FACT SHEET ON SECONDHAND SMOKE

James Repace, MSc., Physicist
Repace Associates, Inc., Secondhand Smoke Consultants
Bowie, Maryland 20720, U.S.A.

Ichiro Kawachi, PhD, Associate Professor
Department of Health and Social Behavior
Harvard School of Public Health, Boston, Massachusetts, U.S.A.

Stanton Glantz, PhD., Professor
Department of Cardiology, University of California, San Francisco
San Francisco, California, U.S.A.

Abstract

Breathing secondhand-smoke causes morbidity and mortality from cancer, heart disease, and respiratory disease, as well as acute sensory irritation. It causes the premature death of hundreds of thousands of nonsmokers worldwide. Smoke-free buildings are the only remedy. Secondhand smoke cannot be controlled by ventilation, air cleaning, or spatial separation of smokers from nonsmokers.

Introduction

Secondhand Smoke (SHS) is the toxic waste of tobacco combustion, emitted from the combination of tobacco smoke from the burning ends of cigarettes, pipes, and cigars, and exhaled smoke from smokers. The widespread practice of smoking in buildings exposes nonsmoking occupants to combustion by-products under conditions where airborne contaminant removal is slow and uncertain. Over the past two decades, medical science has shown that nonsmokers suffer many of the diseases of active smoking when they breathe SHS.

Throughout the developed world, nations engage in the practice of pollution control with the intention of protecting human health against the effects of harmful chemical contaminants in food, water, and air. Accordingly, recognized standards of acceptability for harmful contaminants in food, beverages, drinking water, outdoor air, and indoor air in industrial

workplaces has taken hold, and control measures appropriate for these different environments have evolved. However, in many workplaces, including offices, restaurants, and bars, SHS causes exposure to toxic chemicals not permitted in other environments.

This Fact Sheet explores SHS issues in the following areas: hazard, exposure, dose, dose-response, risk, and control.

Hazard: Epidemiological studies around the world have investigated whether passive smoking causes elevations in lung cancer, heart disease, and other diseases. These secondhand smoke epidemiological studies generally assess exposure using surrogate exposure variables such as spousal smoking. They also often suffer from the lack of a truly unexposed control group. These problems tend to obscure risks. Nevertheless, the epidemiological studies of passive smoking provide convincing evidence of the detection of an effect at environmental levels of exposure. The most powerful evidence of effect is the existence of dose-response relationships: of the 30 world studies of passive smoking & lung cancer extant in 1992, 14 reported a test for exposure-response, and 10 were statistically significant at the 95% confidence level ($p \leq 0.05$) [U.S.EPA, 1992]. The probability of ten or more studies reaching this level by chance alone is less than 1 in ten billion. This evidence was sufficient for the U.S. Environmental Protection Agency to conclude that SHS was a "known human carcinogen." By 1997, the number of published epidemiological studies of lung cancer and passive smoking had increased to 37; these studies, with supporting evidence of tobacco-specific carcinogens in the body fluid of passive smokers confirm the carcinogenicity of SHS (Hackshaw et al., 1997). Despite press reports to the contrary, the recent WHO study by Boffetta et al.(1997) is completely consistent with these earlier studies.

The body of evidence from spousal smoking studies suggests that the average excess risk of lung cancer from passive smoking is 24% (95% CI: 13% to 36%) [Hackshaw et al., 1997]. However, for nonsmokers exposed to the smoke of a pack of cigarettes per day or more, the risk increase can be considerably greater; the EPA summarized 12 studies that assessed this risk. For 9 studies in 5 countries, the risk in this category ranged from 57% to 220%; 3 other studies in 2 countries reported risks in the 10% to 20% range (U.S. EPA, 1992, Table 5-11). In the U.S. in 1980, the average smoker smoked 32 cigarettes per day (Repace and Lowrey, 1980). The large cohort study by Hirayama (1983) exemplifies the dose-response trend (Figure 1,

below). The lack of completely unexposed controls depresses odds ratios (Repace and Lowrey, 1985). Hirayama's controls may suffer less from this problem than studies in other cultures due to the traditional exclusion of Japanese women from non-domestic workplaces. A lung cancer observed in the 1980's and 1990's is generally the result of 20 to 40 years' SHS exposure, dating back to 1940's to the 1970's when few restrictions on smoking existed in most workplaces, homes, or restaurants.

*(Hirayama T., Proc. 5th World Conf. Smoking & Health (1983)*
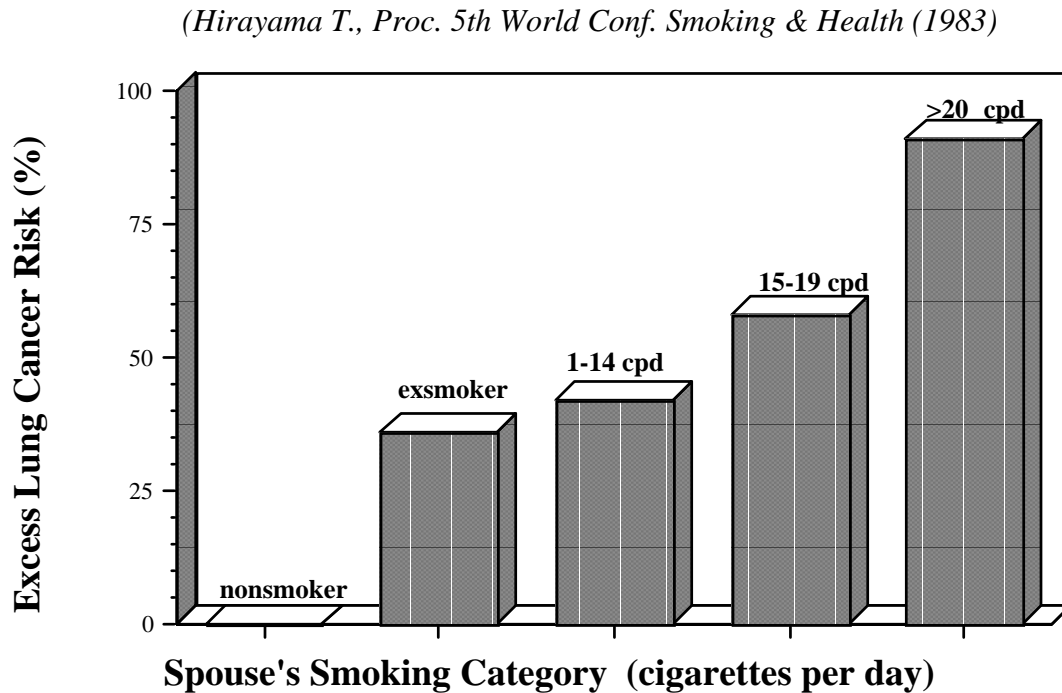
Figure 1. Dose-response in passive smoking (Hirayama, 1983).

