Title
A Killer on the Loose: An ASH special investigation into the threat of passive smoking to the U.K. workplace

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A KILLER ON THE LOOSE

An Action on Smoking and Health special investigation into the threat of passive smoking to the U.K. workforce

ash.

action on smoking and health

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www.repace.com

Action on Smoking and Health

ASH is a campaigning public health charity working for a comprehensive societal response to tobacco aimed at achieving a sharp reduction and eventual elimination of the health problems caused by tobacco. 120,000 people per year die from smoking-related diseases in the UK and tobacco is a major cause of illness and health inequalities. Tobacco is a powerfully addictive drug that most of its users would like to quit using.

ASH works by formulating the best information and analysis of the tobacco problem and credible responses, then communicates that to the public, opinion-formers and decision-makers in order to generate the public and political impetus for the measures that will reduce tobacco use and ultimately tackle the epidemic of disease and death that it causes.

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CONTENTS

1 EXECUTIVE SUMMARY

- An estimated 12,000 U.K. nonsmokers die annually from secondhand smoke (SHS) exposure at home, at work, and in social venues. In fact, SHS pollution now causes as many deaths annually as did the great London Smog 50 years ago and triple the annual number of road deaths from traffic accidents.

- Within the at-work category, data is sufficient to calculate risks for three subgroups: about 900 office workers, 165 bar workers, and 145 manufacturing workers are estimated to die from passive smoking each year in the U.K. That's more than three deaths a day in these three categories alone.*

- For manufacturing workers, three-fold as many are estimated to die from passive smoking than work-related deaths from all other causes. 17% of bar workers are estimated to die from passive smoking at current exposure levels. The SHS-caused deaths among office workers adds an estimated 9% to the total occupational mortality from all causes in all occupations.

- Recent U.S. and Canadian measurements show that during smoking, secondhand smoke accounts for about 90% of the fine-particle air pollution levels and 95% of the airborne carcinogens in hospitality venues.

- Under the hospitality-industry-sponsored *Public Places Charter on Smoking*, which promotes ventilation as a control for secondhand smoke, it is estimated that five of every 100 bar workers would die from workplace passive smoking, yielding 66 deaths per year.

- Engineering half-measures, proposed in the *Charter*, were evaluated by modelling and compared with air quality measurements in Canadian and U.S. venues. These methods clearly show that the Charter-specified air exchange rate would create an air pollution hazard, violating the daily U.K. air quality standard for particulate air pollution by three-fold.

- Attempts to control the toxic and carcinogenic properties of secondhand smoke by ventilation are futile, requiring tornado-strength rates of air flow.

- The intent of the Health and Safety at Work Act 1974, which places a general duty of care for employers to provide a safe working environment, is not being satisfied for passive smoking. Without an Approved Code of Practice (ACoP) or legislation to **ensure** smoke-free workplaces, nonsmoking workers will continue to die needlessly.

* Mortality from secondhand smoke in the U.K. nonsmoking population and in the three groups of workers has been estimated by several methods:
- from cotinine, a by-product of nicotine, in the body fluids of nonsmokers
- by extrapolation from U.S. estimates
- and from indoor air pollution exposure models.

2 INTRODUCTION

Exposure to tobacco smoke is widespread among nonsmokers, with many exposed unknowingly. It is a rare nonsmoker who does not carry around a measurable body burden of tobacco combustion products in his or her body fluids, as studies in both the U.K. and the U.S. show clearly. Tobacco smoke exposure in smokers causes 120,000 deaths per year in the U.K. Because smoking became widespread in society before its terrible hazards became understood, it has become widely tolerated and economically entrenched. It is a well-known sociological fact that familiar risks tend to be underestimated and discounted by people, while risks from unknown technologies are much more widely feared. However, while societies have come to expect standards of quality in the delivery of food, water, and air in the outdoors and in the industrial workplace, and for new industrial products, these expectations have spread more slowly to indoor air pollution in non-industrial workplaces.

And as the ranks of society's decision-makers have often included nicotine-addicted smokers, it has been difficult for the non-addicted population to restrict smoking to areas where toxic tobacco smoke will not harm them. Moreover, because the tobacco industry is willing to spend large sums of money to ensure that its products and their toxic by-products remain unregulated, governments at every level of society have massively failed to protect the population against either active or passive smoking. Nevertheless, as the tools of modern epidemiological, biophysical, and physical science have become applied to the problem of passive smoking, it has become obvious that secondhand smoke (SHS) creates quantifiable risks to both nonsmokers and smokers that are quite large compared to the risks encountered from any other environmental pollutant. The annual risks of death from passive smoking in the U.S. are more than 600 times greater than all of the federally-regulated hazardous outdoor air pollutants combined, and 38% larger than all deaths from motor vehicle accidents. In the U.K., the estimated number of annual deaths from passive smoking at about 12,000, is comparable to that of the great London smog of 50 years ago, greater than the 10,000 occupational deaths in the U.K. annually, and triple the 3,450 current annual number of road deaths from traffic accidents (Dept. for Transport, 2002; www.transtat.dft.gov.uk).

The estimates for individual worker populations likewise are significant relative to mortality from occupational hazards, with the 146 to 900 estimated passive smoking deaths per year among hospitality, office, and manufacturing workers ranging from three-fold to 19-fold the number of deaths from other occupational hazards among all manufacturing workers. The total for all three worker categories is about 1,200 deaths per year, or roughly 10% of the total from passive smoking.

As a wealth of scientific data has been amassed for air pollution control over the past 50 years resulting from notorious outdoor air pollution episodes, the outdoor air has gradually been brought under control. Workplace air pollution, particularly in the wake of the asbestos debacle, has a great deal of professional regulation. However, occupational and environmental health professionals, have generally ignored SHS as an air pollutant. Perhaps this is due to the inherent difficulties in measuring indoor air in non-industrial workplaces such as offices, bars, and restaurants, and because SHS is a pollutant generated by people, not by industrial processes in workplaces. Therefore the issue has largely remained in the province of public health officials, who have repeatedly called attention to the seriousness of this problem, while lacking regulatory authority. Into this vacuum, affected industries, afraid of real or imagined economic losses, have argued for engineering "solutions" such as ventilation or designated smoking areas. These "solutions" however, ignore the normal occupational or environmental health regulatory paradigms which involve rigorous identification of hazard, exposure, dose, dose-response, risk, and control to within an acceptable level of risk by established principles involved in regulating toxic substances. When such established principles are applied, it becomes obvious that the control measures advanced, for example by the UK hospitality industry's *Public Places Charter*, are seriously lacking in professionalism, and ignore the risks of SHS to workers and the public. It is clear that any engineering solution is doomed to failure because it would require tornado-like levels of ventilation (Figure 1) to satisfy air pollution and toxic substance standards (Repace and Lowrey, 1985b).
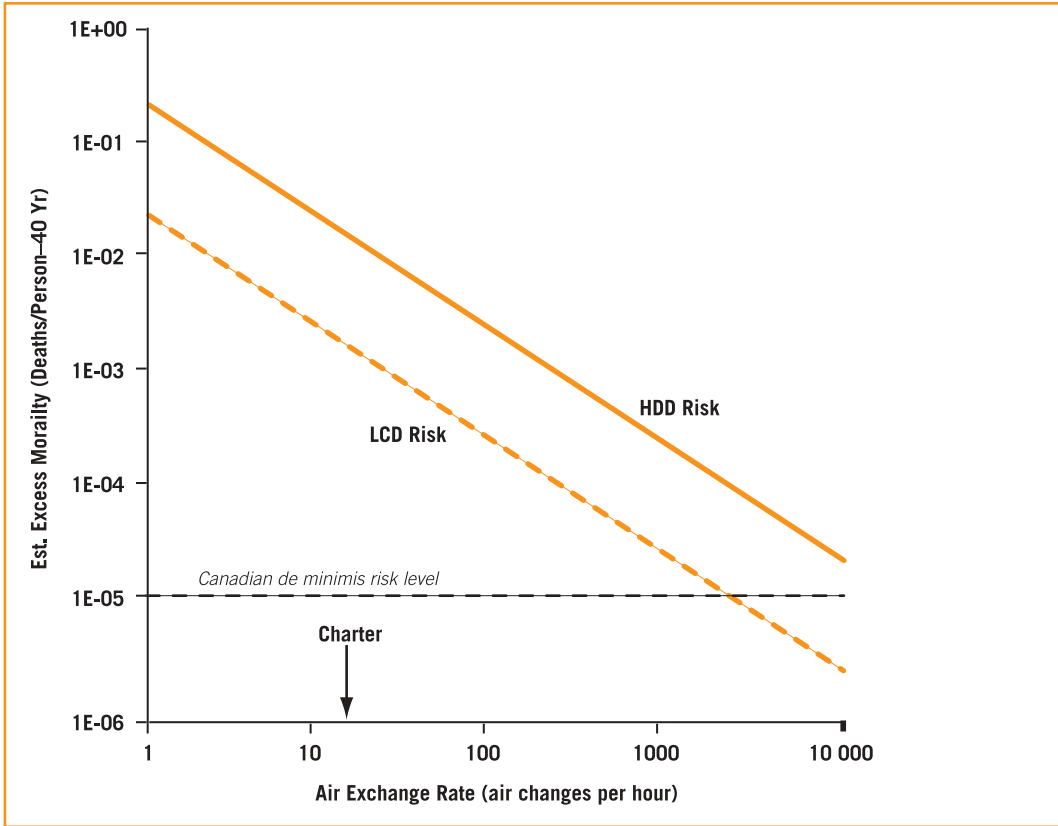
Figure 1 illustrates the vast increase in air exchange rate required to get acceptable risk at the small-population *de minimis* or "acceptable" risk level. With a population ten times that of Canada, the U.S. *de minimis* risk level is 1 death per million persons per lifetime, and is used by regulatory agencies to evaluate the risks of hazardous pollutants in air, water, or food. The *Public Places Charter*-specified ventilation rate of 12 air changes per hour is consistent with an unacceptable risk. To make it acceptable – in other words below the Canadian or U.S. *de minimis* risk level – ventilation rates would have to be increased more than 3,300-fold, to 40,000 air changes per hour.

This means that the only acceptable means of control of SHS is the banning of smoking in the workplace and in enclosed public spaces.

