both with increased frequency and severity of the asthmatic symptoms and with the established ETS effects on the immune system and airway hyperresponsiveness lead to the conclusion that ETS is a risk factor for induction of asthma in previously asymptomatic children.

Regarding the effects of passive smoking on lung function in children, the 1986 NRC and Surgeon General reports both conclude that children of parents who smoke have small decreases in tests of pulmonary output function of both the larger and smaller air passages when compared with the children of nonsmokers. As noted in the NRC report, if ETS exposure is the cause of the observed decrease in lung function, the effect could be due to the direct action of agents in ETS or an indirect consequence of increased occurrence of acute respiratory illness related to ETS.

Results from eight studies on ETS and lung function in children that have appeared since those reports add some additional confirmatory evidence suggesting a causal rather than an indirect relationship. For the population as a whole, the reductions are small relative to the interindividual variability of each lung function parameter. However, groups of particularly susceptible or heavily exposed children have shown larger decrements. The studies reviewed

suggest that a continuum of exposures to tobacco products starting in fetal life may contribute to the decrements in lung function found in older children. Exposure to tobacco smoke products inhaled by the mother during pregnancy may contribute significantly to these changes, but there is strong evidence indicating that postnatal exposure to ETS is an important part of the causal pathway.

With respect to lung function effects in adults exposed to ETS, the 1986 NRC and Surgeon General reports found the data at that time inconclusive, due to high interindividual variability and the existence of a large number of other risk factors, but compatible with subtle deficits in lung function. Recent studies confirm the association of passive smoking with small reductions in lung function. Furthermore, new evidence also has emerged suggesting a subtle association between exposure to ETS and increased respiratory symptoms in adults.

Some evidence suggests that the incidence of acute upper respiratory tract illnesses and acute middle ear infections may be more common in children exposed to ETS. However, several studies failed to find any effect. In addition, the possible role of confounding factors, the lack of studies showing clear dose-response relationships, and the absence of a plausible biological mechanism preclude more definitive conclusions.

In reviewing the available evidence indicating an association (or lack thereof) between ETS exposure and the different noncancer respiratory disorders analyzed in this report, the possible role of several potential confounding factors was considered. These include other indoor air pollutants; socioeconomic status; effect of parental symptoms; and characteristics of the exposed child, such as low birthweight or active smoking. No single or combined confounding factors can explain the observed respiratory effects of passive smoking in children.

For diseases for which ETS has been either causally associated (LRIs) or indicated as a risk factor (asthma cases in previously asymptomatic children), estimates of population-attributable risk can be calculated. A population risk assessment (Chapter 8) provides a probable range of estimates that 8,000 to 26,000 cases of childhood asthma per year are attributable to ETS exposure from mothers who smoke 10 or more cigarettes per day. The confidence in this range of estimates is medium and is dependent on the suggestive evidence of the database. While the data show an effect only for children of these heavily smoking mothers, additional cases due to lesser ETS exposure also are a possibility. If the effect of this lesser exposure is considered, the range of estimates of new cases presented above increases to 13,000 to 60,000. Furthermore, this report estimates that the additional public health impact of ETS on asthmatic children includes more than 200,000 children whose symptoms are significantly aggravated and as many as 1,000,000 children who are affected to some degree.

This report estimates that ETS exposure contributes 150,000 to 300,000 cases annually of lower respiratory tract illness in infants and children younger than 18 months of age and that 7,500 to 15,000 of these will require hospitalization. The strong evidence linking ETS exposure to increased incidence of bronchitis, bronchiolitis, and pneumonia in young children gives these estimates a high degree of confidence. There is also evidence suggesting a smaller ETS effect on children between the ages of 18 months and 3 years, but no additional estimates have been computed for this age group. Whether or not these illnesses result in death has not been addressed here.

In the United States, more than 5,000 infants die of SIDS annually. It is the major cause of death in infants between the ages of 1 month and 1 year, and the linkage with maternal smoking is well established. The Surgeon General and the World Health Organization estimate that more than 700 U.S. infant deaths per year from SIDS are attributable to maternal smoking (CDC, 1991a, 1992b). However, this report concludes that at present there is not enough direct evidence supporting the contribution of ETS exposure to declare it a risk factor or to estimate its population impact on SIDS.

Mary Smith
Director of
Indoor
Environment
EPA - Office Indoor Air
and Radiation -
Wash. D.C.

233-9030
233-9370

Risk Assessment - 1992 EPA report
- 3,000 lung cancer deaths
- bronchitis/pneumonia in kids
- nothing new in science - risk assessment

- Still in courts - sued by tobacco industry -

- Day case module, w/ Am. Academy of Pediatrics -
- education - Community education credit for taking
the course
- work to influence parents
prior to public service campaigns
- planning on doing public service campaigns -

Cooperative agreement: Consumer Federation
of America (non lobbying)
- Am. Medical Assn. -

EPA Focus -

→ Pentium
Early May
Shaden
- Makes
on ETS -

G-7 declaration action item - on ETS - sis. pub. health risk -
cooperate on education and public awareness
- SIDS
- low birth rate
- scientific conference on [kids] -

{ Environment
ministries &
Communique

Cooperative

G-7 piece July - other public outreach -

- District Cr. - N.C. - { sharing case -
procedural challenge -

but Thurs. - Afternoon Fr.

Elissa Feldman Associate Director -

301-717-8025

- ETS That pediatricians would
use in giving talks to community
groups

- Public Service Campaign

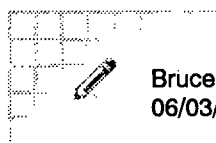
- In next two weeks sign coop
agreement with consumer
research center - non-binding arm
of consumer Fed of America

- Productions will take place over the
next year

- Part of general budget \$350,000 -

- AMA will endorse

- Statement by Surgeon General on second
ETS -



Bruce N. Reed
06/03/97 12:54:52 PM

Record Type: Record

To: Elizabeth Drye/OPD/EOP

cc:

Subject: Re: Smoking in Federal Buildings 

Does this EO affect military operations? embassies?

THE WHITE HOUSE
WASHINGTON

April 2, 1993

MEMORANDUM FOR EXECUTIVE OFFICE OF THE PRESIDENT EMPLOYEES

FROM: DAVID WATKINS *D. Watkins*
ASSISTANT TO THE PRESIDENT FOR
MANAGEMENT AND ADMINISTRATION

SUBJECT: Smoking Policy

The Environmental Protection Agency and the Surgeon General have concluded that passive smoking exposure is a major health hazard. Effective today, the following smoking policy will be implemented in the Executive Office of the President (EOP).