Strong evidence of the hazard of secondhand smoke also comes from studies of smokers. In the United States and other developed countries, cigarette smoking causes most cases of lung cancer and chronic obstructive pulmonary disease, and a substantial fraction of coronary heart disease deaths (Thun et al., 1997). Smokers suffer increased rates of cancers of the lung, larynx, oral cavity esophagus, bladder, kidney, urinary tract, and pancreas (NCI Monograph 8, Preface, 1997).In the largest and most recent study of active smokers, the American Cancer Society's study of more than 1 million men and women, among active cigarette smokers, 52% of all male deaths and 43% of all female deaths are attributed to their smoking (Thun et al., 1997). Of those smokers who die from smoking, 55% die in middle age (i.e., from 35 yr. to 69 yr.). Of those who die in middle age, 22 years of life expectancy are lost, and of those who die in old age, 8 years of life are lost

(Peto, Lopez et al., 1994) **In other words, half of those who deliberately inhale cigarette smoke die from it. More than half of those die in the prime of life.**

Given the enormous toxicity of tobacco smoke, is it reasonable to presume that breathing any amount of secondhand smoke can possibly do no harm? Can it be presumed that lower levels of exposure, such as encountered in passive smoking are safe? The most heavily exposed passive smokers are active smokers who do not inhale. Figure 1 below shows the relative lung cancer risks (from right to left, respectively) for nonsmokers, smokers who do not inhale, and smokers who inhale (SG, 1979). Similarly, cigar smokers who do not inhale suffer major, statistically significant increases in cancer of the larynx (relative risk, 10.6), cancer of the lung (1.97), cancer of the pancreas (1.55), sites which are distal to the oral cavity and pharynx (6.98). Moreover, many of the compounds in tobacco smoke are known occupational carcinogens, such as arsenic (lung), benzene (blood), vinyl chloride (liver, brain), 2-napthalymine and 4-aminobiphenyl (bladder). In fact, in the United States, arsenic, benzene, and vinyl chloride are regulated hazardous air pollutants, and the latter two bladder carcinogens are banned in dye manufacture. This evidence is sufficient by itself to indict secondhand smoke as a hazardous substance to be avoided.

**Fig. 2. Lung Cancer Risks in Smokers by Inhalation**
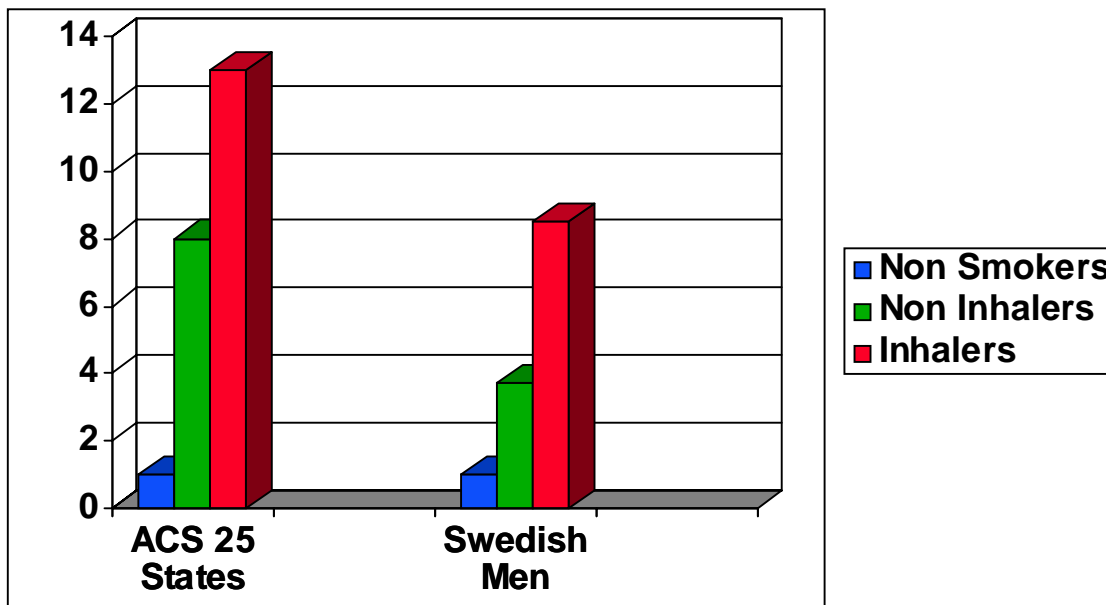
(U.S. Surgeon General, 1979, p. 5-15)

Secondhand Smoke and Cardiovascular Disease

Law et al. (1997) review the evidence from 19 published studies of passive smoking and heart disease; they report that the average excess risk of ischemic heart disease from passive smoking epidemiological studies is 23% (95% CI:14% to 33%), and conclude that platelet aggregation provides a plausible explanation for the mechanism and magnitude of the effect.

Kawachi, et al. (1997) in a prospective study of coronary heart disease (CHD) in 32,000 female U.S. nurses aged 31 to 61 yr., for nonsmoking women exposed only at work, observed a dose-response gradient for passive smoking and CHD. Adjusted relative risks of CHD were 1.00 [for no exposure], 1.58 *(95% CI, 0.93-2.68)* [occasional exposure], and 1.91 *(95% CI, 1.11-3.28)* [regular exposure]. Thus, regular exposure to SHS at work caused a 91% increase in CHD, shown in Figure 3 below.

[Kawachi, et al., Circulation 95 2374-2379 (1997)]
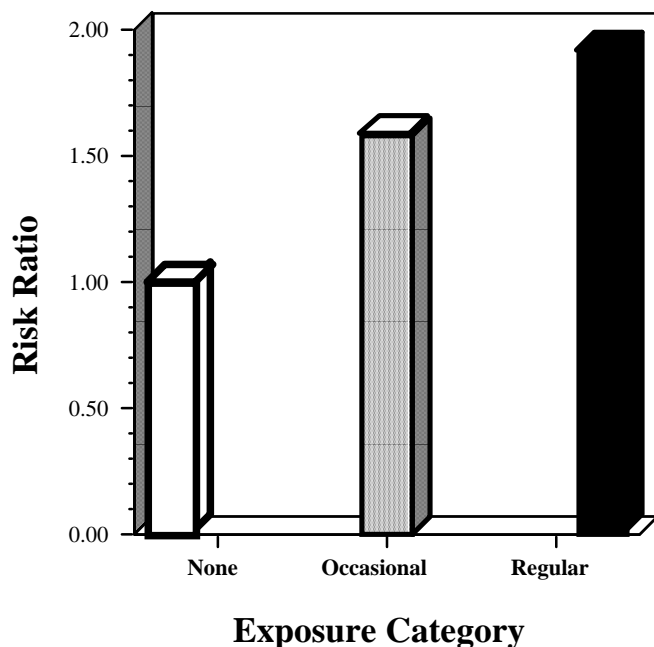
Figure 3. Risk ratio for CHD for nonsmoking nurses exposed only at work.