3 PASSIVE SMOKE: THE SCIENCE

3.1 Is the passive smoking risk under-estimated?

In 1998, the UK Scientific Committee on Tobacco and Health (SCOTH) summarised the dangers to non smokers from passive smoking. The SCOTH report concluded that SHS is a cause of lung cancer and ischaemic heart diseases, and that such exposure represents a substantial public health hazard, causing thousands of deaths in the UK annually. The Committee recommended that smoking should not be allowed in the workplace, and that smoking in public places be restricted on the grounds of public health. Some 27% of the U.K. population smokes. However, of the 27 million UK workers, only 11%, or three million workers, reported being exposed to SHS in 2002 (ASH, 2002). This number is likely an underestimate, as tobacco smoke pollution is recirculated by ventilation systems or diffusion to nonsmoking areas of buildings. For example, although the U.S. Centers for Disease Control measured the nicotine metabolite, cotinine, in the blood of 88% of the nonsmoking population, only 40% reported exposure, as shown in Figure 2. (Pirkle et al., 1996)

Estimates of the risk of SHS derived from epidemiological studies based on spousal smoking report about a 30% average increase in the risk of fatal heart disease and lung cancer (SCOTH, 1998; CALEPA, 1999). However, finding persons who have *truly* been unexposed to SHS all of their lives is difficult, because many people are unaware that they are being exposed, as figure 2 illustrates. This confounds epidemiological studies, which measure risk by comparing nonsmokers reporting exposure, shown in Zone C below, to nonsmokers reporting no exposure, shown in the palest orange below. However, many in Zone A actually have SHS exposures greater than those in the Zone C (Zone B overlap) causing studies of passive smoking to underestimate risk (Johnson and Repace, 2000).
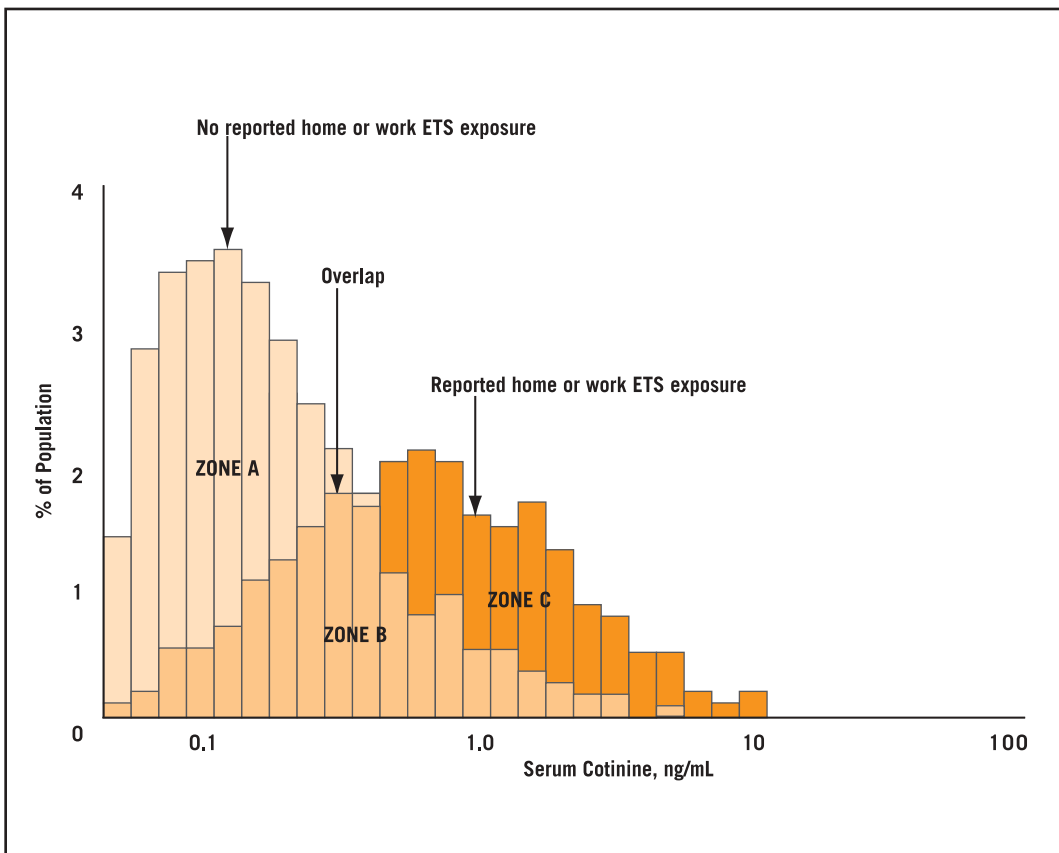

Figure 2
NHANES III Distribution of Cotinine in U.S. Population ≥ 4 years of age
Adapted from The National Health & Nutrition Examination Study III (NHANES III).

88% of U.S population is exposed to ETS, but only 40% report exposure.

3.2 The effect on coronary circulation

Breathing high SHS concentrations causes acute cardiovascular effects, depressing the ability of a nonsmoker's blood vessels to dilate, down to a smoker's impaired levels after only 30 minutes exposure. This is shown in Figure 3.

And who has such high exposures? Jarvis (2001) reports that London bar workers have SHS doses that are seven times greater than the average English nonsmoker; high SHS carbon monoxide levels are also found in Galway Pubs (Repace, 2002; Mulcahy and Repace, 2002).

3.3 Effects of Tobacco Smoke on Smokers

The results of the British Doctors Study by Doll, Peto, et al in 1994 are shown in figure 4. This study, and others, demonstrate that half of all smokers will die from smoking, one quarter in middle age (35-69), and one quarter in old age (Peto, Lopez, et al., 1994). In the UK in 1995, an estimated 120,000 people died from smoking, accounting for one fifth of all UK deaths (ASH, 2001). Each cigarette smoked causes a 13 minute loss of life expectancy. However, cigarettes, the most toxic of industrial products to which humans are routinely exposed, are alone in being exempt from regulation. This forms the basis for the problem of passive smoking.

In fact, secondhand tobacco smoke is so toxic that its effects can be observed even in smokers, as is illustrated by Figures 5a and 5b.
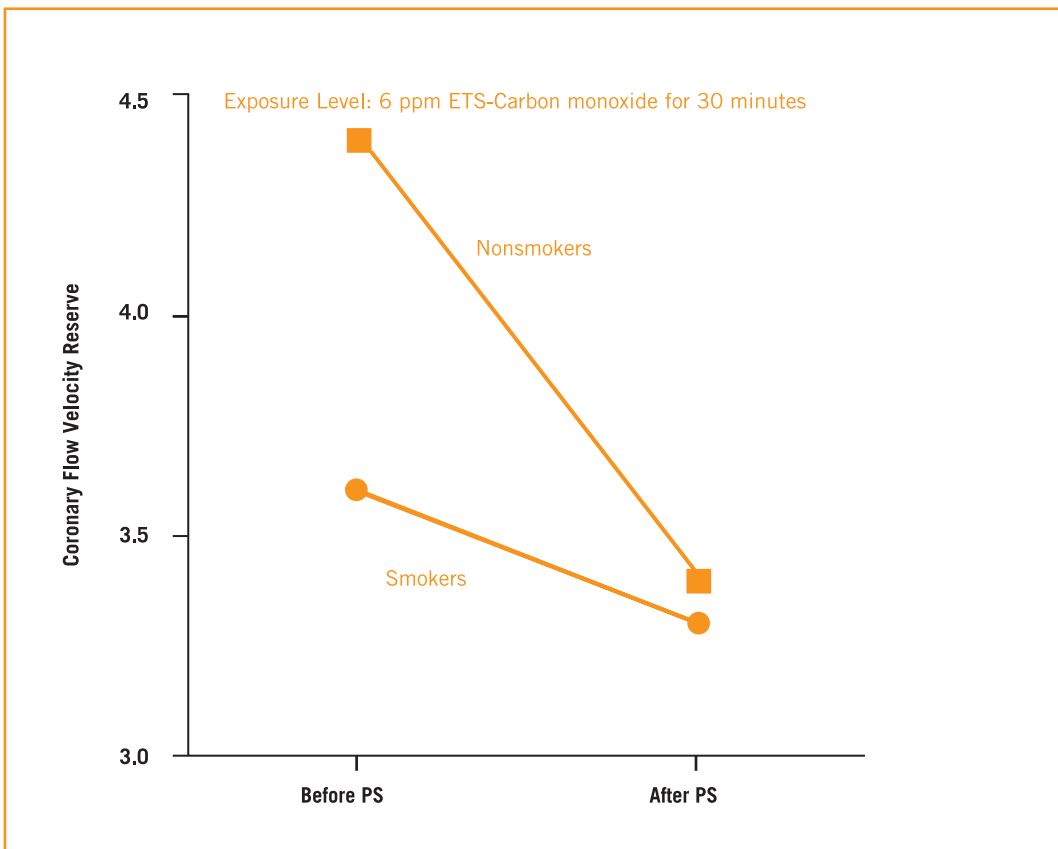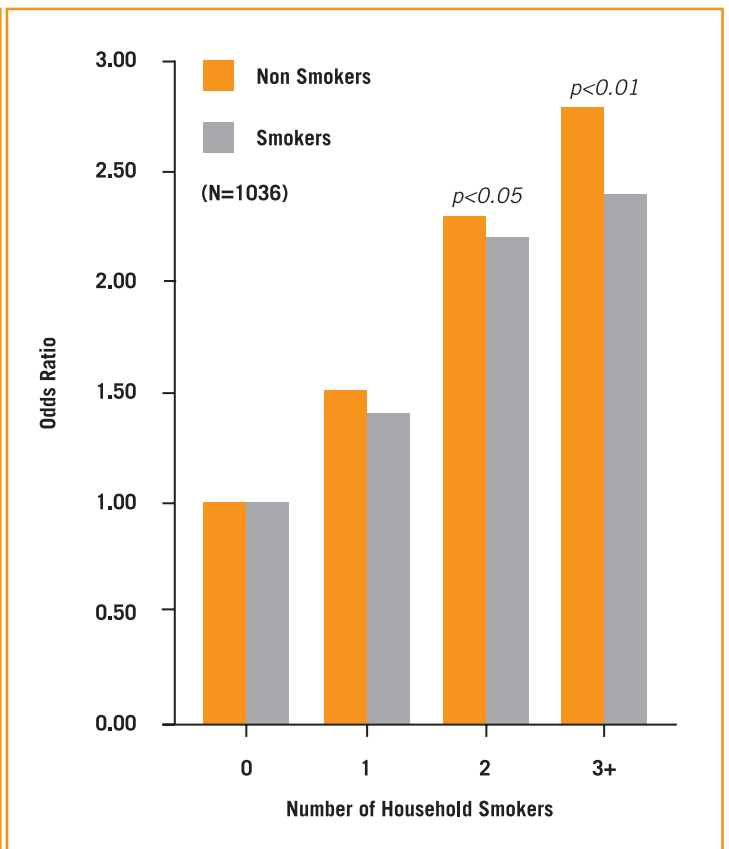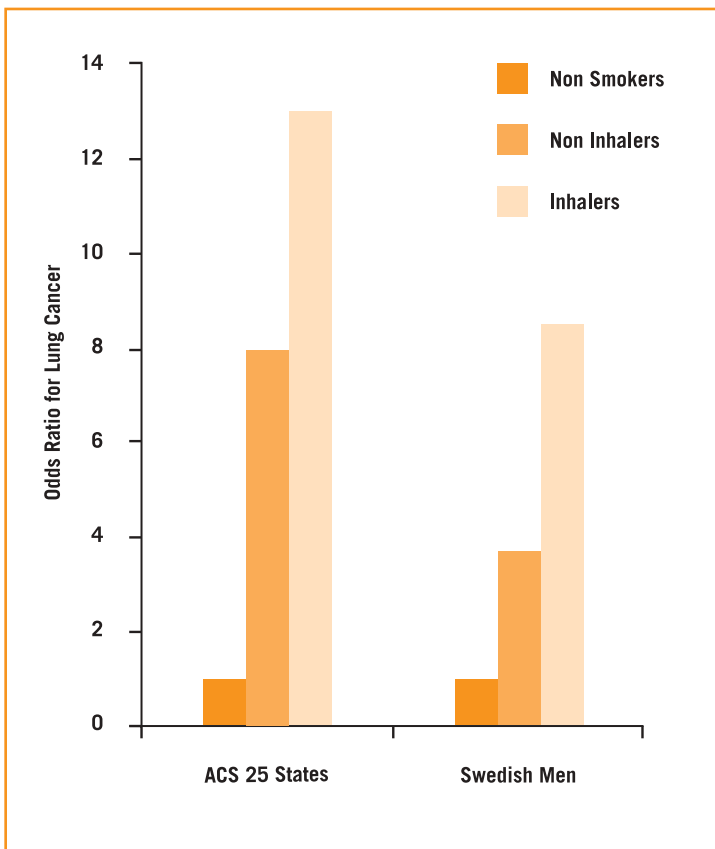