Smoking is prohibited in all buildings in the EOP complex. This includes the New and Old Executive Office Buildings, the Winder Building, and the Jackson Place Townhouses.

If you have any questions regarding this policy, please contact John Dawson, Acting Director, Facilities Management Division, Office of Administration, on 395-2335.

We appreciate your cooperation.

THE WHITE HOUSE

WASHINGTON

September 28, 1995

MEMORANDUM FOR ALL EXECUTIVE OFFICE OF THE PRESIDENT EMPLOYEES

FROM:

JODIE R. TORKELSON
ASSISTANT TO THE PRESIDENT FOR
MANAGEMENT AND ADMINISTRATION

SUBJECT:

Smoking Policy

This is a reminder that, by GSA regulation, smoking is prohibited in all buildings of the Executive Office of the President (EOP) complex. This includes the Old Executive Office Building, the New Executive Office Building, the Winder Building, and all of the Jackson Place Townhouses.

Although smoking is permitted outside of the buildings, be courteous to others and do not smoke near doorways, walkways and stairways.

Your compliance with the no smoking policy is greatly appreciated. Should you have any questions regarding this policy, please contact my office at extension 6-2861.

Withdrawal/Redaction Marker

Clinton Library

DOCUMENT NO. AND TYPE	SUBJECT/TITLE	DATE	RESTRICTION
001. memo	Gary Walters to Ann Stock et al re: Smoking [partial] (1 page)	02/01/1993	b(6)

COLLECTION:

Clinton Presidential Records
Domestic Policy Council
Elizabeth Drye
OA/Box Number: 13021

FOLDER TITLE:

Second Hand Smoke

2014-0046-S
rc1420

RESTRICTION CODES

Presidential Records Act - [44 U.S.C. 2204(a)]

- P1 National Security Classified Information [(a)(1) of the PRA]
- P2 Relating to the appointment to Federal office [(a)(2) of the PRA]
- P3 Release would violate a Federal statute [(a)(3) of the PRA]
- P4 Release would disclose trade secrets or confidential commercial or financial information [(a)(4) of the PRA]
- P5 Release would disclose confidential advice between the President and his advisors, or between such advisors [(a)(5) of the PRA]
- P6 Release would constitute a clearly unwarranted invasion of personal privacy [(a)(6) of the PRA]

C. Closed in accordance with restrictions contained in donor's deed of gift.

PRM. Personal record misfile defined in accordance with 44 U.S.C. 2201(3).

RR. Document will be reviewed upon request.

Freedom of Information Act - [5 U.S.C. 552(b)]

- b(1) National security classified information [(b)(1) of the FOIA]
- b(2) Release would disclose internal personnel rules and practices of an agency [(b)(2) of the FOIA]
- b(3) Release would violate a Federal statute [(b)(3) of the FOIA]
- b(4) Release would disclose trade secrets or confidential or financial information [(b)(4) of the FOIA]
- b(6) Release would constitute a clearly unwarranted invasion of personal privacy [(b)(6) of the FOIA]
- b(7) Release would disclose information compiled for law enforcement purposes [(b)(7) of the FOIA]
- b(8) Release would disclose information concerning the regulation of financial institutions [(b)(8) of the FOIA]
- b(9) Release would disclose geological or geophysical information concerning wells [(b)(9) of the FOIA]

THE WHITE HOUSE

WASHINGTON

February 1, 1993

MEMORANDUM TO: ALL EXECUTIVE RESIDENCE STAFF

ANN STOCK
SOCIAL SECRETARY

JANET GREEN
SPECIAL ASSISTANT TO THE PRESIDENT AND
DIRECTOR OF WHITE HOUSE OPERATIONS

(b)(6), (b)(7)c, (b)(7)e, (b)(7)f
DEPUTY CHIEF, WHITE HOUSE BRANCH
USSS UNIFORMED DIVISION

0001]

RICHARD S. MILLER
SPECIAL AGENT IN CHARGE
USSS PRESIDENTIAL PROTECTIVE DIVISION

ANN EDWARDS
DIRECTOR OF PRESS ADVANCE

LISA CAPUTO
PRESS SECRETARY FOR MRS. CLINTON

FROM: GARY J. WALTERS
CHIEF USHER



SUBJECT: SMOKING

In compliance with recent medical opinions and the desires of the President and Mrs. Clinton "to provide a smoke-free environment to enhance the health of all Americans", we will require those who desire to smoke, do so outside the Executive Residence. My earlier memorandum of November 12, 1991, on the same subject, is no longer applicable.

There will be two designated smoking areas for those who wish to continue smoking, although you are encouraged to **STOP SMOKING** for your own health. Sand filled ashtray stands will be provided at the east and west ends of the tradesman's entrance under the North Portico. These are the only two areas adjacent to the Executive Residence where smoking will be permitted.

THURS.
10:00 AM
Waxman ETS bill 1994 - passed subcommittee

90% ↓ in use in 6 years by brand
if companies fail to do it

1st yr \$1 pack
non compliance: 2nd yr \$2 1/2

3rd yr \$3 1/2 Entire tobacco line

H _____ V8 _____ H

Scruggs - declare bankruptcy, reorganize, no payments.

- won't happen

ETS - Mahan, Hansen - House

Senate - Durbin performance std
Cautenberg ETS bill

Pres Conference -

Koop / Kessler - going on Thursday, 1st meeting
meet intensively in June - come up
w/ package by the end of the month
Koop / Kessler surgery

Public
Health
Committee
9:30 am
12:30 am

- Settlement agreement - analyze -

- If not, there is what policy -

- ASSIST. States

- Minority committee / public

- public session -

- public - -

If there is a settlement - Koop / Kessler -

ETS

- Jan - Feb '94 hearing - pull w/surgeon
generals

- March '94 -

McDonalds of Bk, Wendy's Pizza (bars), Dunkin'
Donuts.
Nat. ~~Chain~~ ^{Corner} Chain Restaurants
Building Owners' Manager's Assn.
Commercial real estate industry

MD, CA restaurant Assn

Am. Council P. of Ltr. Ind.