1.   No safe threshold has been established for cigarette smoking and risk of cardiovascular disease. Even smoking as few as 1-4 cigarettes per day is associated with a doubling in risk of coronary heart disease (CHD) (Kawachi et al., 1994).

2.   Many cardiotoxic compounds are more concentrated in sidestream smoke than in mainstream smoke. For example, carbon monoxide (which is known to aggravate angina symptoms) is 8-11 times more concentrated in sidestream smoke than mainstream smoke.(U.S. EPA, 1992)

3.   At least seventeen epidemiological studies have been published on the relationship of passive smoking and risk of CHD. A meta-analysis of 19 studies (including three unpublished reports) found a summary relative risk of CHD from exposure to *spousal* ETS of 1.30 (95% CI: 1.22 to 1.38, P < 0.001).(Law et al, 1997)

4.   A meta-analysis of eight epidemiological studies of *workplace* ETS exposure and CHD found a summary relative risk of 1.18 (95% CI: 1.04 to 1.34).(Glantz and Parmley, 1991; 1995; Wells, 1998)

5.   Several plausible mechanisms exist by which ETS exposure can increase the risk of CHD (Kawachi, 1998), including carboxyhemoglobinemia, increased platelet aggregability, increased fibrinogen levels, reduction in HDL-cholesterol, and direct toxic effects of compounds such as 1,3 butadiene (a vapor phase constituent of ETS which has been shown to accelerate atherosclerosis in animal models (Penn and Snyder, 1996).

6.   ETS exposure has also been linked to progression of atherosclerosis as measured by B-mode ultrasound of the carotid wall (Howard et al., 1994; Diez-Roux et al., 1995; Howard et al., 1998), as well as to early arterial damage as assessed by endothelium-dependent brachial artery dilatation (Celermajer et al., 1996).

7.   The death toll attributable to passive smoking from CHD is estimated to be 10 to 20 times as large as deaths from lung cancer (Wells, 1988, 1994; Glantz and Parmley, 1991; 1994; Steenland, 1992)

Tunstall-Pedoe et al. (1995), in a Scottish cross-sectional study of passive smoking and heart disease in 786 men and 1492 women, found that increasing quantitative measures of serum cotinine in ng/ml correlated to physician-diagnosed heart disease risk, with an odds ratio of 2.7 *(95% CI, 1.3-5.6)* for the highest vs. the lowest exposure quartile, adjusted for age,

housing tenure, total cholesterol, and blood pressure. This is illustrated below in Figure 4.

Figure 4. Dose-response for heart disease and SHS

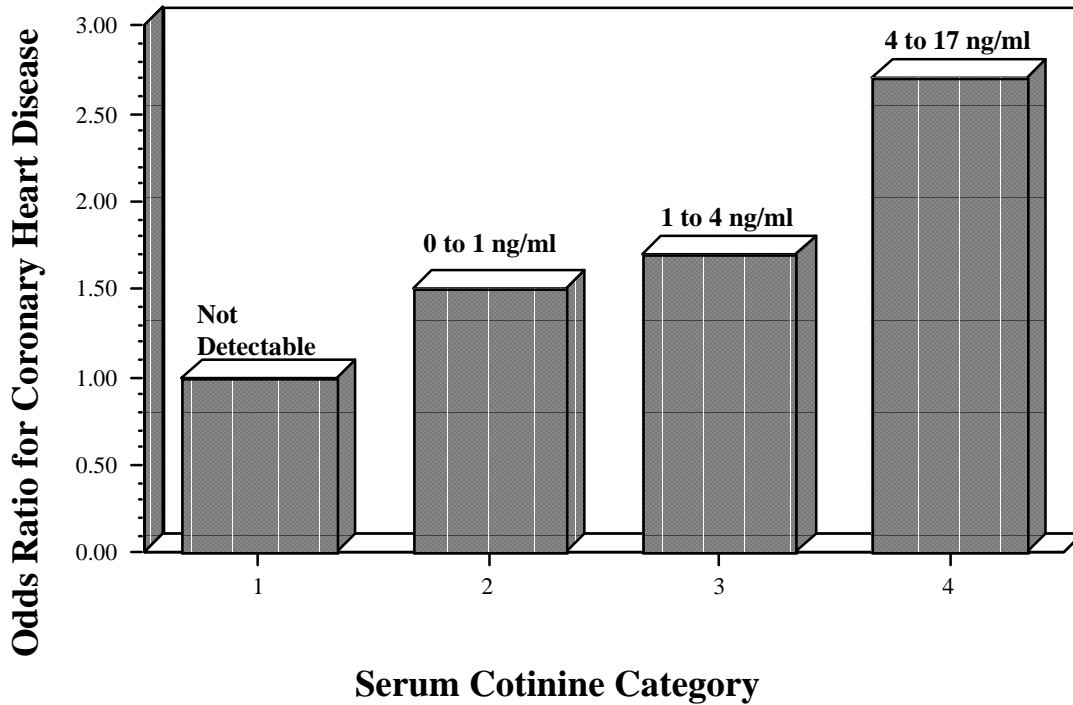*(Tunstall-Pedoe, et al., J Epidemiol and Comm Health 49: 139-143 , 1995)*

Figure 4. Risk of physician-diagnosed coronary heart disease in nonsmokers as a function of the level of the nicotine metabolite, cotinine in blood serum, in units of nanograms per milliliter (ng/ml). This is further powerful evidence that SHS exposures are not "low" as the tobacco industry asserts.

Figure 4A below shows the strong dose-response between tobacco smoke exposure and risk of acute stroke in New Zealand (Bonita, et al., 1999).

Passive Smoking as well as Active Smoking increases the risk of acute stroke
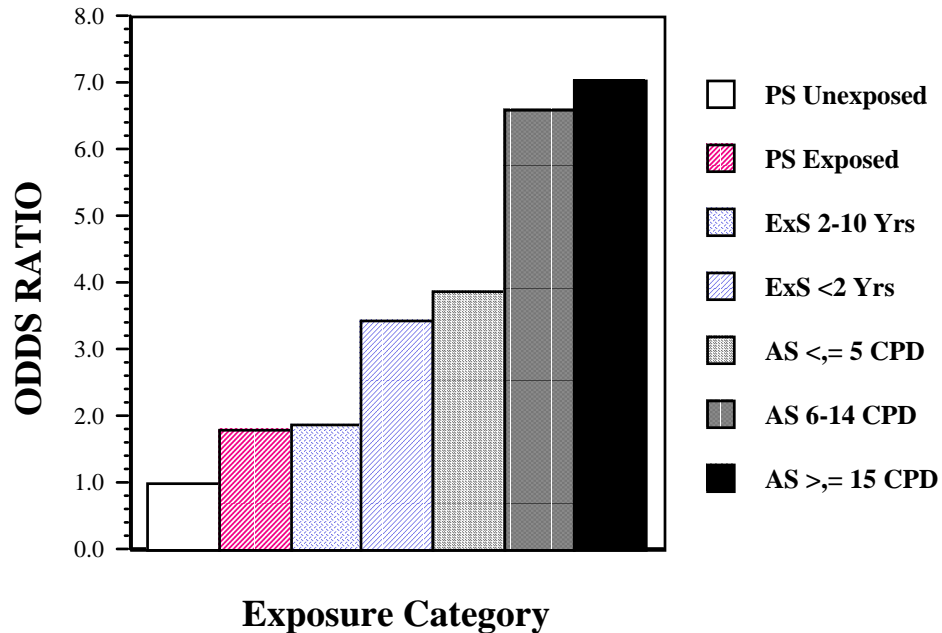
Bonita, et al., *Tobacco Control* 8:156-160 (1999)