Figure 3

Acute Effects of Passive Smoking on Coronary Circulation In Healthy Young Adults
(Otsuka et al. JAMA 2001; 286436-411)

Coronary flow velocity reserve, the ability of the arteries supplying the heart to dilate and supply more blood flow in response to exertion, is impaired by passive smoking, placing a strain on the heart. Even short-term exposure to the levels of SHS (also known as ETS) commonly found in English and Irish pubs degrades nonsmokers' blood flow to the impaired level of smokers.

PASSIVE SMOKE: THE SCIENCE

Figure 4 (left)
U.K. male doctors study:
40 yrs, 1951-1991. Effects of cigarette smoking on survival
This 40 year-long study of survival as a function of age shows that no matter how long a person lives, tobacco smoke exposure causes a loss of life expectancy.

Figures 5a,b (below)
a SHS kills smokers as well as nonsmokers. Lung cancer risk to non-inhaling smokers is a major fraction of inhalers' risk.
(Surgeon General, 1979).

b Cancer risk in smokers and nonsmokers who reside with other smokers.
(Sandler D. et al, 1985).

Figure 4 legend:
- ● Never Smoked Regulary
- ▢ Current Smokers (1-14 cig/day)
- ▲ Current Smokers (15-24 cig/day)
- ★ Current Smokers (25+ cig/day)

No Evidence of a Threshold

3.4 Secondhand Smoke Toxicity

Societies regulate and control toxic chemicals in air, water, and food by virtue of the observed adverse health effects in humans and animals. The irony is that although many of the toxic chemicals in SHS are individually known and regulated industrial workplace carcinogens and toxins, indoor air pollution caused by SHS in workplaces has been rarely regulated, For example, from studies on industrial workers it is known that 4-aminobiphenyl causes bladder cancer; arsenic causes lung and lymphatic cancer; (NIOSH, 1994), benzene causes leukemia, benz(α)pyrene causes lung cancer; 1,3 butadiene causes cancer of the blood-forming organs, cadmium causes prostate, blood, and lung cancer; chromium VI causes lung cancer; formaldehyde causes nasal sinus cancer; ß-napthylamine causes bladder cancer; nickel causes lung and nasal cancer; [210]Polonium causes lung cancer; vinyl chloride causes liver cancer; and vinyl cyanide (acrylonitrile) causes brain tumours, as well as lung and bowel cancer. These and many other chemicals are found in SHS. There are at least 142 poisonous substances in tobacco smoke, including 6 substances that are U.S. Environmental Protection Agency (EPA)-regulated hazardous air pollutants, 68 that are known human or animal carcinogens, 47 that are EPA-listed as hazardous wastes, and the balance are various toxic chemicals.

3.5 The Scientific Consensus on SHS

There is an international consensus that secondhand smoke kills. It has been condemned as a health hazard by all U.S. environmental health, occupational health, and public health authorities, including the National Toxicology Program (2000), the National Cancer Institute (1993; 1995), Occupational Safety & Health Administration (1994), the Environmental Protection Agency (1992), the National Institute for Occupational Safety and Health (1990), the Surgeon General (1986), and the National Academy of Sciences(1986), as well as by the SCOTH Committee in the U.K. and the World Health Organisation.

Figure 6 illustrates the SHS lung cancer impact for 93,500 Japanese women as a function of their husbands' smoking rate.

Figure 7 shows the risk of coronary heart disease in Scottish nonsmokers as a function of the level of the nicotine metabolite, cotinine, in nonsmokers' blood from SHS exposure.

We also know that passive smoking, as well as active smoking, increases the risk of acute stroke

Figure 8 shows the strong dose-response between tobacco smoke exposure and risk of acute stroke in 2,400 New Zealand men and women (Bonita, et al., 1999).
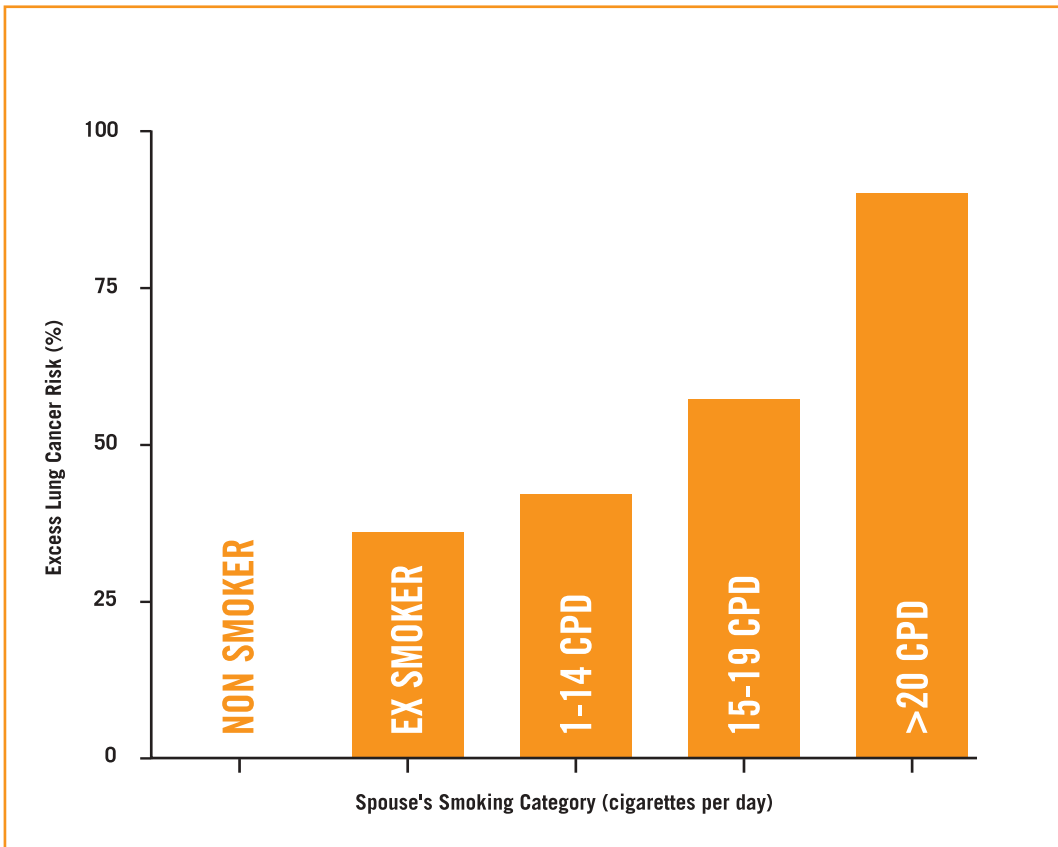

Figure 6
Exposure-response between lung cancer and spouse's smoking rate
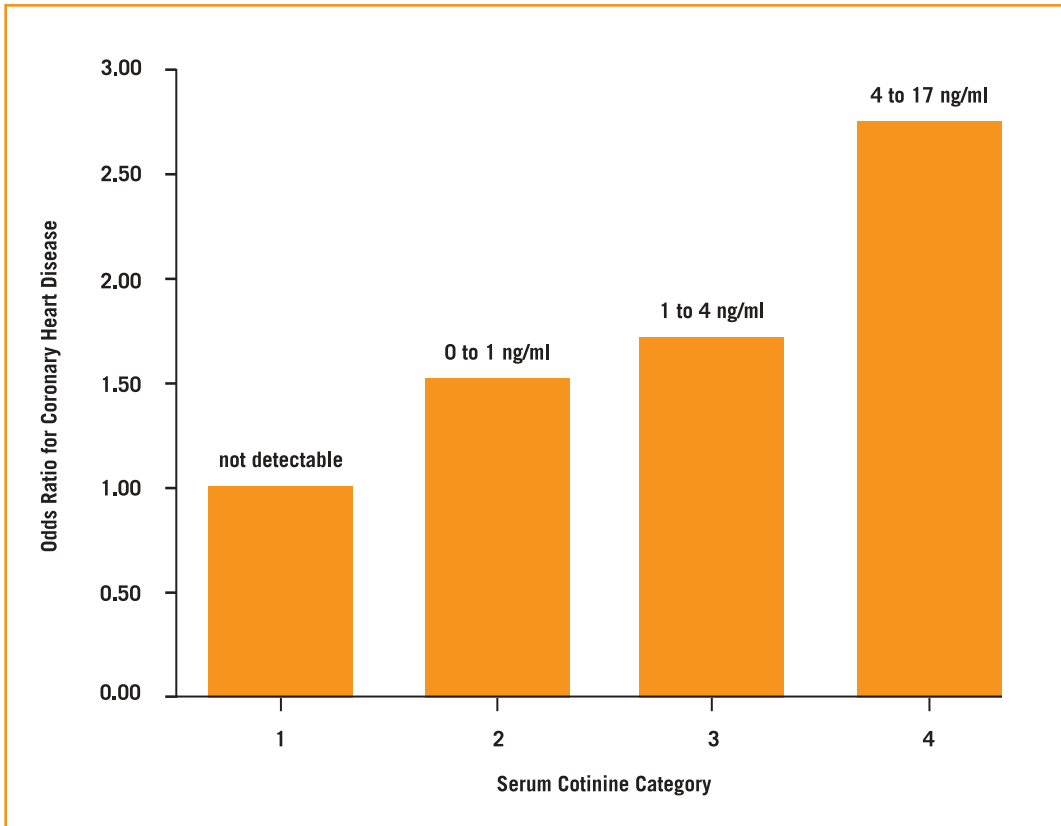*(Hirayama T., Proc. 5th World Conf. Smoking & Health, 1983)*