- AIAA #2

- AIA Architects

- Artists

- Merch

- So. Cal Edison

- The Rauss Company

Health Organiz

ASTHO

22 State Attorneys General -

Our Fire Protection Assn

PTA

- Time Magazine -

~~ETS~~

- Appropriation -

- FDA Commissioner -

- Second-hand smoke..

ETS/EPA

- Litigation risk assessment - NC

- education

\$350,000

- PSA next year w/AMA, Consumer

- child care workers, pediatricians

- G-8 Paragraph

- CSTA

Response to negotiators

Kendall/Koon Commission

International -

Steve - @ Liabilities on Ruminant Damages.

OSHA workplace std

Steve Gashell

Steve Gashell

~~219-7162~~

219-7162
Ext 117

Settlement Talks

April 94 proposed
comment period
closed.

Jan '96

Not a ban - confines smoking
to designated, ventilated space.

- John Banzhaf - Asst -

Sued OSHA

- Asst is alleging that
Carter era ...

- Science base is only
epi - may want to
corroborate with
tox studies, interaction
w/other science.

Re. EPA - Significant - 10

VO. 105 - Test.
but was right this to
do

- application of workplace
rules to bars/casinos -

- doesn't cover municipal
employees.

- preemption concern

- draw it up could
be preempted

- Not getting it out in
next year.

- more research not 10

EO

Lawyer

* Smoking in Fed Buildings.



jmande @ Bangate.fda.gov

06/02/97 03:15:00 PM

Record Type: Record

To: Elizabeth Drye

cc:

Subject: 2d hand smoke

French Panel Attacks 2nd-Hand Smoke

Associated Press

Thursday, May 29, 1997 3:01 pm EDT

By MATTHEW GLEDHILL

PARIS (AP) -- Second-hand smoke kills thousands of people in France every year, the National Medical Academy said in its strongest indictment of smoking in a country where cigarettes are still highly popular.

It called for harsher measures to prevent the effects of passive smoking, calling smoking "the most serious cause of (indoor) air pollution" in France.

A report released Wednesday by cardiologist Maurice Tubiana concluded that up to 3,000 non-smokers in France die each year of heart-related illness directly related to inhaling the smoke of others.

Non-smoking men whose partners smoke are 20 percent more likely suffer heart disease, while an increase of 10 percent was noted among women in the same situation, the report said.

Due to higher levels of heart disease in the United States, passive smoking there could be responsible for between 30,000 and 60,000 deaths each year, Tubiana said.

Non-smokers living with a partner who smokes have 35 percent greater possibility of contracting lung cancer, the report added.

In contrast to sharply lower rates in the United States, just under half of French men smoke, as do one-third of French women and young people between 14 and 19.

Non-smoking sections have been required in public places since 1991, but the law is often ignored. Cigarette smoke is inescapable in restaurants and bars.

"I don't mind if you eat while I smoke," is a common French joke to foreigners unaccustomed to the lax attitude to smoking.

French trains and airplanes have reduced the number of smoking sections, but national carrier Air France still maintains a separate "smoking bar" on many long-haul flights.

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

April 3, 1997

cc: Ten Klein

~~Eli~~

Elizabeth Dye
+ return

OFFICE OF
THE ADMINISTRATOR

MEMORANDUM FOR ELENA KAGAN, DOMESTIC POLICY COUNCIL

FROM: Gary S. Guzy *Gary S. Guzy*
Counselor to the Administrator

*Eli - could you look
into second-hand
smoke projects in PP 2-3?
Thanks.*

SUBJECT: EPA POLICIES TARGETED TO CHILDREN IN THEIR EARLIEST
YEARS

In response to the President's February 24 Memorandum, attached please find a list of EPA's existing and planned efforts to enhance the development of children during their earliest years of life. Our compilation builds upon EPA's September 1996 report *Environmental Health Threats to Children* and will be expanded through the work of EPA's new Office of Children's Health Protection.

Because so many of our efforts are directly linked to communities across the country, we also have collected information about programs taking place at EPA's ten regional offices. We will forward this list to you shortly.

If you have questions or would like further information, please call me or Courtney Manning at (202) 260-7960.

cc: Pauline Abernathy
Nicole Rabner



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EPA POLICIES TARGETED TO CHILDREN IN THEIR EARLIEST YEARS

Existing and Planned Projects and Programs

April 3, 1997

THE EPA OFFICE OF CHILDREN'S HEALTH PROTECTION AND CHILDREN'S ENVIRONMENTAL HEALTH INITIATIVE

In February, 1997 Administrator Browner established a new Office of Children's Health Protection to carry out the Agency's National Agenda announced in September 1996 to address the wide array of complex environmental threats to children's health, from asthma-exacerbating air pollution to toxic chemicals that can lead to serious health problems. This new office will be located within EPA's Office of the Administrator. It will:

- **Review EPA health standards to make sure they are protective of children,** beginning with selecting five of the most significant current EPA standards, and establishing procedures for reviewing new standards as they are developed.
- **Coordinate children's health issues across the Agency,** by chairing a new EPA Board on Children's Environmental Health that will assure integration of EPA activities affecting children; and by working with the Agency's Science Policy Council, Regulatory Policy Council and program and regional offices to coordinate regulatory and other actions that affect children's health.
- **Research and set new policies on children's susceptibility and exposure to pollutants.** To ensure that all EPA efforts use the best information in developing protections for children, the new Office will work with researchers at EPA, other federal agencies and academic institutions to identify and expand research on children's health. The new Office will also develop new, comprehensive policies that address children's cumulative and simultaneous exposures to environmental health threats, and will work with other offices within EPA to develop a research agenda on children's environmental health issues.
- **Expand community right-to-know and education on children's health.** The new Office will carry out a Family Right-to-Know Initiative to expand access to vital information about children's environmental health, so that families can make informed choices concerning environmental exposures to their children. The new Office also will lead Agency efforts to provide parents, teachers, and health professionals with education and information about protecting children from environmental health threats in their homes, schools, and communities.

Funding: \$7.5 million in FY97

New Policy on Evaluating Health Risks to Children

New Program

In October 1995, EPA established a new Agency-wide policy that, for the first time, will ensure that we consistently and explicitly evaluate environmental health risks of infants and children in all of the risk assessments, risk characterizations, and environmental and public health standards that we set for the nation.