- **MAJOR FINDING: 82% INCREASED RISK OF STROKE ASSOCIATED WITH PASSIVE SMOKING IN BOTH MEN AND WOMEN**

- **STUDY CONFIRMS RISK OF STROKE IN MEN AND WOMEN ACTIVE SMOKERS**

- **STUDY SHOWS EX-SMOKERS HAVE RISKS INTERMEDIATE BETWEEN ACTIVE AND PASSIVE SMOKERS**

- **STUDY DEMONSTRATES RISK OF STROKE WITH ACTIVE SMOKING IS EVEN HIGHER WHEN REFERENCE GROUP EXCLUDES ETS EXPOSED NONSMOKERS**

- **521 CASES, 1851 CONTROLS ; POPULATION-BASED NEW ZEALAND STUDY. ALL RESULTS STATISTICALLY SIGNIFICANT, STRONG DOSE-RESPONSE OBSERVED**

The most recent comprehensive report on secondhand smoke (referred in the report as environmental tobacco smoke or ETS) in the U.S. is that issued by the California Environmental Protection Agency in 1997. The California EPA report concluded that SHS was a cause of cancer, heart disease, and respiratory disease. The major conclusions of the CalEPA Report (Tables ES.1 and ES.2) are reproduced in Figures 5 - 7 below.

Figure 5.

1997 California EPA Report on ETS:
Health Effects of Exposure to Environmental Tobacco Smoke

HEALTH EFFECTS ASSOCIATED WITH EXPOSURE
TO ENVIRONMENTAL TOBACCO SMOKE

Effects Causally Associated with ETS Exposure

Carcinogenic Effects
Lung Cancer
Nasal Sinus Cancer

Cardiovascular Effects
Heart Disease Mortality
Acute and Chronic coronary heart disease morbidity

Table ES.1

   The most recent report on SHS from the UK, the SCOTH Report (1998), also concluded that passive smoking is a cause of lung cancer and childhood respiratory disease, and that passive smoking is a cause of ischaemic heart disease and cot death (SIDS), middle ear disease and asthmatic attacks in children. **The SCOTH report concludes that restrictions on smoking in public places and work places are necessary to protect non smokers (SCOTH, 1998).**

Figure 6.

1997 California EPA Report on ETS
Health Effects of Exposure to Environmental Tobacco Smoke

Effects Causally Associated with ETS Exposure

Developmental Effects
**Fetal Growth: Low birthweight or small for gestational age
Sudden Infant Death Syndrome**

Respiratory Effects

**Acute lower respiratory tract infections in children
(e.g., bronchitis and pneumonia)**

Asthma induction and exacerbation in children

Chronic respiratory symptoms in children

Eye and nasal irritation in children

Middle ear infections in children

Table ES.1

In addition to the above, Wells (1998) recently reported that there are now four studies each, which suggest an increased breast cancer risk from both passive smoking (combined OR for never-smokers: 1.71 (95% CI 1.30-2.25), and active smoking (combined OR 2.17 (95% CI 1.63-2.88), respectively.

Figure 7.

1997 California EPA Report on ETS:
Health Effects of Exposure to Environmental Tobacco Smoke

Effects with Suggestive Evidence of a Causal Association with ETS Exposure

Developmental Effects
Spontaneous abortion
Adverse impact on cognition and behavior

Respiratory Effects
Exacerbation of cystic fibrosis
Decreased pulmonary function

Carcinogenic Effects
Cervical cancer

Table ES.1

Table 1, adapted from the California EPA Report (CalEPA, 1997) gives the State's estimates of passive smoking-induced disease morbidity and mortality for the U.S.A., and per 10 million population, assuming that the nonsmoking population is similar in exposure and age to the U.S.A., for purposes of estimation in other countries.

Table 1: 1997 California EPA Report on Secondhand Smoke
Estimated Annual Morbidity and Mortality in Nonsmokers
Associated with SHS Exposure (Table ES.2, adapted)

Condition	Number of People or Cases	
	In the U.S.A.	per 10 million pop.
Developmental Effect Low Birthweight	**9,700 to 18,600 cases**	**360 to 690 cases**
Sudden Infant Death Syndrome (SIDS)	**1900 to 2700 deaths**	**70 to 100 deaths**
Respiratory Effects in Children Middle ear infection	**0.7 to 1.6 million physician office visits**	**26,000 to 59,000 physician office visits**
Asthma induction	**8,000 to 26,000 cases**	**300 to 960 cases**
Asthma Aggravation	**400,000 to 1,000,000 children**	**15,000 to 37,000 children**
Bronchitis or Pneumonia in infants and toddlers ($\leq$ 18 mos.)	**150,000 to 300,000 cases 7,500 to 15,000 hospitalizations 136 to 212 deaths**	**5500 to 11,000 cases 280 to 550 hospitalizations 5 to 8 deaths**
Cancer Lung	**3,000 deaths**	**100**
Nasal Sinus	**not available**	**not available**
Cardiovascular Ischemic Heart	**35,000 to 62,000**	**1,300 to 2,300**

Disease	**deaths**	**deaths**

Secondhand Smoke Exposure

SHS is a complex mixture of gas and particle-phase chemicals generated during the burning and smoking of tobacco products (CalEPA, 1997). Chemicals present in SHS include irritants and systemic toxicants such as hydrogen cyanide and sulfur dioxide, mutagens and carcinogens such as benzo(a)pyrene, formaldehyde and 4-aminobiphenyl, and reproductive toxicants such as nicotine, cadmium, and carbon monoxide (CalEPA, 1997). Many SHS constituents have been identified as hazardous by state, federal, and international agencies. To date, over 50 compounds in tobacco smoke have been identified as carcinogens and six as developmental or reproductive toxicants by the State of California. Table 2 shows 43 known or suspected carcinogens in tobacco smoke identified by the International Agency for Research on Cancer (IARC, 1987).

