Mr James Repace's responses to questions raised by Hon Tommy CHEUNG at the meeting of the Bills Committee on Smoking (Public Health) (Amendment) Bill 2005 on 24 October 2005

Question 1

(a) Whether you had done any studies on the number of people contracting lung cancer before and after the implementation of total smoking ban in catering and entertainment premises in the United States and other places; if not, why not, and what was your assessment on this issue?

J.L. Repace's Response:
I have estimated the number of lung cancer deaths per year in the United States at 5,000 ± 2500 (Repace and Lowrey, 1985; 1990). The portion of these excess deaths due to workplace exposures would be eliminated after workplace smoking bans. I have also estimated the number of excess lung cancer and heart disease deaths combined due to passive smoking among U.S. office workers only as 4400 per year (4000 from heart disease and 400 from lung cancer) at a 28% workplace exposure prevalence (Repace et al., 1998). All of these premature deaths would be prevented after an office workplace smoking ban. Exposure prevalence and magnitude is greater in hospitality venues than in offices. With respect to other places, I have estimated (Repace, 2003) that, overall, 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, I estimated that 165 bar workers died every year in the U.K. from passive smoking, based upon cotinine measurements in London barstaff. This report is attached **(Appendix I)**.

Question 2

(b) What was the basis for saying that the cost of passive-smoking mortality was 150 deaths per year among 200,000 catering workers in the United States, and what was the relevance of applying such figures to Hong Kong?

J.L. Repace's Response:
The basis for the Hong Kong estimates was Hong Kong COSH Report #8 **(Appendix II)**, of which I am a co-author. It was not based on U.S. catering workers, but actual measurements of cotinine, a biomarker for passive smoking in Hong Kong catering workers.

Question 3

(c) What was the basis for saying that an average worker had 3000/100,000 lifetime risk, a de minimus risk level was one death/1,000,000 persons and ventilation must be increased to 81,000 air changes per hour to yield acceptable risk for Hong Kong catering workers from secondhand smoke?

J.L. Repace's Response:
The quantitative basis for this estimate is a recent paper published by the American Society of Heating, Refrigerating and Ventilating Engineers, in their journal, ASHRAE IAQ Applications. This estimate was adjusted downward to account for the lower prevalence of heart disease mortality in Hong Kong than in the U.S. This report is attached **(Appendix III)**.

Other references provided by Mr Repace

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

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 (1985).

Repace JL, and Lowrey AH. Risk Assessment Methodologies in passive smoking-induced lung cancer. RISK ANALYSIS, 10: 27-37, (1990)

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).

Appendix I

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

Report written by James Repace, MSc. Health Physicist

James Repace, MSc., is a health physicist and an international secondhand smoke consultant who has published 60 scientific papers on the hazard, exposure, dose, risk, and control of secondhand smoke. He has received numerous national honours, including the Flight Attendant Medical Research Institute Distinguished Professor Award, the Robert Wood Johnson Foundation Innovator Award, the Surgeon General's Medallion, and a Lifetime Achievement Award from the American Public Health Association. He holds an appointment as a Visiting Assistant Clinical Professor at the Tufts University School of Medicine. He is a former senior policy analyst and scientist with the U.S. Environmental Protection Agency, serving on both the Air Policy and Indoor Air Staffs, Office of Air and Radiation, and in the Exposure Analysis Division, Office of Research and Development. He served as a consultant to the Occupational Safety and Health Administration, U.S. Department of Labor, on its proposed rule to regulate secondhand smoke and indoor air quality. He was also a research physicist at the Naval Research Laboratory in the Ocean Sciences and Electronics Divisions.

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.

Action on Smoking and Health
102 Clifton Street
London
EC2A 4HW
Tel: 020 7739 5902
www.ash.org.uk

ISBN 1 872428 59 2
Published 9 April 2003

Design bwa design 0207 490 3148

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).