This is not a new idea to the many programs throughout the Agency that currently consider children's health issues in assessing overall risk. This is, however, a major step forward in establishing a consistent nationwide children's environmental health policy. We know that children have a greater potential for exposure to environmental hazards and our assessments of health risks do not always fully take into account the potential effects on this vulnerable population. The National Academy of Sciences has called for policy changes to reflect children's health factors in evaluating environmental risks.

EPA's new policy will allow the Agency to make better public health decisions that reflect not just data on adults, but on children, whenever possible. By making children a health priority, we expect that this policy will encourage new, much-needed research to provide the child-specific data we need to thoroughly evaluate the health risks children and infants face from pollution in our air, land and water. *(A description of EPA's other science-related activities is listed in the last section.)*

SECOND-HAND SMOKE

Infants and young children whose parents smoke are among the most seriously affected by exposure to environmental tobacco smoke, or second hand smoke, which is a carcinogen and presents significant health risks to children. Children are at increased risk of lower respiratory tract infections such as pneumonia and bronchitis. *A 1992 EPA risk assessment found that second hand smoke is responsible for between 150,000 and 300,000 lower respiratory tract infections in infants and children under 18 months of age annually and results in 7,500 to 15,000 hospitalizations each year.* EPA is taking or planning to take the following actions to address the health risks of second-hand smoke:

Second-hand Smoke -- Public Service Campaign

Planned Project

EPA will partner with the American Medical Association (AMA) to conduct a national TV, radio, print and transit public service advertising campaign to educate parents about how second-hand smoke contributes to early childhood ear infections and respiratory problems. Campaign materials will be produced in both English and Spanish. The promotion will be targeted to low income, urban populations where second-hand smoke exposure is believed to be highest. Materials distribution would take place through AMA, the Centers for Disease Control and Prevention and EPA.

Impact and Funding: *In 1996, 29% of U.S. households still exposed young children to second-hand smoke.* The funding level is \$350,000 in FY97. A similar amount is planned in FY98 for another version of the campaign.

Daycare Outreach regarding Indoor Air Quality

Current Project

Building on the success of a pilot project underway in the State of Pennsylvania, EPA is working with the National Resource Center for Health and Safety in Childcare to expand a project that is reducing childhood exposure to second-hand smoke.

This pilot, which will be expanded to 5-8 more states this year, provides a self-learning program for childcare center staff to assist them in educating parents about reducing childhood exposure to second-hand smoke. After successfully completing the program's test and committing to implement risk-reducing actions, childcare staff members are awarded continuing education credits in the state's licensing system.

Approximately 100 providers have implemented the program, *reaching more than 1000 young children in Pennsylvania*. Exportation to additional states will take place during FY 1997 and FY 1998, and, in the future, include other indoor air problems which children face.

Impact and Funding: It is anticipated that 50-200 facilities per state will be served. The funding level in FY97 is \$30,000.

Second-hand Smoke -- Pediatrician Speaker Kits

Planned Project

Working with EPA, the American Academy of Pediatrics (AAP) is promoting community-based action to reduce the risks, particularly to young children, from second-hand smoke and other indoor air pollutants. The AAP will use its existing child health care network and membership outreach systems to develop and market a kit for pediatricians to use when addressing public groups in their community on second-hand smoke and other indoor air quality issues. EPA estimates that *exposure to second-hand smoke annually causes up to 300,000 cases of childhood respiratory infection and causes as many as 1 million asthmatic children to have additional episodes and increased severity of symptoms*.

Funding: \$37,000 for FY1997.

INDOOR AIR QUALITY

Indoor Air Quality in Schools

Current Project

It is estimated that between 550,000 and 1.8 million pre-school aged children can be found in school buildings every day. Healthy indoor air, among other factors, contributes to a healthy and favorable learning environment for children. To address this issue, EPA, in conjunction with the National PTA, National Education Association, Association of School Business Officials, Council for American Private Education, American Federation of Teachers, and the American Lung Association, has developed an easy-to-use kit called *IAQ Tools for Schools*. The kit empowers schools to carry out a practical plan of action to prevent and resolve indoor air quality problems at little or no cost using simple activities and in-house staff.

The *IAQ Tools for Schools* Kit has repeatedly been called the best guidance for schools to use in preventing and resolving indoor air problems. It is endorsed by many of the major national school organizations, and whole school districts and major urban school systems have adopted the Kit. Many national advocacy organizations are promoting the Kit as an easy and effective program for minimizing adverse health and learning impacts for children. Strong support for the Kit has been voiced by State departments of education and departments of health, and State legislatures are using the Kit as a guide to develop indoor air quality standards in schools.

Funding: Funding for *IAQ Tools for Schools* outreach and implementation is \$1.3 million for FY1997 (this is for all elementary and secondary schools, regardless of whether preschool children are found in the school). An equivalent amount is targeted for FY98.

PROTECTION FROM UV RAYS

Children's Sun Protection Campaign

Current Project

In order to address *overexposure to the sun's harmful ultraviolet light, which can seriously damage a young child's skin*, EPA will develop and launch a Children's Sun Protection Campaign during this year and next.

The Campaign, which includes targeting pre-school children, will educate parents and teachers about the risks of overexposure to the sun, especially the harmful UV radiation that is most intense between 11:00 am and 2:00 pm. It will encourage parents and teachers to take sun protective actions to reduce a child's lifetime risk of developing skin cancer. EPA will promote the program through partnerships with Broadcast Meteorologists and major educational associations such as the National Parent Teachers Association and the National Education Association.

Funding: The funding level in FY97 is \$50,000 for planning; \$250,000 in FY98 for implementation.

PROTECTING YOUNG CHILDREN FROM LEAD POISONING

Lead poisoning is a top environmental health hazard for young children, *affecting nearly 1 million children age five and under*, according to Centers for Disease Control and Prevention (CDC) data. Lead poisoning causes IQ deficiencies, reading and learning disabilities, impaired hearing, reduced attention spans, hyperactivity, antisocial behavior, and other problems in young children. Pregnant women poisoned by lead can transfer lead to a developing fetus, resulting in adverse developmental effects.

Although lead-based house paint has long since been taken off the market, children living in older homes are threatened by chipping or peeling lead paint, and excessive amounts of lead-contaminated dust. More than 80 percent of U.S. homes built before 1978 -- some 64 million -- contain lead paint.