Table 2. 43 Chemical compounds identified in tobacco smoke for which there is "sufficient evidence" of carcinogenicity in humans or animals according to the International Agency for Research on Cancer (1986).

acetaldehyde	dibenzo(a,i)pyrene	N-nitrosdi-n-propylamine
acrylonitrile	dibenzo(a,e)pyrene	N-nitrosopyrrolidine
arsenic	dibenzo(a,l)pyrene	N-nitrosodi-n-butylamine
benz(a)anthracene	dibenzo(a,h)pyrene	*ortho*-toluidine
benzene	formaldehyde	styrene
benzo(a)pyrene	hydrazine	urethane
benzo(b)fluoranthene	indeno(1,2,3,-cd)pyrene	vinyl chloride
benzo(k)fluoranthene	lead	1,1-dimethylhydrazine
cadmium	nickel	2-nitropropane
chromium VI	N-nitrosodiethanolamine	2-napthylamine
DDT	N-nitrosodiethylamine	4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone [NNK]
dibenz(a,h)acridine	N'-nitrosodimethylamine	4-aminobiphenyl
dibenz(a,j)acridine	N'nitrosonornicotine	5-methychrysene
dibenz(a,h)anthracene	N-nitrosopiperidine	7H-dibenzo(c,g)carbazole

These include aromatic hydrocarbons, di- and polycyclic aromatic hydrocarbons, aldehydes, n-nitroso compounds, polycyclic aza-arenes, other nitrogen compounds, pesticides, halogenated compounds, and heavy metals, which include a variety of human or animal organ-specific carcinogens, e.g., arsenic, cadmium, chromium, nickel, and NNK (lung cancer), benzene (leukemia), formaldehyde and nickel (nasal sinus), 2-naphthylamine and 4-aminobiphenyl (bladder), lead (renal), vinyl chloride (liver).

Exposure assessment of SHS is critical in evaluating the magnitude of individual or group risk, and the effectiveness of strategies to reduce exposure. Exposure to SHS can be assessed through measurement of SHS markers such as respirable particles (RSP) or nicotine in indoor air, SHS biomarkers in saliva, urine, or blood, or through the use of models (Repace et al., 1998; CalEPA, 1997; Jaakkola and Jaakkola, 1997; Repace, 1987; Repace and Lowrey, 1993;). Often individuals are unaware of exposure, particularly outside the home (Cal EPA, 1997) making ascertainment of an adequate control group in epidemiological investigations difficult or impossible (Repace and Lowrey, 1985). Under typical conditions of smoking, building occupancy, and ventilation, indoor smoking produces levels of RSP far in excess of the U.S. federal fine particle standard for particulate matter 2.5 microns in diameter or below, 15 micrograms per cubic meter ($\mu g/m^3$) [See Appendix A.] This standard is designed to provide increased protection against a wide range of PM-related health effects, including premature mortality and increased hospital admissions and emergency room visits, primarily in the elderly and individuals with cardiopulmonary disease; increased respiratory symptoms and disease, in children and individuals with cardiopulmonary disease such as asthma; decreased lung function, particularly in children and individuals with asthma; and alterations in lung tissue and structure and in respiratory tract defense mechanisms.

To illustrate the effect of this air pollution on restaurant and bar workers, Eisner et al. (1998) studied the association between ETS exposure and respiratory symptoms in a cohort of 53 bartenders before and after California's prohibition on smoking in all bars and taverns. 74% of the bartenders initially reported respiratory symptoms; of those symptomatic at baseline, 59% no longer had symptoms at follow-up. 77% initially reported sensory irritation symptoms; at follow-up, 78% of these had symptom resolution. After ETS exposure completely ceased, objective measures of

pulmonary function showed marked a 5% to 7% improvement after only one month. Eisner et al. (1998) conclude that establishment of smoke-free bars and taverns was associated with improvement of respiratory health.

It is sometimes important to estimate SHS occupational hazards for specific groups of workers, such as flight attendants and casino workers, who have sued the tobacco industry for damages to their health. The risks of passive smoking can be estimated for groups of individuals based upon levels of the nicotine metabolite, cotinine, in blood, saliva, or urine (Repace and Lowrey, 1993; Repace et al., 1998) [Appendix B]. For example, Repace et al. (1998) estimated the risk of fatal lung cancer and heart disease from SHS exposure for office workers as a function of salivary cotinine; as illustrated in Figure 8 below. The horizontal axis shows the cotinine level. In a group of 89 office workers in the State of Rhode Island the median cotinine level was 0.5 ng/ml (Emmons et al., 1994). The vertical axis shows the estimated excess working lifetime exess risk (i.e., probability) of fatal lung cancer or fatal heart disease associated with that level.

U.S. federal occupational and environmental health regulatory decision rules for assessing the harm of environmental agents are shown for comparison. The *de minimis* risk level (1×10^{-6}) corresponds to an excess lifetime risk of fatality of one death per million persons at risk, and is considered "acceptable" from a regulatory standpoint. D*e manifestis* risks (3×10^{-4}) are those that are so high that U.S. federal regulatory agencies almost always act to reduce them (Travis et al.(1990). A very unsafe level is the "significant risk," level which is often used as a benchmark by the U.S. Occupational Safety and Health Administration (OSHA, 1994). Risks after control are either reduced to zero, or to below the de minimis risk level.
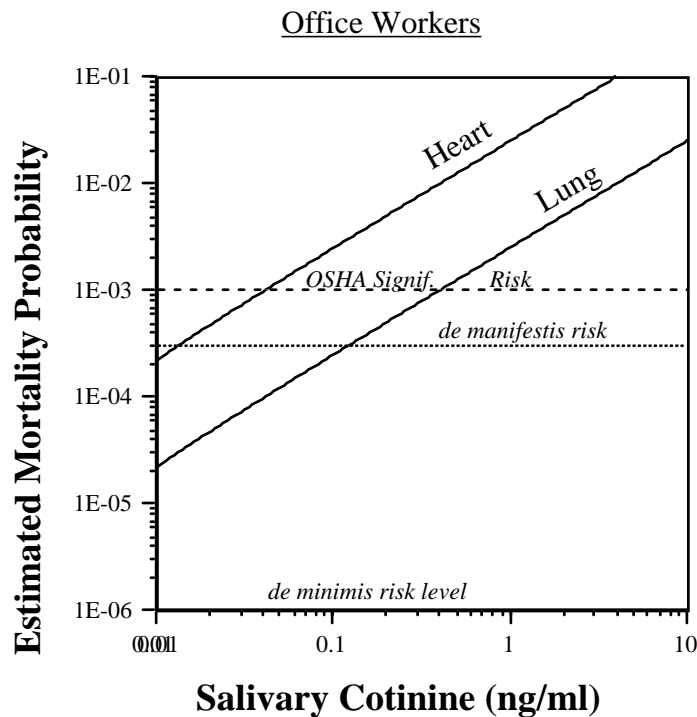
Office Workers

Figure 8.

Lifetime risk of mortality for a group of office workers in workplaces with unrestricted smoking estimated as a function of salivary cotinine. For heart disease and lung cancer mortality combined, more than 95% of U.S. nonsmoking office workers exposed under such conditions are estimated to exceed highly unsafe (i.e., *significant risk*) occupational regulatory levels. The *de manifestis* risk level is the obligatory regulatory level and the *de minimis* levels is the level of maximum acceptable risk (Repace et al., 1998).