Excess Lung Cancer Risk (%)

NON SMOKER — EX SMOKER — 1-14 CPD — 15-19 CPD — >20 CPD

Spouse's Smoking Category (cigarettes per day)
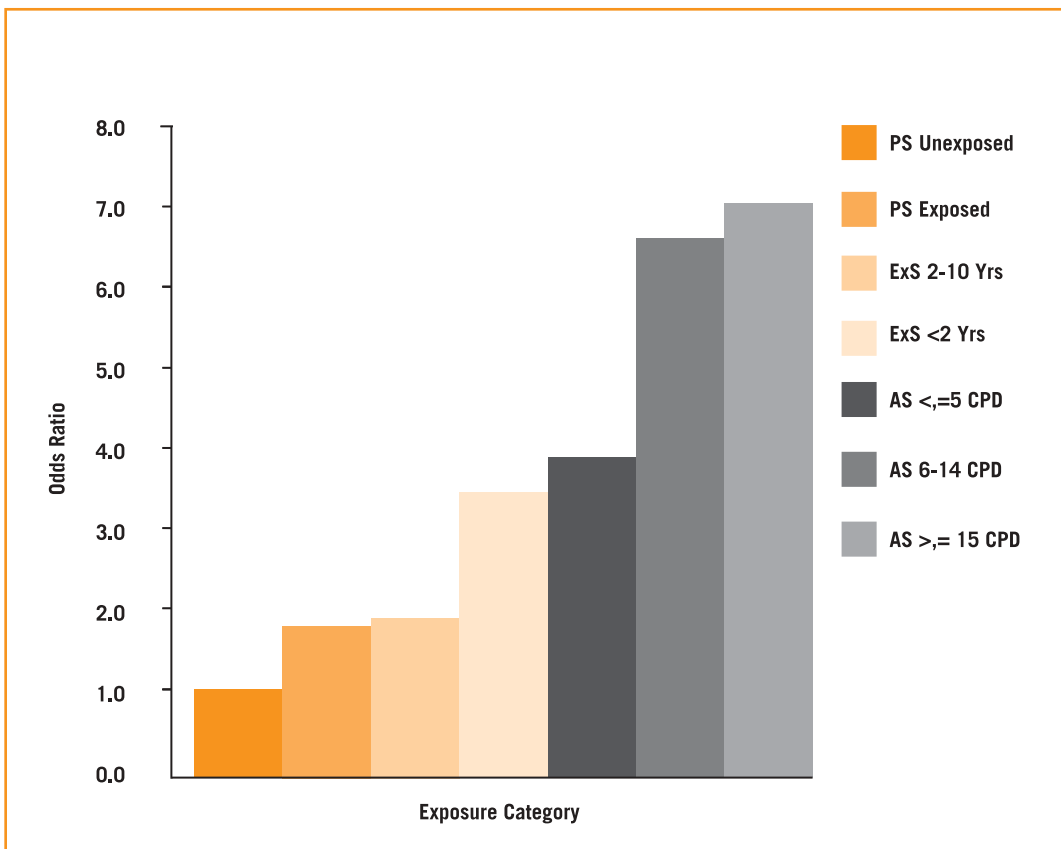
4 PASSIVE SMOKE: THE THREAT TO THE UK WORKFORCE

4.1 Estimated Total Mortality from Passive Smoking in the U.K

One method of estimating U.K. SHS mortality is to assume the population age and passive smoking exposure distributions for the U.S. and the U.K. are the same, and use the estimates of Wells (1999) for the U.S. population to estimate the U.K. passive smoking risk by the population ratio. The results, shown in Table 1, yield an estimated 12,300 deaths per year, of which 10,185 are from lung cancer and heart disease, and the remainder from other known or suspected causes.

Alternatively, this can be done from U.K. cotinine studies. Jarvis (2001) reported data for salivary cotinine (a nicotine metabolite which is a standard biomarker for passive smoking) for various groups of nonsmokers from the Health Survey for England in 1998, shown in Table 2 on page 11. A subset of London Bar workers is shown for comparison. Repace et al. (1998) developed dose-response relationships between salivary cotinine and estimated lifetime risk of passive-smoking-induced death from heart disease and lung cancer. The combined relationship estimates for a working lifetime of 40 years, 11 deaths per 1000 persons aged 35 years or more who have an average salivary cotinine of 0.4 nanograms per millilitre (ng/ml) over that period. Table 2 shows that the average English nonsmoker has a salivary cotinine burden of 0.86 ng/ml. The current population of the U.K. is 59 million (all ages); the adult population of the UK in 2001 at or above 35 years of age (the age range for lung cancer and heart disease, etc.), is about 26.6 million persons (UK Statistics, 2003), of which 73% or 19.4 million are nonsmokers.

Cause	USA	UK	
Lung Cancer	3 060	623	
Heart Disease	47 000	9 562	
Breast Cancer	8 700	1 700	
Cervical Cancer	500	102	
Nasal Sinus Cancer	200	41	
Brain Cancer, Leukemia and Lymphoma	1 000	203	
TOTAL DEATHS	60 460	12 300	per year
POPULATION (2001)	290 million	59 million	

Equation One

The estimated lifetime mortality M, assuming all of the U.K. (including Wales, Scotland, and Northern Ireland) is exposed to the same extent as England, is estimated at about 11,480 heart disease and lung cancer deaths per year.

$$M = (0.86 \text{ ng/ml})\{(11 \text{ deaths})/(10^3 \text{ persons-40 yrs-0.4 ng/ml})\}(19.42 \times 10^6 \text{ persons}) = 11,480 \text{ deaths/yr}$$

If this is adjusted upward by the ratio of total deaths to lung and heart deaths in Table 1, the result is 13,900 total deaths. Both methods support the "thousands" of U.K. deaths per year estimated by the SCOTH Report in 1998.

4.2 Passive Smoking Risk in Subgroups of Workers: Bar workers

The total estimated passive smoking mortality figures for the U.K. do not illuminate the risk to specific groups of workers. To understand this issue, we first turn to an analysis of the London bar staff cotinine data reported by Jarvis (2001). It shows that the exposure of bar staff is much higher than that of the average English non smoker, including non smokers married to smokers, who form the basis for nearly all epidemiological studies of passive smoking in adults.

Figure 9, on page 12, shows a graph of bar staff salivary cotinine versus estimated lifetime mortality probability. The risk is estimated from salivary cotinine S by Equation 2 (Repace, et al., 1998), and the salivary cotinine data are due to Jarvis (personal communication).

Figure 9 gives the workers' percentile distribution for this risk as a function of salivary cotinine dose while Figure 10, on page 12, gives the risk by percentile. Figure 9 shows that half the bar workers have an *estimated lifetime mortality probability* from on-the-job passive smoking of 10% (unadjusted for competing causes of death). The average bar staff, with a salivary cotinine level of 6.16 ng/ml, has an estimated mortality rate of (0.0275)(6.16) = 17%. This is an absolute risk; in other words, 17 out of every 100 bar staff would be expected to die from heart disease or lung cancer as a result of their workplace exposures to SHS. This absolute probability estimate is not to be confused with the "30%" *relative risk* increase reported from epidemiological spousal smoking studies, which is 30% above a background lung cancer mortality probability for U.S. nonsmokers of about five per 1000, or in absolute terms, a lifetime probability of dying of (1.3)(5 x 10-3) = 6.5 per thousand, or 0.65%.

About 1% of U.K. workers work in pubs, bars, and restaurants, very few of which are smoke-free (BMRB 2002). Assuming 30% of these work in pubs and bars and that about two-thirds of adults aged 15+ are currently in employment (BMRB, 2002), an estimated 53,200 persons are employed in pubs in the U.K ((0.003)(2/3)(26.6 million persons aged 35+). In fact, industry estimates report 53,000 pubs in England and Wales (Public Places Charter Group, 2001), so this likely underestimates the number at risk. As stated above, 17% of pub workers would be expected to die from SHS in the workplace over a period of 40 years, placing the annual estimated death toll among all pub and bar workers in the U.K. at 226 deaths per year. (53,200)(0.17)/ (40). Assuming a 27% smoking prevalence, **165 of these would be nonsmokers.**

Studies of bar workers in Ireland (150 deaths/year; Mulcahy et al., 2002) and restaurant workers in Hong Kong (170 deaths/ year; Hedley et al., 2002) report similarly high risks from secondhand smoke exposure. Supporting this is the study of Eisner et al. (1998), who found that the respiratory health of California bartenders – both nonsmokers *and* smokers – improved measurably after the California workplace smoking ban.

Equation Two
Risk (Deaths/ 40-Yr Working Lifetime) = 0.0275 S (ng/ml)

Table 2
Salivary Cotinine in all English Nonsmokers and London Bar staff *(Jarvis, 2001)*

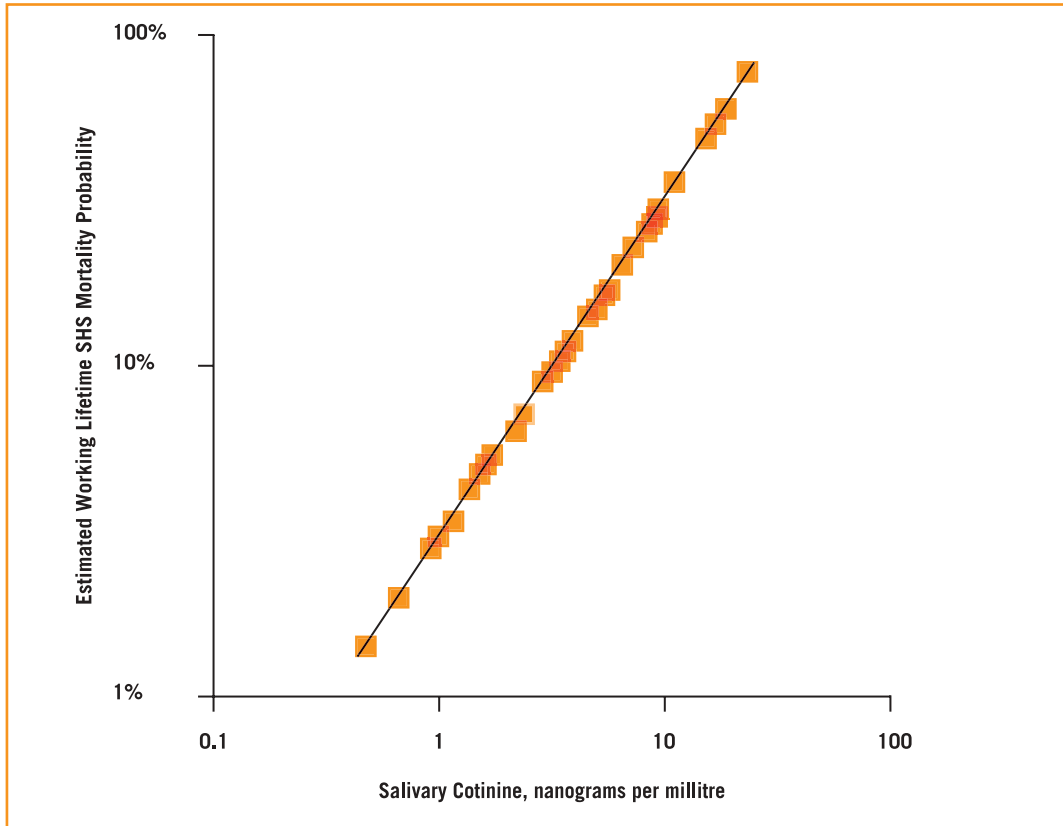|	London bar staff, 2000		Health Survey for England 1998		
	Including cotinine <15ngml	Including cotinine <30ngml	All nonsmokers	Nonsmokers from nonsmoking households	Nonsmokers married to smokers
N	39	44	7123	3558	653
Arithmetic mean	4.22	6.16	0.86	0.51	1.94
Geometric mean	2.91	3.71	0.35	0.27	0.99
Median	3.20	3.65	.40	.30	1.20
95th percentile	10.8	21.7	3.5	1.80	6.56