To address this most pressing remaining need in lead poisoning prevention, EPA's lead initiatives will continue to be a central piece of EPA's strategy for protecting children. EPA has identified children *under the age of six* as the population of highest concern in the development of its lead risk reduction program and has placed significant emphasis on the specialized exposure concerns related to children. EPA has, and will continue to assure that all standards are especially protective of children and have a continued focus on the exposure situations which are most relevant to this sensitive sub-population.

Real Estate Disclosure Rule

Current Program

As part of the Administration's community right-to-know efforts, EPA and HUD took action in March 1996 to require sellers and landlords to disclose any known lead-based paint to home buyers, allowing them the option of conducting a lead hazard assessment. Approximately 64 million dwellings -- all built before 1978 -- contain some lead-based paint, although much of it can be managed and maintained safely by using simple, low-cost common sense procedures. Particularly at risk are families renovating older structures and low-income families living in dilapidated housing. Parents can unknowingly poison their children when they disrupt lead-based painted surfaces during renovations, for example.

Funding: \$32,000 for FY1997

Residential Lead Exposure

Current Project

EPA is currently in the process of identifying what specific levels of lead contamination in residential dust and soil present hazards to children, as well as identifying what conditions of paint present hazards. As part of this project, EPA is developing a comprehensive assessment of residential lead risks to children. This project will result in clear, identifiable standards for what constitutes a lead hazard to children in paint, dust and soil.

Funding: \$1 million for FY1997

Lead Hazards During Renovation and Remodeling

Planned Program

Renovation and remodeling activities are often forgotten when thinking about lead hazards, but these activities can actually exacerbate existing situations by disturbing lead paint and creating lead dust, often where it is least expected. Parents can unknowingly poison their young children when they disrupt lead-based painted surfaces during renovations, for example.

EPA will develop a rule to ensure that high-risk renovation and remodeling activities are done safely, by requiring training and safe practices. EPA is currently developing a regulation to require that renovation and remodeling contractors educate their customers about lead by distributing a brochure previously developed by EPA.

Funding: \$759,000 in FY1997

**Rulemaking on Do-It-Yourself Debris From
Removal of Lead Based Paint**

Planned Project

EPA is considering various options to facilitate accelerated removal of lead based paints from households. EPA plans to clarify the regulations for disposal of the debris generated from renovations and remodeling -- to encourage homeowners and contractors to accelerate the *safe removal of lead from children's home environments*.

This activity is specifically aimed at protecting young children, because they are most at risk from lead based paint in households.

Funding: \$50,000 for FY1997

Educating Parents About Childhood Risks from Lead Exposure

New Program

EPA has launched a childhood lead poisoning outreach program directed at parents and other caregivers or service providers (e.g., pediatricians) of children under 6 years old in high risk target areas. This lead poisoning prevention and lead hazard awareness effort is one of the ways EPA is increasing lead-based paint hazard awareness and promoting lead poisoning prevention in high risk target populations such as young children.

Funding: \$592,000 for FY1997

Clean up of Lead-contaminated Waste Sites

Current Project

EPA is expanding its efforts to address exposure and cleanup of lead through such actions as conducting consistent, comprehensive assessments to evaluate the impact on children of lead-contaminated hazardous waste sites. EPA is working closely with the Agency for Toxic Substances and Disease Registry (ATSDR) and the Department of Housing and Urban Development to examine blood lead levels in children and to develop guidance on addressing the impacts of environmental lead on children in urban environments.

Funding: \$4 million in FY 1997

PROTECTING YOUNG CHILDREN FROM PESTICIDES

Improving Scientific Knowledge About Children's Exposure to Pesticides **Planned Project**

A 1993 National Academy of Sciences (NAS) report, *Pesticides in the Diets of Infants and Children*, concluded that then-current scientific and regulatory approaches did not adequately protect infants and children from pesticide residues in food. The Academy called on EPA to make significant changes in assessing exposure to pesticides, analyzing the potential for harmful or toxic effects, and using these data to characterize actual risks. Thus, the Academy report provided a major challenge to EPA to improve the safety of our food supply and provide greater assurance that our children are protected.

Building on the recommendations from the NAS report, the 1996 Food Quality Protection Act (FQPA) requires a reassessment of all existing pesticide tolerances (i.e., the amount of pesticide residue allowed by law in or on a harvested crop) to ensure that they meet the new safety standard, including the additional protections for children. EPA will review

more than 9,000 tolerances within the next ten years under this program. In August of 1997, EPA will publish a schedule for review of existing tolerances. Pesticide tolerances that pose the greatest risks, particularly to children, will be given the highest priority. In order to meet the new FQPA standard, EPA will make an explicit determination whether pesticide tolerances are safe for children, including consideration of aggregate risks from all sources of exposure and common mechanisms of toxicity.

Funding: \$9 million in FY1998

Science Issues Raised by the Food Quality Protection Act

New Project

EPA is developing science policies and procedures for incorporating the new child protective standards required by the 1996 Food Quality Protection Act (FQPA) into all pesticide risk assessments. Among other provisions, FQPA requires (1) use of an additional safety factor to account for potential pre- and post-natal toxicity and the completeness of data with respect to exposure and toxicity to infants and children; (2) consideration of *in utero* exposure to pesticide chemicals; (3) consideration of aggregate exposure to pesticides (i.e., from all sources including dietary, drinking water and residential); and (4) consideration of the available information concerning the cumulative effects of pesticide residues and other substances with a common mechanism of toxicity.

As these policies are refined, through the EPA's Scientific Advisory Panel and other peer review processes, they will be incorporated into the Agency's pesticide risk assessments to ensure the adequate protection of infants and children.

Funding: \$2.215 million in FY1997

Improvement in the Food Consumption Survey

Current Program

Reliable and comprehensive food consumption data is essential for accurate risk assessments, particularly when assessing risks to children, who often have very different food consumption patterns than adults. *Because children eat proportionately more food and drink more fluids than adults do, they are more exposed to environmental threats.* Children eat far larger amounts of certain foods for their body weight than adults and thus may ingest more pollutants per pound of body weight.

These differing consumption patterns can be easily underestimated in an incomplete survey. EPA uses the United States Department of Agriculture's (USDA) Continuing Surveys of Food Intakes by Individuals and the Department of Health and Human Service's National Health and Nutrition Examination Survey to estimate food consumption for the U.S. population, including children.

EPA is working with USDA to identify weaknesses in the existing data and recommend improvements, including increases in the sampling size for the infant and children population subgroups to better assess children's food consumption patterns.