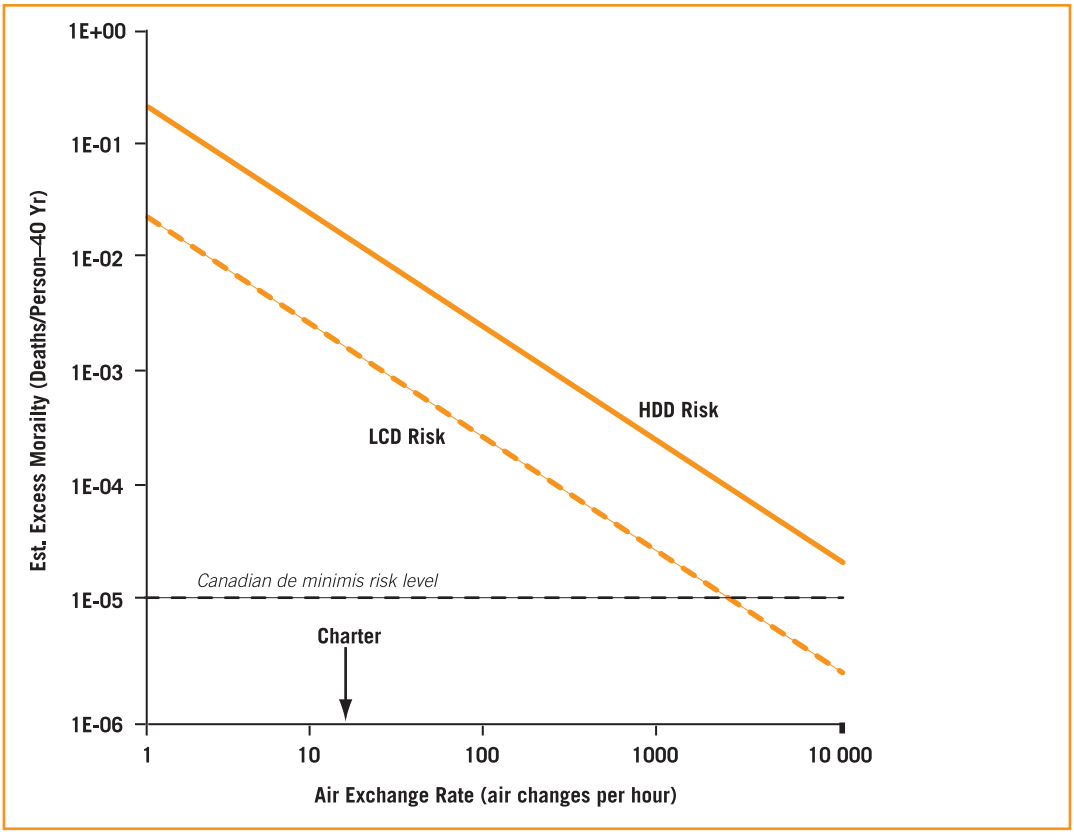


Figure 1
Working Lifetime ETS Risk in Bars

Bar workers' estimated lung cancer and heart disease death risk vs. Ventilation or Air Cleaning Rate at 50% smoking prevalence and 50% occupancy (50 occupants per 100 m^2, 3 metre ceiling). The arrow shows the charter-specified ventilation rate of 12 air changes per hour.

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).