Figure 9
London Bar Staff:
Salivary Cotinine vs. SHS Risk
(Cotinine data: Jarvis, 2001)

Estimated working lifetime mortality risk for London Bar Staff from passive smoking-induced heart disease and lung cancer.
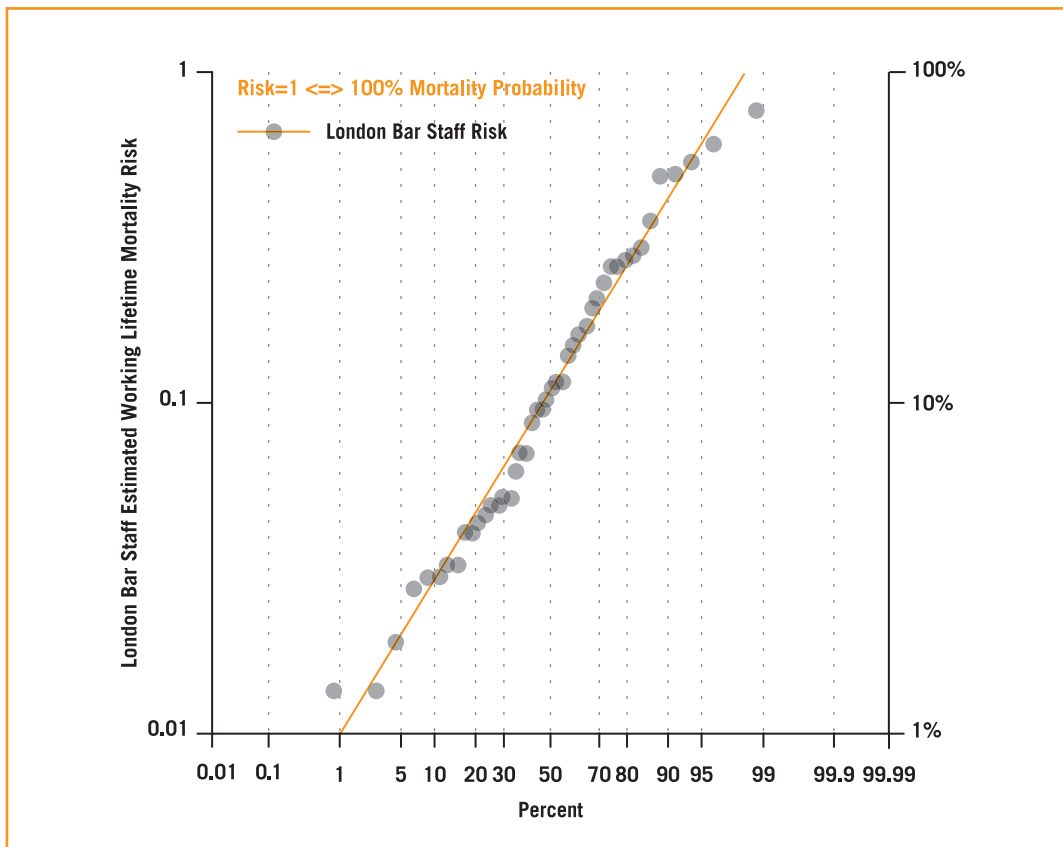

Figure 10
Estimated SHS risk of London Bar Staff by Percentile
(Data: Jarvis 2001)

4.3 Passive Smoking Risk in Subgroups of Workers: Office Workers

Emmons et al. (1992; 1994) measured saliva cotinine in 89 U.S. nonsmokers (mostly office workers) in the late 1980's exposed to SHS only at work. Emmons et al. (1992) reported that these workers had median cotinine levels of 0.5 ng/ml (95th percentile, 2.4 ng/ml). Repace et al. (1998) developed a combined physical-pharmacokinetic model to estimate salivary cotinine in nonsmoking U.S. workers based upon smoker density and office ventilation rates. Repace et al's 1998 model estimated an arithmetic mean salivary cotinine level, 0.70 ng/ml, for the typical office worker (median, 0.5 ng/ml and 95th percentile 2.0 ng/ml). Repace et al.'s modelled mean value is less than the arithmetic mean of 0.86 ng/ml, reported by Jarvis (2001) in Table 1 for all English nonsmokers (median, 0.40 ng/ml, and 95th percentile, 3.5 ng/ml). Using a risk assessment model, Repace et al. (1998) estimated that 4,000 heart disease deaths and 400 lung cancer deaths occur annually among office workers from passive smoking in the workplace. These values can be scaled to the U.K. as follows: 4,400 U.S. office worker deaths times the ratio of the U.K. to the U.S. populations (4400)(59/290) = **895 deaths per year among U.K. nonsmoking office workers.**

4.4 Passive Smoking Risk in Subgroups of Workers: Industrial Workers

Industrial workers' risks cannot be estimated so simply as office or bar workers until cotinine studies are performed on such groups. Industrial workers may work in such widely disparate sectors as manufacturing, mining, construction, transport, and agriculture. Exposure venues may vary from cramped and poorly-ventilated mine shafts or the holds of ships to the well-ventilated open fields of farms and the windy tops of tall buildings under construction. However, we do know that about 6% of U.K. workers are employed in manufacturing. (BMRB Access Poll, 2002)

Manufacturing Workers

Due to a lack of UK data, estimates for the impact of passive smoking on manufacturing workers are based on figures gleaned at a cutting tool manufacturing plant in the State of Wisconsin in 1997. It has been assumed that manufacturing companies in the UK would have similar dimensions and ventilation.

Figure 11 shows an equation for estimating the SHS respirable particulate (RSP) concentration. Substituting the values from the case study into this equation yields an estimated concentration in units of micrograms per cubic meter (µg/m³) of

SHS-RSP = 22,000 (nhs/V)/(Cv) = (22,000)(16/19,587)/(0.52) = 35 µg/m³

Repace et al. (1985; 1993; 1998) estimated that exposure to a SHS-RSP concentration of 75 µg/m³ during a work shift for a working lifetime of 40 years yields a fatal lung cancer risk of 1 death per thousand workers at risk, and fatal heart disease risk of 1 death per hundred workers at risk, for a combined risk of 11 deaths per thousand workers at risk.

Thus, exposure to 35 µg/m³ of SHS-RSP during a working lifetime yields an estimated risk of (35/75)(11 per 1000) five deaths per thousand workers. The U.S. Occupational Safety and Health Administration's (OSHA) "significant risk" level for industrial workers is 1 death per thousand workers per working lifetime of 45 years. The risk to the nonsmoking cutting tool workers from their co-workers' smoking exceeds OSHA's significant risk level by more than a factor of five.

SECONDHAND SMOKE AIR POLLUTION EQUATION

$$\text{SHS-RSP} = 22000 \left(\frac{n_{hs} \; \textbf{Number of Habitual Smokers}}{\dfrac{V \; \textbf{Volume of room, } m^3}{C_v \; \textbf{Air Exchange Rate, } h^{-1}}} \right)$$

Units: micrograms per cubic meter (µg/m³)

The secondhand smoke respirable particulate pollution level is dirctly proportional to the habitual smoker density, and inversely proportional to the air exchange rate.

Figure 11
Respirable Particulate (RSP) Air Pollution from SHS depends upon the average smoking rate, the size of the room, and the ventilation rate: Equation Three. The equation yields the concentration assuming uniform dilution, and may underestimate personal exposure.

Case Study Passive Smoking in a US cutting tool factory.
The plant employed nhs = 16 smokers and 19 nonsmokers on the first shift. It had a volume of V = 19,587 cubic metres (m³). The plant was ventilated by five exhaust fans attached to various industrial machinery, which provided an outside make-up airflow of 6,332 m³/hr, equivalent to an air exchange rate of C_v = 0.32 air changes per hour (h⁻¹). Infiltration (unintentional ventilation caused by leaks) was estimated to contribute an additional 0.2 air changes per hour for a total C_v = 0.52 h⁻¹.

According to the BMRB Access poll, 6% of U.K. workers (15+ in age) are employed in manufacturing. Assuming that 6% of workers (35+ in age) are employed in manufacturing, of the 26.6 million workers aged 35+, an estimated 1.6 million are in manufacturing. If the heroic assumption is made that the exposure in the Wisconsin cutting tool plant is characteristic of all British manufacturing workers, then the estimated SHS mortality among the latter workers is (5/1000)(1,600,000) 8,000 deaths per 40 years, or 200 deaths per year, of the same order as estimated for bar workers. About 27% of those deaths would be in smokers. Of the deaths, roughly 10%, or 20 deaths per year would come from lung cancer, and roughly 90%, or 180 deaths per year from ischaemic heart disease. **An estimated 146 of the total deaths would be in nonsmokers.**

How does 200 deaths per year from SHS compare to occupational health statistics for manufacturing workers in the U.K. from industrial exposures? According to the Health and Safety Executive (HSE), almost half of new cases qualifying for benefit in 2000 were in the metal machinery and related trades (www.hse.gov.uk/statistics/index.htm). For the sum total of all prescribed diseases in the manufacturing sector (asthma, dermatitis, musculoskeletal disorders, occupational deafness, vibration white finger, asbestosis, and mesothelioma; table A2.10, p. 196), a total of 24.5 per 100,000 workers in 1999-2000 were afflicted. This compares with 500 per 100,000 estimated for heart disease and lung cancer from SHS. For all occupational cancers other than mesothelioma, about 80 cases obtained disablement benefits in 1999/2000; about 40 of these were lung cancer.

Based on plausible assumptions, it appears that the mortality rate from SHS in manufacturing workers is at least an order of magnitude higher than all the reportable occupational health conditions. In terms of fatal injuries in the manufacturing sector, there were 47 occupational deaths reported in the manufacturing sector in 2001/2002 (table 12a, (http://www.hse.gov.uk/ statistics/industry/index.htm#man).