Funding: \$75,000 for FY1997 (in addition to the base costs for running the program, which are found in USDA's budget). **Children Impacted:** 51 million

Improvements in the Dietary Risk Evaluation System**New Program**

In response to the 1993 National Academy of Sciences Report *Pesticides in the Diets of Infants and Children*, EPA initiated efforts to improve its Dietary Risk Evaluation System that will help to provide more realistic pesticide exposure and risk estimates for children. These efforts also respond directly to new requirements in the 1996 Food Quality Protection Act to improve protections for infants and children.

Funding: \$200,000 FY1997

National Pesticide Residue Database**Planned Project**

Children in their earliest years consume much more of certain foods per pound of body weight than adults do, particularly fruits, vegetables and juices, making them especially vulnerable to pesticide residues. To address this and other issues raised in the *Pesticides in the Diets of Infants and Children* report, an interagency working group composed of staff from EPA, FDA and USDA has developed a National Pesticide Residue Database. When implemented, such a database will enhance the Agency's risk assessment capability by improving the availability and quality of data on actual pesticide residues in food, including those foods commonly consumed by children.

Funding: \$0.5-1 million for start up and \$1 million annually

Outdoor Residential Exposure Task Force**New Program**

Young children are often disproportionately exposed to lawn chemicals because of their unique behavior, such as crawling in the grass. To assess those risks, EPA has organized an Outdoor Residential Exposure Task Force in order to develop data on exposure on pesticides that have been applied to lawns.

Funding: \$75,000 in FY1997

NAFTA Research Program**New Program**

As part of the NAFTA research program, EPA will develop and implement an approach to examine the cumulative risks and adverse health effects in young children resulting from persistent exposures to pesticides. Research will be conducted in communities along the U.S.-Mexican border.

Funding: \$2.2 million for FY1997

SETTING STRONG STANDARDS AND TAKING TOUGH ACTIONS TO PROTECT CHILDREN'S HEALTH**Protecting Young Children Living Near Toxic Waste Sites****Current Project**

EPA has been extensively involved in a project with the Agency For Toxic Substances and Disease Registry (ATSDR) called the "ATSDR Child Health Initiative." The Child Health Initiative is a program which places special emphasis on identifying and preventing the adverse effects of chemical hazards at toxic waste sites, on the health of the fetus, infants, children and youth.

This program was specifically launched to address the unique vulnerabilities of children who are often placed at greater risk of adverse health effects when exposed to toxic substances. These unique characteristics have been confirmed by more than ten years of public health assessments, toxicological investigations, and epidemiologic studies. As a result of this work, when two recent ATSDR health advisories led to relocation of hundreds of families and children from homes contaminated with mercury or with highly toxic pesticide, special attention was focused on the children's medical and psychological status. EPA participates on an ATSDR expert panel which makes recommendations to the ATSDR Board of Science Councilors on ATSDR efforts to protect children at Superfund toxic waste sites.

Funding: Approximately \$60-80 million for FY1997

Combustion Rulemaking to Reduce Dioxin, Furan, Lead and Mercury Pollution

Current Project

EPA's ongoing combustion rulemakings will help reduce the amount of lead, mercury, dioxin, and furan, and other heavy metals emitted into the air by hazardous waste combustion units in this country.

The goal of these regulations is to reduce, by 2005, emissions of dioxins and furans by 90%, particulate matter by 50%, and acid gases by 50% from levels emitted in 1994. Reductions in mercury are also expected to be significant. These pollutants are a particular danger to children, who may suffer from brain and central nervous system damage or adverse developmental effects such as delayed walking and talking, as a result of exposure. *For example, exposure to high doses of mercury during pregnancy and the first few months of life may pose particular threats to a child's developing nervous system.*

EPA is also examining alternative technologies for the treatment and disposal of mercury-bearing wastes.

Funding: N/A

Protecting Young Children from toxic PCBs

Current Program

PCBs, or polychlorinated biphenyls, were banned by EPA in 1976 because they cause cancer; some 20 years later, however, this toxic chemical continues to persist in the environment, often in contaminated fish. *Children whose mothers have high levels of PCBs when pregnant may develop learning disabilities and experience delayed development.*

EPA continues the development and implementation of programs that will improve measures designed to ensure safe remediation and incineration of toxic PCBs. New rules to streamline procedures will speed the clean up of PCBs, providing a particular benefit to children, who are uniquely vulnerable.

Funding: \$500,000 for FY1997

Existing Chemicals**Planned Program**

EPA will develop and incorporate a child-centered focus for the review of existing chemicals. Testing actions will be developed to prioritize evaluation of chemicals with high exposure potential to children and chemicals suspected of being health hazards to children.

Funding: \$100,000 for FY1997

New Chemical Assessment**Planned Program**

EPA will develop an individual assessment component for new chemicals that may be used in products designed for use by or for children. By FY1998, EPA will have methodologies and criteria in place to assure that new chemicals entering the marketplace have been assessed for exposures related to children, and standards set through the new chemical process to assure that exposures are acceptable. Among uses of particular concern could include paints, fabrics, dyes and plastics.

COMMUNITY RIGHT TO KNOW AND EDUCATION**Consumer Pesticide Right-to-Know Brochure****Planned Project**

In response to a 1996 Food Quality and Protection Act requirement, EPA is developing a brochure to provide to large retail grocers that discusses the risks and benefits of pesticides and provides information to consumers on how they can reduce their exposure to pesticide residues on food. This brochure, which will be published annually, will allow parents to make more informed choices for their families.

Funding: \$100,000 in FY1997

Consumer Labeling Initiative**Current Project**

In 1995, EPA began its Consumer Labeling Initiative to develop labeling for products that is clear, easy to understand, and that allows parents to make more informed choices for their families. EPA has initiated a pilot project focused on indoor insecticides, outdoor pesticides and household antimicrobial products, all products that may pose special risks for young children.

Funding: \$370,000 in FY 1997

Design for the Environment for Kids**Planned Program**

Building on its successful Design for the Environment (DfE) voluntary program, EPA is developing a *DfE for Kids* program for products used in the home or in schools. DfE is a voluntary program with individual industries, professional organizations, state and local governments, other federal agencies and the public to promote safer substitutes, technologies and chemical processes. A DfE program targeted specifically at products used in children's environments will help give families new ways to reduce their exposure to hazardous chemicals at home and in schools.