Control: Why are ventilation, spatial separation, or air cleaning not valid control measures for SHS, as the tobacco and hospitality industries so often assert? Spatial separation of smokers from nonsmokers within a space does not affect either the smoker density nor the ventilation rate, and so cannot reduce the average SHS concentration. Insofar as ventilation or air cleaning, the U.S. OSHA(1994) has stated: "The carcinogenicity of [SHS] discounts the use of general ventilation as an engineering control for this contaminant." This is illustrated in Figure 9 below (Repace et al., 1998). To achieve de minimis risk would require in excess of one hundred thousand cubic feet per minute per occupant (50,000 litres per second per occupant), which would need tornado-like levels of air flow to achieve. Even if SHS is treated as as simple respirable particulate air pollution, for comparison with the U.S. National Ambient Air Quality Standard (NAAQS) for fine particles ($PM_{2.5}$), it is clear that ventilation designed essentially to limit carbon dioxide

from human metabolism is incapable of controlling massive clouds of tobacco tar particles (See Appendix A).
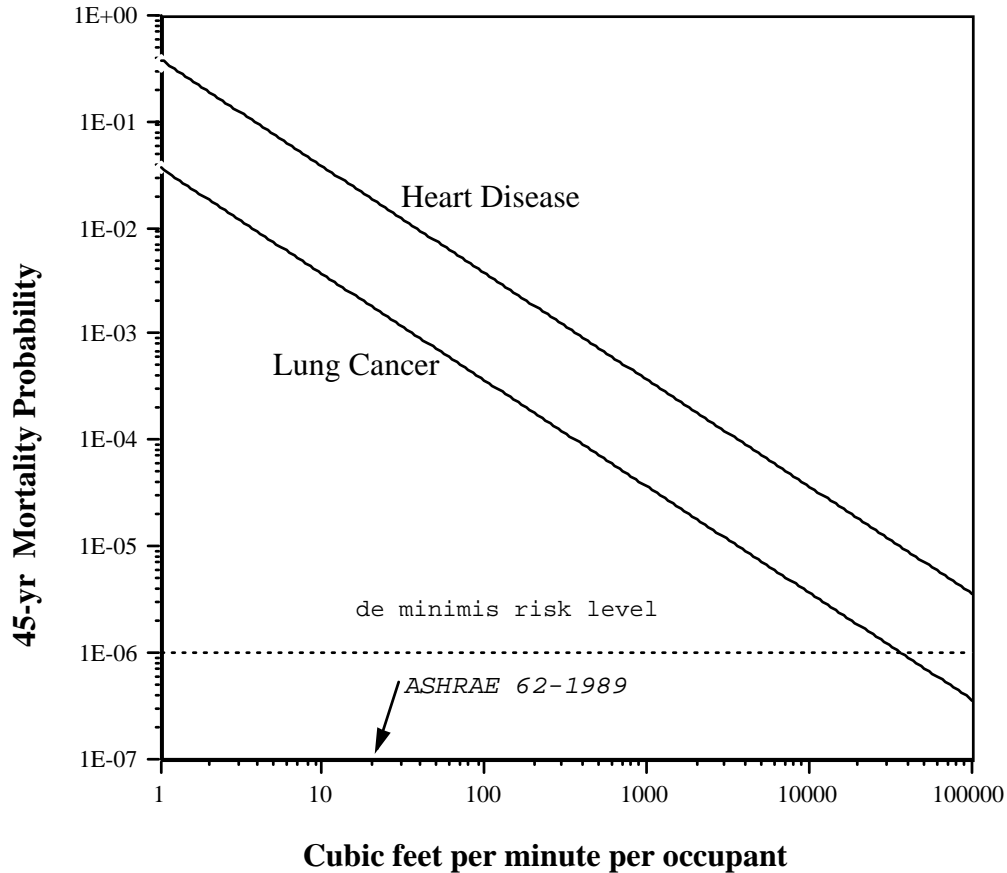

Figure 9. Ventilation cannot control SHS to an acceptable i.e., *de minimis*, risk level (Repace et al., 1998). Working lifetime risk for office workers exposed to unrestricted smoking (~2 smokers per 100 m^2 or 1000 ft^2 of office space) versus mechanical ventilation rate. A mechanical ventilation rate of 10 litres per second per occupant is equivalent to 20 cubic feet per minute per occupant (the ventilation rate recommended for offices and restaurants by the cognizant engineering authority in North America, The American Society for Heating, Refrigerating, and Ventilating Engineers (ASHRAE, 1989).

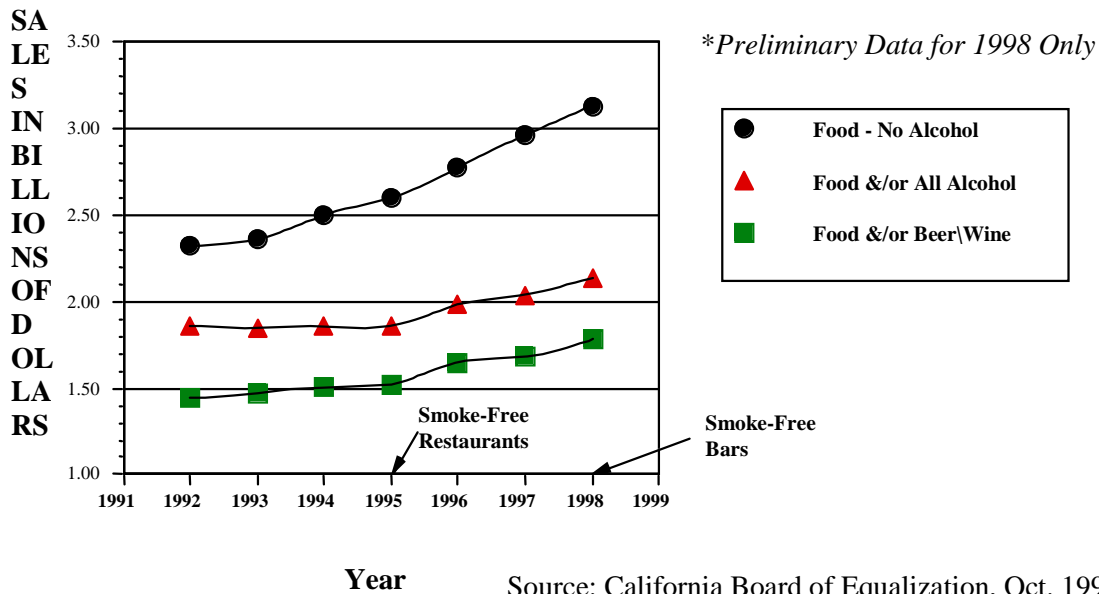# Smoke-free Restaurant Laws

Smoke-free restaurants and bars are universally mandated in the State of California, and they are working well and are spreading to other States and localities in the U.S.A. Studies in the U.S.A. on the economic impact of restrictions on smoking on restaurant sales for 81 localities in six states, 67 of which are 100% smokefree in restaurants, based upon objective sales tax

data, indicate that smokefree restaurant laws do not affect restaurant business (Glantz and Smith, 1994; 1997; Glantz, 1999).   Smoke-free workplaces are necessary primarily to protect the workers, and secondarily to protect the public from SHS. The tobacco industry has fomented fear of economic losses among restaurant owners, who often oppose restrictions on economic grounds, fearing a loss of business by smokers. However Biener and Fitzgerald (1999) have documented that many individuals avoid restaurants and bars because of SHS.