Thus the estimated number of fatalities from passive smoking is quadruple the number of all fatal occupational injuries among workers in the manufacturing sector, and for nonsmokers only, it is triple. While the estimates of exposure for SHS in this sector must be confirmed with cotinine studies, it indicates that by U.K. occupational health criteria, this is a serious impact.

5 PASSIVE SMOKE: THE POLICY IMPLICATIONS

5.1 Why Ventilation is not an Adequate Solution

The hospitality-industry-sponsored self-regulatory Atmosphere Improves Results (AIR) initiative promotes *The Public Places Charter on Smoking* (Charter, 2001), which describes the efforts of the industry to "promote practical techniques to resolve the public smoking issue, through ventilation and/or non-smoking areas." The self-stated aim of the Charter is to "improve customer choice by highlighting those premises with smoking restrictions and/or ventilation that meets the Charter standard" In this, it appears very similar to the tobacco industry-sponsored "Accommodation Program" in the U.S.

The ventilation standard promoted by AIR promotes a minimum fresh air mechanical ventilation requirement of 12 air changes per hour (h^{-1}) for a room with a 2.5 m ceiling (8.5 ft), or 7.5 h^{-1} for a room with a 4 m ceiling. In addition, a comfort requirement is suggested so that staff and customers are comfortable (defined as no smoke haze, no stinging eyes, no smell of smoke on clothes.) No attempt is made to establish a level that is safe by occupational or environmental health standards. AIR observes that the Charter is a self-regulatory program that has the same provisions as the draft Health and Safety Commission's Approved Code of Practice (ACoP) for passive smoking at work, except that where the Charter leaves it up to the proprietor which policy he chooses, the draft ACoP provides a hierarchy based on banning smoking in whole or in part, and allows employers to be prosecuted or sued if their staff could show that their "welfare" had been harmed by environmental tobacco smoke (www.airinitiative.com)

According to the Charter, there are approximately 53,000 pubs in England and Wales, of which 40,000 are owner-operated tenancies, leaseholds, or freehold independent traders. The remaining 13,000 are managed outlets, and tend to be the larger premises with very large floor areas. The Charter states that pubs are usually open plan with about half consisting of single room venues with low ceilings, beams, thick walls, and planning restrictions on modifications. "Pubs traditionally have a high level of environmental tobacco smoke because a high proportion (47%) of customers are smokers." It goes on to state that these smaller venues typically have poor ventilation, poor equipment maintenance, and lack of feasibility for nonsmoking areas in many. The Charter asserts that it is promoting voluntary means for operators to reduce staff and customer exposure to smoke.

Analysis of the Charter Ventilation Initiative

A fatal flaw in the Charter is that it seeks to "reduce" SHS levels without providing for a safe and healthy atmosphere for pub staff or patrons, merely a less annoying one. Figure 11 demonstrates that the time-averaged fine-particle concentration of SHS in a space depends upon the average number of cigarettes smoked during the interval, and the volume of the room, as well as the air exchange rate. If ventilation is to be applied, the resultant SHS concentration, being toxic and carcinogenic, should be low enough to be judged "safe," by a professional measure of acceptable air quality. In other words, it is impossible to state that a given ventilation rate will control SHS unless the smoking rate, the room size, the ventilation rate, and the acceptable concentration are all specified. The Charter does not define the risk to staff or patrons either before or after the proposed control measures are implemented, nor indeed does it provide any enforcement measures whatsoever. It is therefore deceptive and unprofessional. We are entitled to ask – and answer – how safe is it?

Charter Air Exchange, Volume, and Smoking Occupancy

To evaluate the safety of the Charter-recommended air exchange rate, the SHS air pollution equation described in Figure 11 is useful. This equation utilizes the number of habitual smokers (n_{hs}) , the air exchange rate (C_v), and the volume of the room (V). From the above paragraphs, the Charter-specified air exchange C_v = 12 h^{-1} for a 2.5 m ceiling or 7.5 $^{h-1}$ for a 4 m ceiling. The number of smokers is n_{hs} = 47% of patrons (P). The room volume and number of patrons are determined as follows. The Air Initiative website specifies a bar-restaurant of 10 metres long by 10 metres wide for 100 m^2 of floor space, equally divided between the bar and the restaurant as an example. If the ceiling height is 2.5 m, then the total volume V is 250 cubic metres (m^3), and if it is 4 m, then the total volume is 400 m^3, with the bar and the restaurant each sharing half of the total, for 125 m^3 and 200 m^3 for the low and high ceilings respectively. The person occupancy is not specified, so it will be taken from the U.S. ventilation standard, called the ASHRAE Standard 62 (1999), Ventilation for Acceptable Indoor Air Quality, which specifies a maximum restaurant occupancy as P = 70 persons per 100 m^2 of occupiable floor area, and a maximum bar occupancy as 100 persons per 100 m^2 of occupiable floor area. Thus, the number of smokers in the bar consistent with the Charter would be n_{hs} = .47(100) = 47. We conservatively assume that the restaurant part of the pub is a no-smoking area. Thus, the dilution volume is 250 m^3.

Expected Air Pollution Level in a Charter Pub

Using Figure 11, for a Charter Bar with a 2.5 m ceiling, V = 250 m^3, n_{hs} = 47 habitual smokers, and C_v = 12 air changes per hour (h^{-1}). The equation yields a predicted respirable particle ($PM_{3.5}$) SHS-RSP = 22,000 $\{(n_{hs}/V)/C_v)\}$ = (22,000)$\{(47/250)/12)\}$ = 345 micrograms per cubic metre ($\mu g/m^3$)

What is the saliva cotinine equivalent of 345 ($\mu g/m^3$) for occupationally exposed bar staff routinely working an eight hour per day work shift for a popular bar at full occupancy? Repace et al.(1998) estimated the following relationship between salivary cotinine S and SHS-RSP:

Equation Three
S (ng/ml) = 0.0057 SHS-RSP ($\mu g/m^3$).

Thus, the estimated salivary cotinine level for bar staff in a Charter-ventilated bar would be $S = (0.0057)(345) = 1.97$ ng/ml. From Equation 2, this yields an estimated Risk = $0.0275 S = (0.0275)(1.97) = 5\%$. In other words, at the Charter-recommended ventilation rate for a pub at full occupancy, an estimated five out of every 100 bar staff will die from job-related passive smoking-induced heart disease or lung cancer during his or her working lifetime. The Charter Group states that 27% of the 43,000 pubs surveyed in September of 2001 stated they were in compliance with the Charter. Figure 10 shows that in 2001, based on the cotinine studies of Jarvis (2001), about 5% of London bar staff had estimated lifetime mortality risks of between 1% and 5%, and 95% had risks greater than 5%. Note that at full compliance, at 5%, the estimated number of deaths per year among bar staff from passive smoking remains unacceptable at $(5/17)(226) = 66$ deaths per year. This demonstrates the fundamental flaw in the ventilation approach.

A second major flaw in the Charter ventilation approach becomes apparent when the estimated concentration is compared to the U.K. National Air Quality Standards: the 24-hr average NAQS for inhalable particles (PM_{10}) is 50 $\mu g/m^3$. The estimated level of 345 $\mu g/m^3$ of RSP ($PM_{3.5}$) for an eight hour work shift averages out to $(8/24)(345) = 115$ $\mu g/m^3$ over a 24-hour period. Assuming the outdoor background is in compliance with the annual NAQS of 40 $\mu g/m^3$ the exposure of the bar staff will violate the 24 hour standard by a factor of $(115+40)/50 = 3$. The Charter on its face yields unclean air.

How Realistic Is the Estimate of Air Pollution for a Charter-ventilated Pub?
A comparison can be made using exact data from a pub in Toronto, Canada on Friday, 13 December 2002.

Case Study Air Pollution Levels Measured in a Toronto Pub
Air pollution levels were measured in a 295 m^3 Toronto pub ventilated at 8.6 h^{-1}, with a 2.9 m ceiling, with 46 persons per 100 m^2 occupancy, and a 42% smoking prevalence, conditions similar to a Charter-compliant U.K. pub. The average indoor RSP level was 199 $\mu g/m^3$ over an 4.4 hour period, measured on 13.12.2002 using a respirable aerosol (RSP) monitor (MIE personal Data Ram, model 1200), and a photoelectric particle-bound polycyclic aromatic carcinogen (PPAH) monitor for airborne carcinogens (EcoChem PAS2000 CE). The data are plotted in Figure 12. Indoor PPAH averaged 152 ng/m^3, while the outdoor averaged 8 ng/m^3, or 5% of the indoor value. The indoor SHS-RSP fraction is estimated by subtracting off the 21 minute average outdoor RSP background of 22 $\mu g/m^3$ (11% of the indoor value) yielding 177 $\mu g/m^3$. This is adjusted to the Charter defaults for smoking prevalence, occupancy, air exchange, and volume as follows: $(177 \ \mu g/m^3)(47\%/42\%)(100 \ P/46 \ P)(8.6 \ h^{-1}/12 \ ^{h-1})(295 \ m^3/250 \ m^3) = 364 \ \mu g/m^3$, within 6% of the 345 $\mu g/m^3$ estimated above.

The Toronto Pub data suggests that 90% of the indoor RSP was due to SHS, and 95% of the indoor PPAH as well. This is in accord with a recent study performed before and after a smoking ban in the U.S., in the State of Delaware. Figure 13 shows the results.