Funding: \$400,000 for FY1997

Poison Prevention**Current Project**

Accidental poisoning from pesticide chemicals around the household remains a serious threat to children. EPA is an active member of the Poison Prevention Council, and annually distributes thousands of fact sheets on pesticides and child safety to medical establishments and the general public. As a result of the Poison Prevention Week outreach in 1995, EPA was able to send a poison prevention message to 3.5 million television viewers.

Funding: \$5,000 for FY1997

Providing Parents with Easy to Access and Easy To Read Environmental Health Information**Current Program**

The EPA Home Page on the Internet provides information directly targeted to parents and teachers about how to protect children's health, including particular risks to young children such as lead poisoning, second hand smoke, asbestos, insect repellents and sun exposure.

EPA estimates that the number of hits to the specific *Protecting Children's Health* page has been increasing at a rate of four times the previous month's number. This page went live at the end of October 1996.

Funding: We have not targeted specific funding to this page -- it is part of the whole effort to redesign the Home Page to make the information more accessible to the public.

PROTECTING MOTHERS AND INFANTS FROM CONTAMINATED FISH AND POLLUTED WATERS

Polluted waters not only affect children when they swim in our lakes and streams, but also when they eat certain freshwater fish. Hundreds of beaches are closed each summer due to raw sewage and other contamination. All over America, warning signs are posted near thousands of rivers, lakes and streams, raising special concerns that pregnant women, children, and others with sensitive or compromised immune systems should not eat fish caught in the water because of contamination. During 1995, over 1,700 fish advisories were posted -- with 73 percent of them related to mercury contamination. *Exposure during pregnancy and the first few months of life to high doses of some chemicals commonly found in fish tissue, such as methyl mercury and PCBs, may pose particular threats to a child's developing nervous system.*

Providing Parents with more information about contaminated fish**Current Program**

EPA has developed a database of fish consumption advisories which is available to the public. The *National Listing of Fish and Wildlife Consumption Advisories* includes all available information describing state-issued fish consumption advisories in the United States. Included in the database is information on the geographic location of the advisory, species of fish concern, chemicals, and segments of the population that are affected.

EPA is working with state health and environmental departments to improve the effectiveness of fish consumption advisories. The Agency is incorporating new risk assessment methods into national guidance for states and tribes. EPA is also developing methods for assessing fish consumption rates in sensitive subpopulations, including children and women of childbearing age.

Funding: \$475,000 in FY1997

Great Lakes Water Quality Initiative

Current Program

EPA is also working to protect the nation's largest body of fresh water through the Great Lakes Water Quality Initiative. By addressing the long-term toxic pollution that persists in the Great Lakes, this initiative is protecting children and families by decreasing their exposure to toxic pollutants which can pose particular health effects on reproduction, and children's development and immune systems.

Beach Health Protection

New Program

Children's health may be at an increased risk from contaminated beaches. Various illnesses can result from the disease-causing pathogens in sewage-contaminated waters; these illnesses range from hepatitis, dysentery, and gastrointestinal illnesses to fever, ear infections, and other health problems. *Children tend to be at increased risk because of greater exposure due to longer periods of playing in the water and accidentally swallowing water, more extensive cuts and abrasions, and a still-developing immune system.*

EPA recently established a Beach Health Protection Program to improve the effectiveness of beach contamination advisories and monitoring. EPA will work with states, tribes, local agencies, environmental groups, and others to reduce microbiological contamination at the Nation's bathing beaches and other recreational water bodies. The 1997 program includes development of a prototype national database of beach health information, convening a national beach health conference to refine our strategic approach, and starting appropriate water quality models for microbiological contaminants.

The Program will also establish an information network to raise the awareness of local authorities, health professionals and the public of health risks and methods of prevention.

Funding and Impact: \$420,000 for FY1997.

PROTECTING INFANTS FROM MICROBIAL CONTAMINANTS IN DRINKING WATER

Drinking water contaminants pose a risk to children, *particularly to infants, who drink more fluids per pound of body weight -- and who may be more vulnerable to the effects of microbial contaminants such as Cryptosporidium, E. coli, Giardia or the Norwalk virus.* EPA estimates that last year, a total of 30 million Americans drank water from systems that violated one or more public health standards -- and roughly 13 million of them are served by systems that

do not filter their water and thus may not adequately protect against microbial contaminants.

In August 1996, Congress passed new drinking water legislation that provides strengthened protections to ensure that American families have clean, safe tap water -- improvements that the Clinton Administration has called for since 1993 -- including provisions to improve consumer information about local tap water. The new legislation gives Americans access to direct, simple information about local water quality, water sources, contaminants, and whether the water poses a risk to health.

To ensure that drinking water is safe for children, our immediate priority is control of microbial contaminants. EPA has taken a number of steps to reduce health risks from microbial contaminants in drinking water systems across the nation:

- EPA has targeted safety standard development and available resources to focus on contaminants in drinking water that pose the greatest threats to public health, such as microbial contaminants, including Cryptosporidium, and on special populations such as the elderly, children, people with HIV/AIDS, and others who are most at risk from unsafe drinking water.
- EPA requires that large water systems test source water -- and, in some cases, treated water -- for Cryptosporidium. Data from this 18-month test, along with information collected by EPA, will help develop new safety rules and standards to protect further against Cryptosporidium and other microbial agents.
- EPA launched the *Partnership for Safe Water* with the nation's water suppliers in March 1995, to achieve voluntary improvements in surface water filtration plants around the nation. Filtration substantially reduces -- but may not entirely eliminate -- Cryptosporidium contamination.
- While EPA has historically developed shorter- and longer-term drinking water Health Advisory criteria for children to protect them against potential exposure to drinking water contaminants, limited information is available to characterize the actual risks to infants and children. The 1997 program will assure that developmental studies are designed to evaluate lifetime effects on infants and children resulting from the short sporadic nature of exposures to contaminated drinking water supplies during their formative years.

Funding: \$300,000 for FY1997

Safe Drinking Water for Children living in Rural Areas

Planned Program

EPA is also working with the U.S. Department of Agriculture to bring clean running water to the children and families who live in the 1.4 million rural households across this country. With its *Water 2000* initiative, the Administration is working with states, companies, banks, and non-profit groups to bring safe, affordable drinking water to America's most remote and needy corners by the end of the century.