In a series of articles in the Journal of Public Health Management and Practice, the impact of smoke-free restaurant laws was examined in New York City and in the State of Massachusetts. These studies are providing convincing evidence of the feasibility, acceptability, and economic viability of smoke-free public places. For example, Bartosch and Pope (1999) compared local tax data before and after the imposition of smoke-free restaurant policies in Massachusetts, and found that there was little or no impact on the communities' restaurant industries.   Hyland et al.(1999) studied taxable sales for eating and drinking places and hotels in New York City before and after the imposition of restrictions on smoking in 1995; they found that sales increased after the smoke-free law was implemented, by 2.1% for eating and drinking places, and by 37% for hotels, compared with modest decreases in the rest of the State, which did not adopt such a law. The series of articles argues convincingly that smoke-free dining areas do not impose economic hardship on proprietors (Novick, 1999), as well as protecting worker and public health.

The tobacco industry has argued that the smoke-free restaurant law in California has resulted in a 30% decline in revenues for this industry. In science, a statement like this is called a lie. Data from the California food service industry are reproduced in Figure 10:

First Quarter Revenue Figures
California: 1992 - 1998*

Preliminary Data for 1998 Only

Legend:
- ● Food - No Alcohol
- ▲ Food &/or All Alcohol
- ■ Food &/or Beer\Wine

Y-axis: SALES IN BILLIONS OF DOLLARS

Smoke-Free Restaurants

Smoke-Free Bars

Year   Source: California Board of Equalization, Oct. 1998

SHS LESSONS FROM CALIFORNIA

• It is possible to reduce tobacco use, and thus nonsmokers' exposure to SHS rapidly through an aggressive anti-tobacco advertising campaign combined with community-based programs that stress changes in the social norms around tobacco, to create a smoke free society.

• A successful program is not simply directed at keeping children from smoking, but protecting nonsmokers from secondhand smoke and creating environments that facilitate smokers' decisions to cut down or quit.

• A successful campaign de-legitimizes both tobacco use and the tobacco industry. Industry de-normalization is the foundation upon which a successful campaign is based.

•   When the California program followed these principles, the rate of decline in tobacco consumption tripled and the rate of decline in smoking prevalence increased significantly. When the [Governor] Wilson Administration toned down and scaled back the program, including shifting the focus to children, the progress slowed or stopped.

•   The single most important target -- for both the tobacco industry and public health -- is young adults.

•   They are open to pro-health messages because they are having kids (and concerned about secondhand smoke) and going to work in smoke free environments.

The SHS issue is of vital importance to the tobacco industry: In 1978, before the first research linking SHS and lung cancer was published, a secret study (Roper, 1978) commissioned by the Tobacco Institute, the tobacco industry's lobbying and political arm in the United States, observed:

> **What the smoker does to himself may be his business, but what the smoker does to the non-smoker is quite a different matter. ...**
>
> ***This we see as the most dangerous development to viability of the tobacco industry that has yet occurred.***[7] [emphasis added]

Ten years later, in 1988, a secret marketing study conducted for Imperial Tobacco (1988) in Canada was even more explicit about the dangers of the passive smoking issue:

> **The shift to *social pressure* has also moved to high gear. Passive smoking has moved from a fringe issue, to by-laws, to the implementation of smoking restrictions in the work-place. *Smoking restrictions have moved from abstract discussion to practice.* This increasing social isolation of the smoker will not only increase his ill-ease with smoking, but will also have *a measurable effect on daily usage rates* resulting in overall industry losses.**[8] [emphasis in original].

The tobacco industry has tried very hard to obfuscate the facts presented in this report. It is hoped that this fact sheet will provide readers with the basic information needed to effect smoke-free workplaces in their countries.

Conclusions

- **There is an international scientific consensus that secondhand smoke kills**

- **Secondhand smoke under typical conditions of smoker density and ventilation poses unacceptable risks to nonsmokers**

- **Secondhand smoke cannot be controlled to acceptable levels of risk by ventilation or air cleaning**

- **There is no objective evidence to support the claim that smoke-free restaurants impose economic penalties on owners**

FACT SHEET ON SECONDHAND SMOKE

**Technical Appendices A and B**

Technical Appendix A.

Why Secondhand Smoke Cannot Be Controlled By Ventilation

This is illustrated by Figure A-1 below. The vertical axis refers to air pollution levels measured in restaurants, bars, and other establishments in the Washington DC metropolitan area; the horizontal axis refers to the smoker density. The dashed lines refer to the calculated air exchange rates, which span the range from 1/2 air change per hour in a naturally ventilated bingo game (data point T), to a maximally-ventilated cocktail lounge (data point F) at 7 air changes per hour. Generally, the best ventilated spaces have the highest smoker densities. The number of burning cigarettes per hundred cubic meters multiplied by 3 gives the estimated density of smokers (Repace and Lowrey, 1980, 1982). This means that 1 burning cigarette per hundred cubic meters is equivalent to 3 smokers per hundred cubic meters, assuming the smokers smoke at the U.S. average rate of 2 cigarettes per hour. 1/2 to 7 air changes per hour is the practical range of ventilation in most buildings. The figure illustrates that under all conditions of typical smoking and ventilation, the annual average level of the U.S. National Ambient Air Quality Standard (NAAQS) for fine particles (PM2.5), which defines clean air, is violated. The NAAQS is designed to protect against air-pollution-induced morbidity and mortality.

Modeling Nicotine Concentrations

The major reports on SHS have paid scant attention to the fact that SHS concentrations can be accurately calculated by means of mathematical models. With the ~40 $\mu g/m^3$ background subtracted, the above respirable particle concentrations can be used to estimate nicotine concentrations by dividing by 10 (Hammond, et al., 1987; Repace and Lowrey, 1993; Repace et al., 1998). Repace et al (1998) and Repace and Lowrey (1993) have shown the following expression describes the nicotine concentration as a function of the habitual smoker density and the air exchange rate. The habitual smoker density D_{hs} is three times the active smoker density (i.e., number of burning cigarettes averaged over the observation interval), and

assumes that the smokers smoke at the U.S. national average rate of 2 cigarettes per hour per smoker (Repace, 1987).