RSP monitor in at 7:10 PM; PPAH monitor in at 7:14 pm; both out at 11:35PM
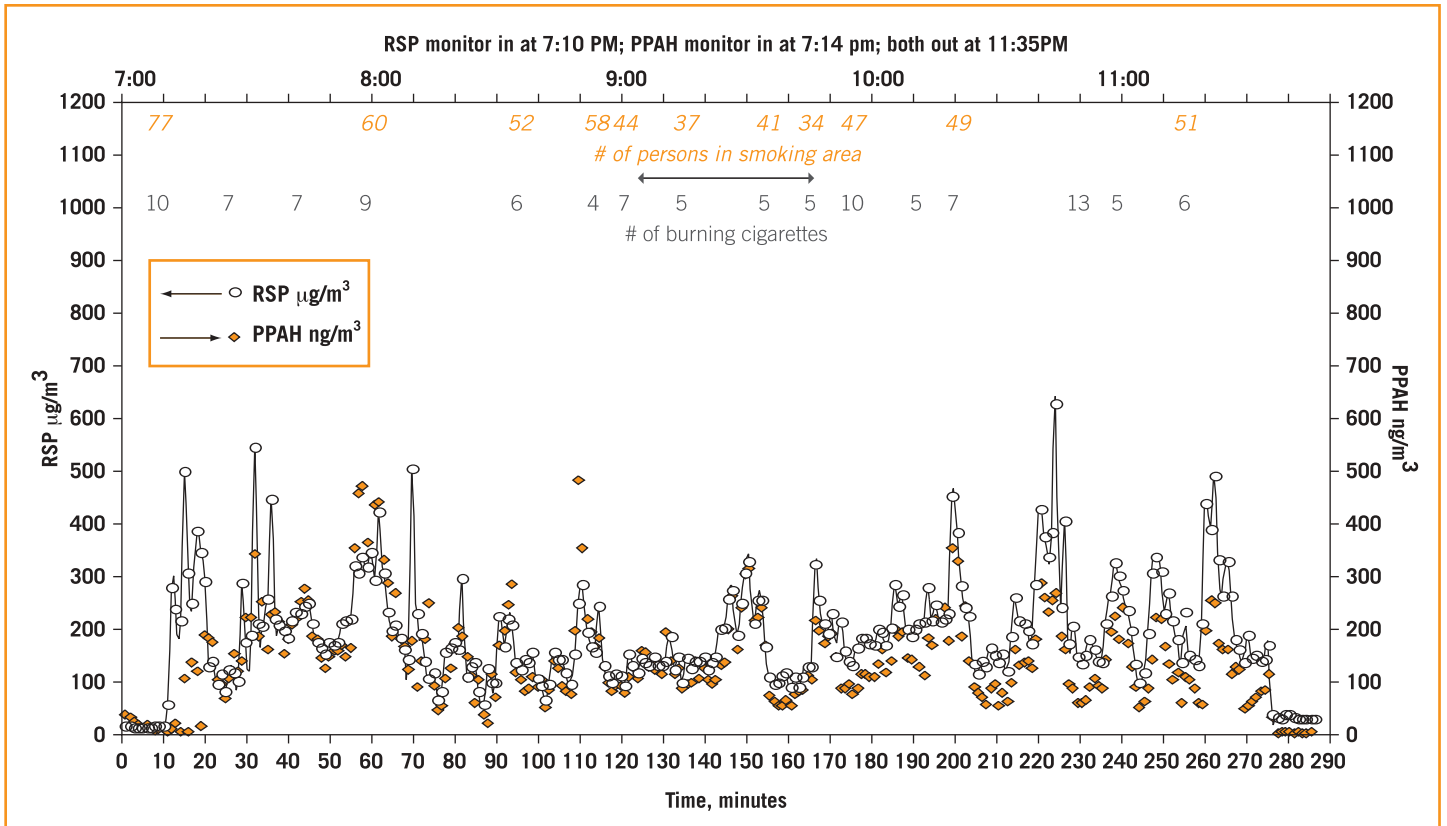

Figure 12 (above)
Metropolitan Toronto, Canada Pub: RSP & PPAH vs Time, Friday Dec 13 2002
(J.L. Repace unpublished)

A Metropolitan Toronto, Canada, pub of smoking prevalence, size, and air exchange rate similar to that specified by the Public Places Charter on Smoking of the AIR Initiative of the U.K. hospitality industry, is heavily polluted with respirable particles (RSP) and airborne carcinogens (PPAH). Indoor levels were measured between 11 and 280 minutes elapsed time, outdoor measurements are from 1-10 and 277-288 minutes. All data points are 1 minute averages.
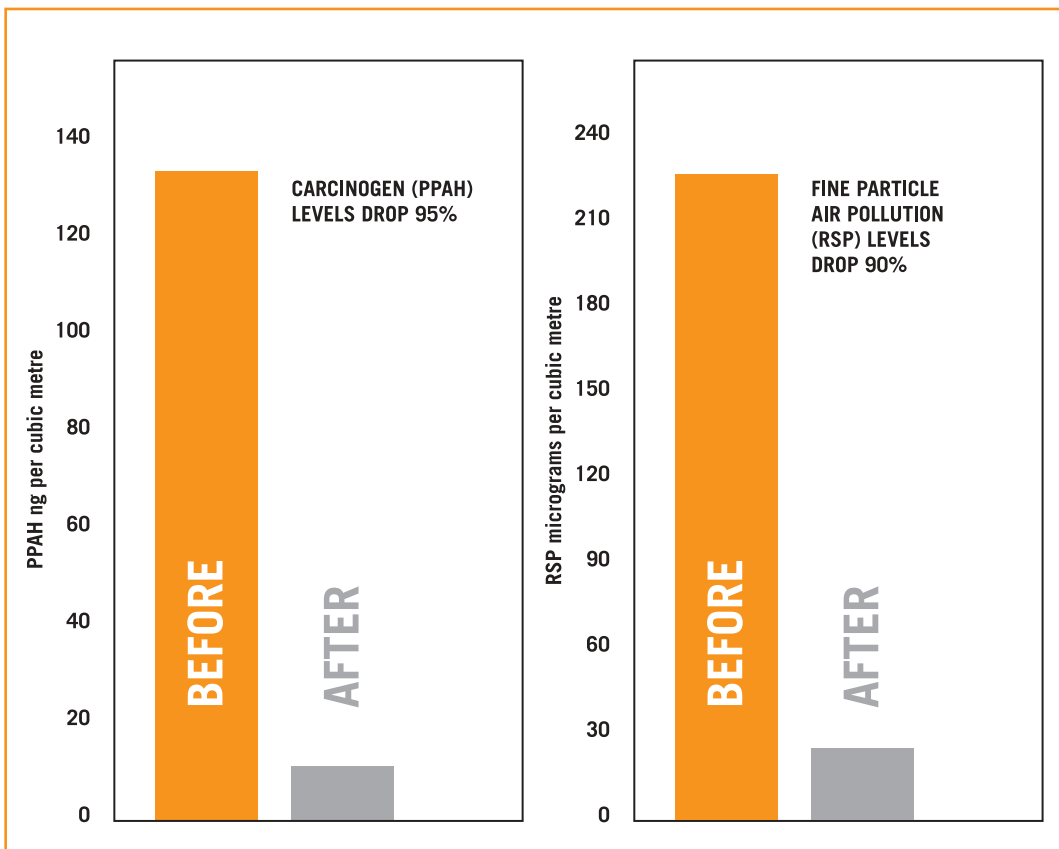
Figure 13 (left)
Delaware Hospitality Venues
(Repace Associates, Inc. Febraury 3, 2003)

Indoor Air Quality testing was conducted on 15th November 2002 and 24th January 2003 to assess the levels of air pollution before and after the Clean Indoor Air Law (smoking ban) went into effect. One casino, five restaurants with bars, one stand-alone bar (taproom) and one pool hall were tested. The results showed that workers and patrons are exposed to significantly lower levels of airborne pollutants and carcinogens thanks to the smoke-free law.

5.2 Economics of Hospitality Industry Smoking Bans

An assessment of 97 studies found that no-smoking policies in restaurants and bars don't harm business, despite concerted efforts by the tobacco industry to prove otherwise (Scollo and Glantz, 2003). In 1995, California banned smoking in all restaurants and other workplaces, and in 1998, extended the ban to include all bars. Delaware followed suit in 2002. In March 2003, New York City banned smoking in bars. Boston will follow suit in May. The California ban on smoking in bars provided immediate respiratory health benefits for bartenders: establishment of smoke-free bars and taverns was associated with improvement in workers' respiratory health for both nonsmokers and smokers (Eisner et al.,1998).

The California regulation also proved to be healthy for its hospitality industry, as Figure 14 shows.

5.3 What the public want

The nonsmoking majority avoids smoky premises. The long-term increase in sales following the California smoking ban may be explained by nonsmokers' aversion to tobacco smoke. In 1995-96, Biener et al. (1999) at the University of Massachusetts (Boston), surveyed a representative sample of 4,929 Massachusetts adults to assess who avoids smoky restaurants and bars, and why. The adult population of Massachusetts (≥18 years) is 4.5 million, including 3.7 million non-smokers, and 800,000 smokers. Biener et al.'s survey found that 76% of the nonsmokers were bothered by tobacco smoke, and that 46% of nonsmokers reported that they avoided smoky places due to offensive odours or health worries. Biener et al. estimated that, in 1996, due to secondhand smoke concerns, more than half a million (515,405) adult nonsmokers avoided patronising restaurants and 364,400 nonsmokers avoided patronising bars. This means that 880,000 Massachusetts nonsmokers avoided smoky restaurants and bars, exceeding by 80,000 persons the entire number of smokers in the State. In other words, secondhand smoke loses trade.

And in the UK, over four in ten people (42%) considered whether or not a place has a non smoking area as an important factor when deciding where to go for a meal. Just under a fifth (19%) regarded whether a place has a non smoking area as an important consideration in their choice of a place to go for a drink. (Office for National Statistics, 2001).

Figure 14
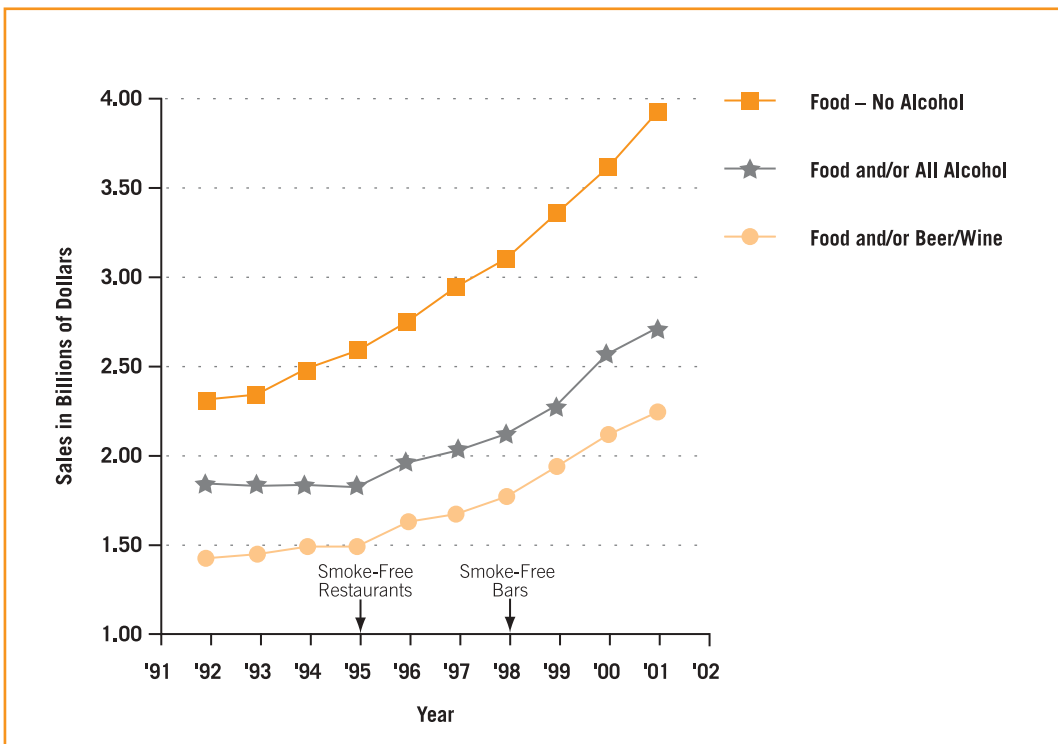**First quarter taxable sales figures for California restaurants and bars, State of California, '92-'01**
(Source: California Dept. of Health; California Board of Equalization)

The sector labelled "Food &/or All Alcohol" includes both stand-alone bars and restaurants with bars. Note that sales were flat in the alcohol sales sector until the smoking ban, and that revenues have increased every year since the ban.