Funding: N/A

APPLYING THE BEST SCIENCE TO PROTECT OUR CHILDREN'S FUTURE

EPA has worked conscientiously to ensure the sound scientific underpinnings for its policy initiatives on protecting children. It is critical that the best science be applied to these problems; that the research be of the absolute highest integrity and caliber; that it be on the cutting-edge in sophistication, allowing for consideration of the special mechanisms that affect children as well as differences in susceptibility, and not just whether an effect to the general population occurs; and that it focus on the issues of greatest risk and concern.

Focusing Toxic Air Pollution Research

Current Program

Asthma -- both individual episodes and asthma-related deaths -- is increasingly on the rise in children. Ongoing research is expected to provide new information critical to understanding how air pollution affects the development of asthma in children. A special effort funds research that focuses on the link between health effects and exposure to toxic urban air pollution. EPA is funding studies that identify whether special subpopulations -- such as infants and children -- are at increased health risks due to higher exposure to toxic urban air pollution, or due to their inherent biological sensitivities. These community-based efforts will examine the pattern, frequency, and magnitude of exposures in specific communities.

- **Proposed New Air Quality Standards:** EPA's research into the health effects of particulate matter (soot) and ozone (smog) led EPA to propose strengthening air quality standards for these air pollutants last year. These proposals are designed to protect children, including children with respiratory diseases such as asthma.
- **Improving Scientific Knowledge About Fine Particle Air Pollution**
In FY97, EPA will significantly expand its research program on particulate matter air pollution. Research will be conducted to identify the way in which particles affect human health, the critical exposure concentrations, and the sizes, chemical compositions, and sources of particles responsible for health effects. Research also will begin to investigate technologies and control practices to reduce fine particles. In addition to research at our laboratories, EPA has awarded a grant to the Harvard University School of Public Health to examine fine particles in urban air and the respiratory health of children.

Funding: \$43.9 million for FY1997

Researching Potential Effects on the Endocrine System from Pesticides and Industrial Chemicals

Current Program

In recent years, increasing scientific and public attention has been focused on the potential effects of synthetic chemicals on the hormone system. These chemicals -- which have been labeled "endocrine disruptors" -- may pose a major health concern for children.

Although there is considerable scientific uncertainty, it is clear that a number of chemicals, including organochlorine pesticides such as DDT and chemicals such as PCBs, can cause endocrine disruption in wildlife and laboratory animals. Because very low levels of chemicals that block or mimic reproductive and thyroid hormones can determine the course of prenatal development, concern exists about the potential for birth defects and *alterations of normal growth and development in children*. Endocrine disruptors may also play an important role in reproductive cancers.

In recent years, increasing scientific and public attention has been focused on the potential effects of synthetic chemicals on the hormone system. EPA has acted to ban many of these chemicals -- including DDT and PCBs -- and has already taken many steps to regulate over 95% of known sources of dioxin in the U.S.

In addition, EPA is involved in important research to try to better understand increasing reports of reproductive, developmental and other problems linked to some chemicals, including processes that may uniquely affect children. EPA has developed a national research strategy -- with the involvement of stakeholders -- for areas like pesticide and chemical screening, testing and controls. EPA's research in FY97 is focusing on important questions to determine: classes of chemicals that may affect the endocrine system; how much exposure produces adverse effects; how humans and wildlife are exposed; effects actually occurring in humans and wildlife; and the combined effects of exposure to multiple endocrine disruptor chemicals.

Funding: \$10.8 million for FY1997

Improved Risk Assessment Procedures For Pesticides

Planned Project

EPA is developing a new application of an existing technique for probable risk assessments, known as the Monte-Carlo method, to better reflect actual exposure to pesticide chemical residues. Application of this technique will greatly enhance our ability *to identify and mitigate pesticide risks to young children*. The development of this new procedure will require improvements in food consumption data and upgrading of computer systems.

Funding: \$500,000 in FY1997

Exposure Research

In addition to looking at health effects, EPA is studying how children are exposed to pesticides and other toxic chemicals. If we understood more about which pathways of exposure are of greatest concern, we could better focus preventive measures.

- EPA's strategy for studying residential exposure to chemicals focuses on children as the most highly exposed subpopulation of concern. The residential exposure research program is an intensive study of how residues of pesticides and other chemicals are transported into and around homes, how residues are transferred/dislodged from surfaces in and around the home onto children, an assessment of the behaviors of children (such as crawling on the floor or putting their hands in their mouth) that bring them into contact with these residues, and what happens to the residues after they are transferred to the children's skin.

- The National Human Exposure Assessment Survey is an important research tool. It is an ambitious study to evaluate total human exposure to multiple chemicals on a community and regional scale. In collaboration with the Minnesota Department of Health, EPA is carrying out a special children's exposure module of the survey. Children will be screened for exposure to certain pesticides, with a goal of identifying "highly exposed" children and their sources of exposure. Researchers will measure the levels of pesticides in the air children breathe; in food and drinking water; and in the soil and dust around their homes. If successful, this screening phase will be used to supplement other Survey studies.
- EPA also will develop models of children's total exposure to several major chemical classes. The research will consider key physiological and behavioral differences between adults and children. This effort will take advantage of data from other efforts in which children's exposure is being measured.

Funding: \$700,000 for FY1997

Cumulative Exposure Project

Current Project

The Cumulative Exposure Project is EPA's most comprehensive effort to develop estimates of pollutant exposures to children in the near term. The project uses existing data and methods to estimate exposures to the general population through air, food, and drinking water, and focuses on identifying pollutants and sources with the greatest impacts on children. Data from the Cumulative Exposure Project will be used to:

- Identify air toxics that pose the greatest public health threat to children nationally. The analysis of air toxics will provide estimates of the levels of toxics in the outdoor air where children live and will compare these levels to health benchmarks to identify locations of greatest concern.
- Address the differential exposure of young children to food and drinking water contaminants, by incorporating children's intake rates and body weights into the analysis. Levels of toxic pollutants in food and air will be compared with health benchmarks to identify exposures of greatest concern.

Impact and Funding: The expected result of the program is the generation of a wealth of data on young children's cumulative exposures to environmental pollutants. General release of the project's results will support efforts by citizens to understand and improve the environmental health of the children in their communities. Over the past three years, EPA has spent approximately \$1.5 million on the project. Funding in FY97 is \$400,000, with another \$400,000 anticipated in FY98.