Repace and Lowrey (1980; 1982)
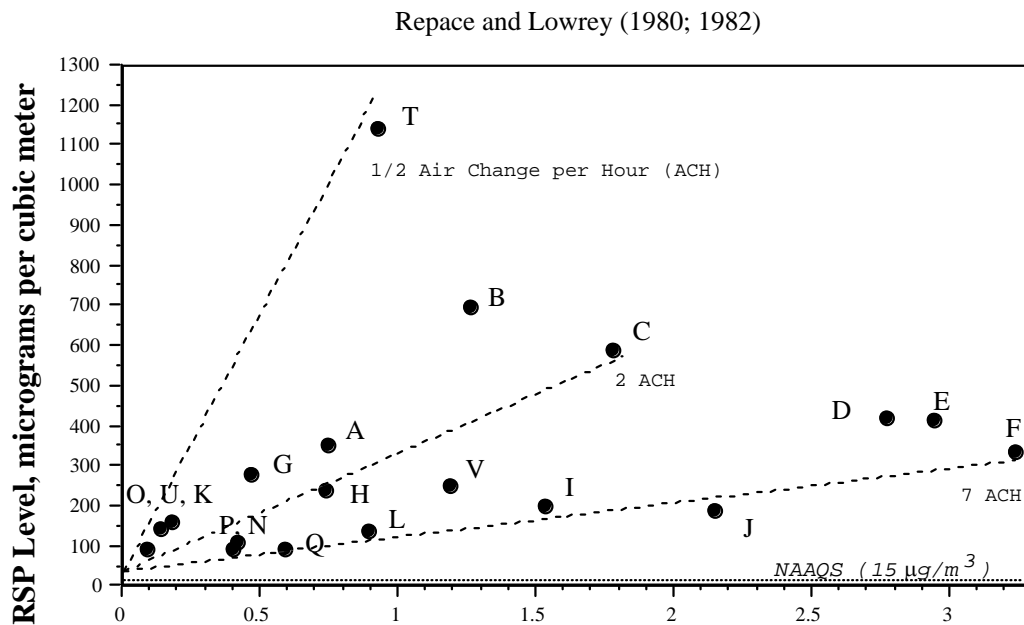
Figure A-1. **Smoking indoors leads to highly polluted air.** Repace and Lowrey (1980;1982) measured fine particle air pollution, i.e., particulate matter 3.5 microns in diameter or less ($PM_{3.5}$) in a variety of establishments (Repace, 1993). Data points E, H, K, L, and N are typical restaurants, B and V are reception halls, J is a hospital waiting room, I is a bowling alley, D, G, and T are bingo games, while O is a sports arena, C and Q are bars, F is a nightclub, U is a dinner theatre, and A is a private home during a party.) All of these establishments are in the Washington, DC metropolitan area. The dashed lines show the estimated air exchange rates. D_s, the number of burning cigarettes per hundred cubic meters, is equal to 1/3 the density of habitual smokers D_{hs}, so that a D_{hs} value of 3 is equal to a D_s value of 1. The U.S. Annual National Ambient Air Quality Standard (NAAQS) for Respirable Particulate Matter 2.5 microns or less ($PM_{2.5}$) is shown for comparison (15 µg/m^3). Thus, under typical conditions of smoking and ventilation, indoor air becomes massively polluted with fine particle air pollution, jeopardizing human health.

As an example of the use of mathematical models to calculate the nicotine and RSP concentration from secondhand smoke, consider a typical office workplace with D_{hs} = 0.71 habitual smokers per hundred cubic meters (This corresponds to a value of D_s = 0.24 in the figure above). Typical engineering practice recommends a ventilation rate equivalent to C_v = 0.84 air changes per hour (Repace et al., 1998) using the nicotine equation below

yields an estimated steady-state nicotine concentration of $N = 22 \, D_{hs}/C_v =$ (22)(0.71)/0.84 = 19 micrograms per cubic meter ($\mu g/m^3$). Assuming the workers are not present during lunch hour, and allowing for growth and decay of tobacco smoke reduces the time-averaged concentration for an 8 hour workday to 81% of steady state (Repace et al., 1998), or 15 $\mu g/m^3$. By comparison, Hammond et al. (observed an 8 hour time-weighted average nicotine concentration for 9 open office workplaces of 16 $\mu g/m^3$.

Nicotine Concentration Equation

(Repace et al., Risk Analysis 18: 71-83, 1998)

$$N = 22 \, D_{hs}/C_v$$

(where D_{hs} = smoker density in habitual smokers per 100 m^3, C_v = air exchange rate in air changes per hour, and N is the equilibrium nicotine concentration in $\mu g/m^3$)

As the nicotine concentration equation shows, the concentration will be high whenever the smoker density is high and the air exchange rate is low, irrespective of whether exposure occurs in homes, workplaces, or social settings, contrary to tobacco industry arguments, which assert that workplaces are much less polluted than homes. The relationship between exposure and dose is discussed below.

Technical Appendix B.

Dosimetry of Secondhand Smoke

The major reports on SHS have also paid little attention to the fact that SHS doses in blood, urine, and saliva can be accurately predicted using mathematical models. The model below shows the factors involved in determining dose of the nicotine metabolite cotinine in blood plasma (i.e., serum). What are the factors determining dose, and what do the clinical epidemiological studies of biomarkers show? What is the range of dose? What are the best methods of assessing dose?

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Steady-State Plasma Cotinine Model

[Repace and Lowrey, Risk Analysis 13: 463-475 (1993)]

$$P = (\phi\alpha\rho/\tau\delta)\ H\ N \text{ (ng/ml)}$$

ϕ = fraction of nicotine converted to cotinine (0.78)

α = fraction of nicotine absorbed (0.71)

ρ = respiration rate (1 m^3/hr)

δ

τ = # minutes/day (1440)

H = exposure duration (hr/day)

N = daily average nicotine concentration (μg/m^3)

    x 1000 ng/μg

</div>

The above equation shows that plasma cotinine is linear with nicotine concentration. While there may be individual metabolic variability (as there is for all drugs and chemicals) in large numbers of individuals, group doses will reflect group exposures (Benowitz, 1996).

As an example of the use of mathematical models to calculate body-fluid cotinine dose, Repace and Lowrey (1993) estimated that the average U.S. nonsmoker in the 1980's had an average daily nicotine dose from secondhand smoke of 143 μg, averaged over work and home exposure. Assuming a reasonable 7 hr daily exposure, and a respiration rate of 1 m^3/hour, this is equivalent to an estimated nicotine concentration of $N = 20$ $\mu g/m^3$. In the above plasma cotinine equation, $P = [(0.78) (0.71) (1) /(1440)(64)]\{7\}\{20\}\{1000\} = 0.84$ ng/ml. By comparison, the U.S. Centers for Disease Control conducted a national probability survey of serum cotinine in the late 1980's and early 1990's; for all nonsmokers with cotinines less than 20 ng/ml, the arithmetic mean serum cotinine was 0.54 ng/ml (D. Mannino, CDC, personal communication). The expected range of serum cotinine from passive smoking appears to be about 0 to 15 ng/ml in nonsmokers (Repace and Lowrey, 1993). Both gas chromatography and radioimmunoassay have been used in measuring body-fluid cotinine (Benowitz, 1996).

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