6 CONCLUSIONS

1 There is an international consensus that secondhand smoke (SHS) exposure is a cause of death from lung cancer and heart disease, and is suspected to cause many of the other diseases known to afflict smokers.

2 In the U.K. population, it is estimated that there are about 12,000 deaths per year due to passive smoking, based both on English biomarker studies as well as extrapolation from credible U.S. estimates.

3 Among the estimated 53,000 U.K. bar workers, it is estimated that 17% will die from passive smoking during their working lifetime, amounting to 165 deaths per year among nonsmokers.

4 For U.K. nonsmoking office workers, it is estimated that there are about 900 deaths per year from passive smoking, based on extrapolation from U.S. estimates, adjusting for relative population size.

5 For U.K. manufacturing workers, it is estimated that there are about 146 deaths per year among nonsmokers. While this estimate must be viewed as preliminary, in perspective, it is triple the annual number of fatal occupational injuries among U.K. manufacturing workers.

6 Under the U.K. hospitality-industry-sponsored *Public Places Charter* on Smoking ventilation standard to control smoking, it is estimated that five out of every 100 bar workers would die from passive smoking during their working lifetime.

7 The U.K. hospitality-industry-sponsored Public Places Charter on Smoking ventilation standard violates the U.K. 24-hour Air Quality Standard for particulates (PM_{10}) for workers by a factor of three for a pub at full occupancy and Charter-specified smoking prevalence.

8 Based on studies in nine venues in the U.S. and Canada, eliminating smoking in hospitality industry workplaces appears to reduce 90% of the fine particle air pollution, and 95% of the airborne carcinogens.

9 Based on many U.S. studies, secondhand smoke causes a net loss of trade for the hospitality industry by causing offense to nonsmokers from odour, irritation, and health concerns.

10 It would require tornado-like quantities of ventilation, in excess of 10,000 air changes per hour, to produce acceptable risk for bar staff from passive smoking.

REFERENCES

1. ASHRAE Standard 62-1989, Ventilation for Acceptable Indoor Air Quality, The American Society for Heating, Refrigerating, and Ventilating Engineers, Atlanta, GA.

2. BMRB International. A compilation of U.K. labour force statistics and smoking restrictions by ACCESS, for ASH, 2002.

3. Biener L and Fitzgerald G. Smoky bars and restaurants: who avoids them and why? J Publ Health Manage. & Practice 5: 74-78 (1999).

4. CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY. (1997). Health Effects of Exposure to Environmental Tobacco Smoke, Final Report. Office of Environmental Health Hazard Assessment.

5. Charter Group. The Public Places Charter on Smoking. An interim progress report to the Department of Health. The British Institute of Innkeeping, Association of Licensed Multiple Retailers, British Beer & Pub Association; British Hospitality Association, The Restaurant Association. 16 November 2001.

6. Eisner MD, Smith AK, Blanc PD. Bartenders' respiratory health after establishment of smoke-free bars and taverns. JAMA 280:1909-1914 (1998).

7. Emmons KM, Abrams DB, Marshall R, Marcus BH, Kane M, Novotny TE, and Etzel RA. An evaluation of the relationship between self-report and biochemical measures of environmental tobacco smoke exposure. Preventive Med 1994; 23:35-39.

8. Emmons KM, Abrams DB, Marshall RJ, Etzel RA, Novotny TE, Marcus BH, and Kane ME. Exposure to environmental tobacco smoke in naturalistic settings. American Journal of Public Health 82: 24-28 (1992).

9. Hedley AJ, McGhee SM, Repace JL, Wong TW, Yu MYS, Chan AYW, Lam TH, Lo PCK, Tsang M, Wong LC, Chan ALN, Ng ESL, and Janghorbani M. Passive smoking and risks for heart disease and cancer in Hong Kong catering workers 2001. Report # 8, Hong Kong Council on Smoking and Health. < http://www.info.gov.hk/hkcosh/enew-index.htm>.

10. Hoffmann D and Hoffmann I. Chemistry and Toxicology. In Smoking and Tobacco Control Monograph 9. Cigars – Health Effects and Trends. National Institutes of Health, National Cancer Institute, Bethesda, MD (1998).

11. Jarvis M. Quantitative survey of exposure to other people's smoke in London bar staff. Department of Epidemiology and Public Health University College London, September 13th 2001.

12. Johnson KC, Repace J. Lung cancer and passive smoking – Turning over the wrong stone. BMJ 321 (7270) 1221 (2000).

13. Mulcahy M, Repace JL. Passive Smoking Exposure and Risk for Irish Bar Staff. Presented at the 9th International Conference on Indoor Air Quality and Climate, Monterey, California, June 30 – July 5, 2002.

14. National Cancer Institute. Respiratory health effects of passive smoking: lung cancer and other disorders; The report of the U.S. Environmental Protection Agency. National Cancer Institute Smoking and Tobacco Control Monograph 4, NIH Publication # 93-3605, National Institutes of Health, Bethesda, MD., August 1993.

15. National Cancer Institute. Smoking and Tobacco Control Monograph 9. Cigars – Health Effects and Trends. National Institutes of Health, National Cancer Institute, Bethesda, MD (1998).

16. National Research Council (1986). Environmental tobacco smoke – measuring exposures and assessing health effects. National Academy Press, Washington, DC.

17. National Toxicology Program. 9th Report on Carcinogens 2000. U.S. Dept. of Health & Human Services, National Institute of Environmental Health Sciences, Research Triangle Park, NC.

18. NIOSH Current Intelligence Bulletin #54. Environmental Tobacco Smoke in the Workplace, Lung Cancer and Other Health Effects. U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health, Cincinnati, OH June 1991.

19. NIOSH Pocket Guide to Chemical Hazards, U.S. Dept. Health & Human Services, Centers for Disease Control & Prevention, June 1994.

20. Ott WR. Mathematical models for predicting indoor air quality from smoking activity. Env Health Persp 107, suppl 2, 375-381 (1999).

21. Peto R, Lopez AD, Boreham J, Thun M, Health C. Mortality from smoking in developed countries 1950-2000. Oxford University Press, Oxford, 1994.

22. Pirkle et al., JAMA 275:12-33-1240 (1996).

23. Repace JL, and Lowrey AH. A Quantitative Estimate of Nonsmokers' Lung Cancer Risk From Passive Smoking. Environment International 11: 3-22 (1985).

24. Repace JL, and Lowrey AH. An enforceable indoor air quality standard for environmental tobacco smoke in the workplace. RISK ANALYSIS, 13:463-475 (1993).

25. Repace JL, and Lowrey AH. An Indoor Air Quality Standard For Ambient Tobacco Smoke based on Carcinogenic risk. N.Y. STATE JOURNAL OF MEDICINE: 85: 381-383 (1985b).

26. Repace JL, and Lowrey AH. Indoor Air Pollution, Tobacco Smoke, and Public Health. SCIENCE 208: 464-474 (1980).

27. Repace JL, and Lowrey AH. Tobacco Smoke, Ventilation, and Indoor Air Quality. ASHRAE TRANSACTIONS 88: Part I, 895 (1982).

28. Repace JL, Jinot J, Bayard S, Emmons K, and Hammond SK. Air nicotine and saliva cotinine as indicators of passive smoking exposure and risk. Risk Analysis 18: 71-83 (1998).

29. Repace JL, Lowrey AH. Is the dose-response curve between tobacco smoke exposure and lung cancer really linear from active smoking to passive smoking? ENVIRONMENT INTERNATIONAL 18: 427-429 (1992).

30. Repace JL. Effects of passive smoking on coronary circulation. (letter) JAMA. 2002; 287 #3, January 16.

31. Repace JL. Indoor concentrations of environmental tobacco smoke: models dealing with effects of ventilation and room size", Ch. 3, IARC Scientific Publications no.81, Environmental Carcinogens – Selected Methods of Analysis – Volume 9 Passive Smoking; I.K. O'Neill, K.D. Brunnemann, B. Dodet & D. Hoffmann, International Agency for Research on Cancer, World, Health Organization, United Nations Environment Programme, Lyon, France, (1987).

32. Repace JL. Indoor concentrations of environmental tobacco smoke: field surveys. Ch. 10, IARC Scientific Publications no. 81, Environmental Carcinogens – Selected Methods of Analysis – Volume 9 Passive Smoking; I.K. O'Neill, K.D. Brunnemann, B. Dodet & D. Hoffman, International Agency for Research on Cancer, World, Health Organization, United Nations Environment Programme, Lyon, France, (1987).

33. Repace JL. Ott WR, Klepeis NE. Indoor Air Pollution from Cigar Smoke. JL. In Smoking and Tobacco Control Monograph 9. Cigars – Health Effects and Trends. National Institutes of Health, National Cancer Institute, Bethesda, MD (1998).

34. Sandler D et al., Lancet, Feb 9, 1985 312-315.

35. Scollo M, Glantz SA. Review of the quality of studies on the economic effects of smoke free policies on the hospitality industry. Tobacco Control 2003; 12: 13-20.

36. SCOTH. Report of the Scientific Committee on Tobacco and Health, UK Department of Health The Stationery Office, 1998.

37. Surgeon General: Smoking and Health, a Report of the Surgeon General. U.S Dept. of Health, Education and Welfare, Public Health Service. 1979.

38. Surgeon General. The Health Consequences of Involuntary Smoking, A Report of the Surgeon General. U.S. Dept. of Health and Human Services, Washington, DC (1986).

39. Tunstall-Pedoe H, Brown CA, Woodward M, Tavendale R. Passive smoking by self-report and serum cotinine and the prevalence of respiratory and coronary heart disease in the Scottish heart study. J Epidemiol and Comm Health 49: 139-143 (1995).

40. U.S. Department of Health and Human Services. A report of the Surgeon General. Reducing the health consequences of smoking. Public Health Service, Washington, DC, 1989.

41. U.S. Dept. of Labor, Occupational Safety & Health Administration. 29 CFR Parts 1910, 1915, 1926, and 1928 Indoor air quality, proposed rule Fed Reg 59 # 65, Tues April 5, 1994, 15968-16039.

42. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Office of Research and Development. Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders. Report No. EPA/600/6-90/006F. Washington, DC, 1992.

43. U.S. EPA, Health Effects of Passive Smoking: Assessment of Lung Cancer in Adults, and Respiratory Disorders in Children. EPA/600/6-90/006F, December (1992).

44. U.S. EPA, Office of Air and Radiation. EPA's Revised Particulate Matter Standards. Fact Sheet, Office of Air Quality Planning & Standards, July 17, 1997.