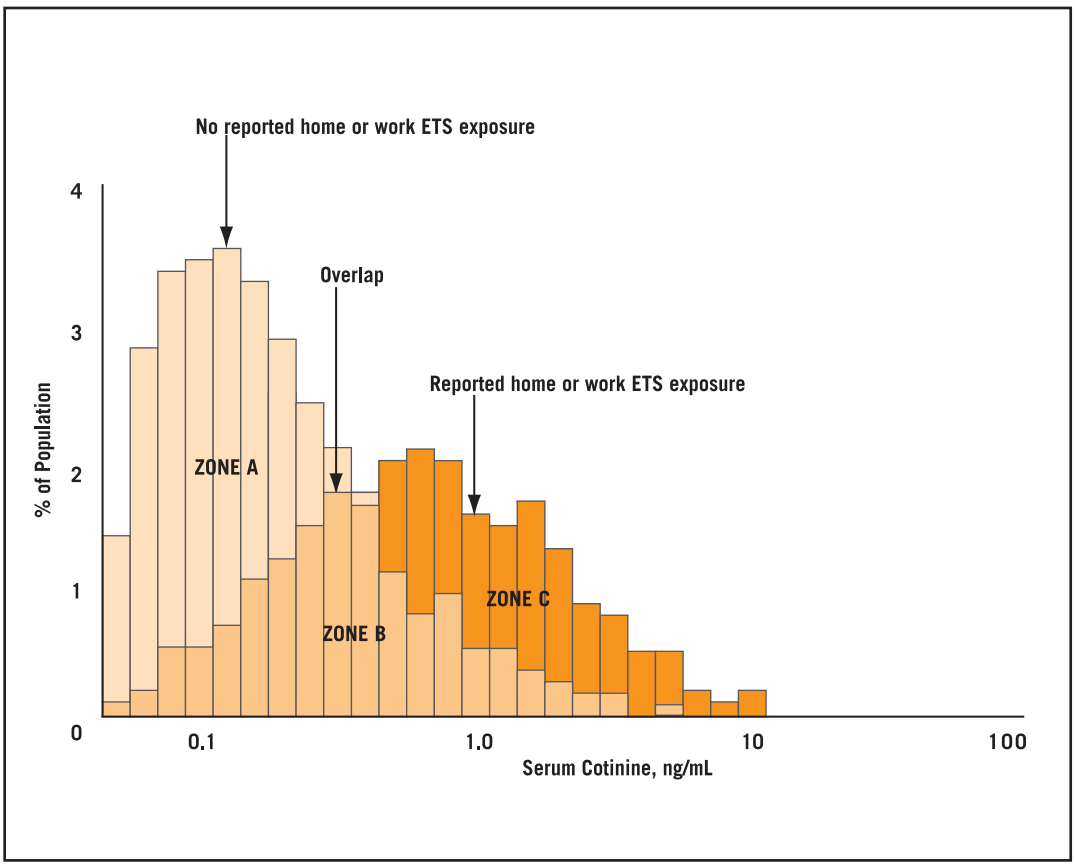


Figure 2
NHANES III Distribution of Cotinine in U.S. Population ≥ 4 years of age
Adapted from The National Health & Nutrition Examination Study III (NHANES III).

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.

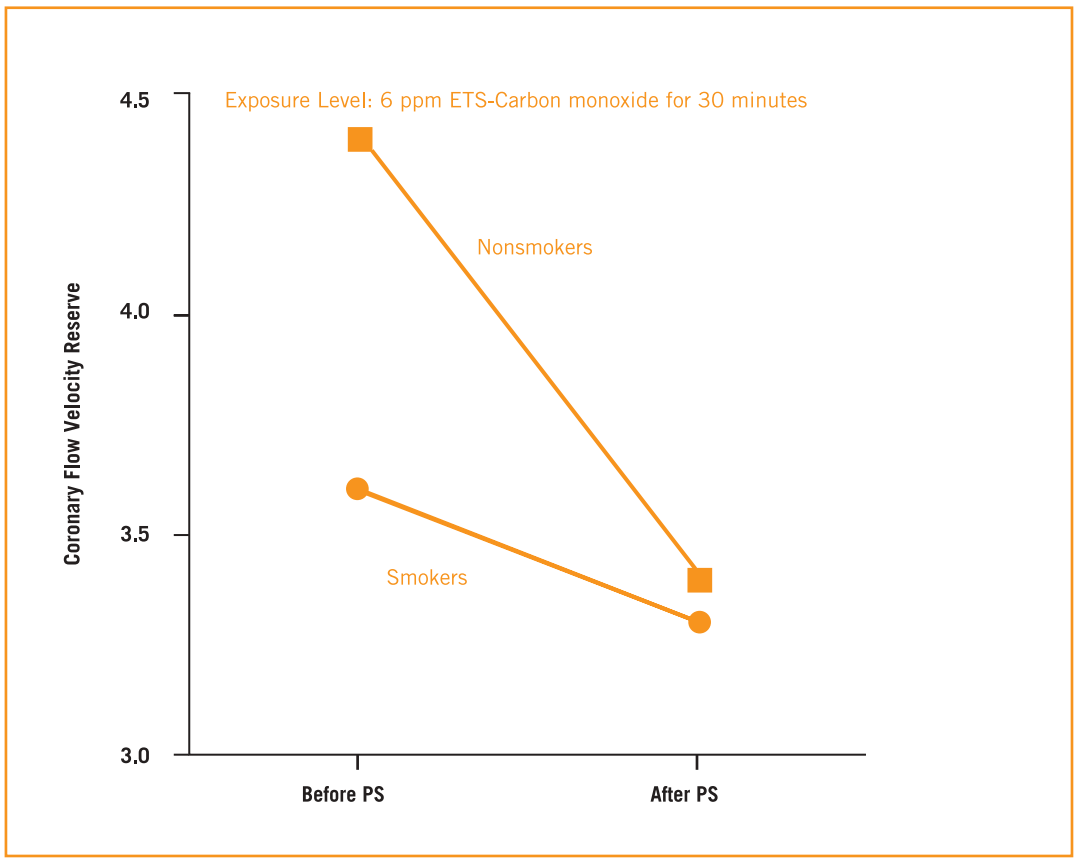


Figure 3
Acute Effects of Passive Smoking on Coronary Circulation In Healthy Young Adults
(Otsuka et al. JAMA 2001; 286436-411)

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.

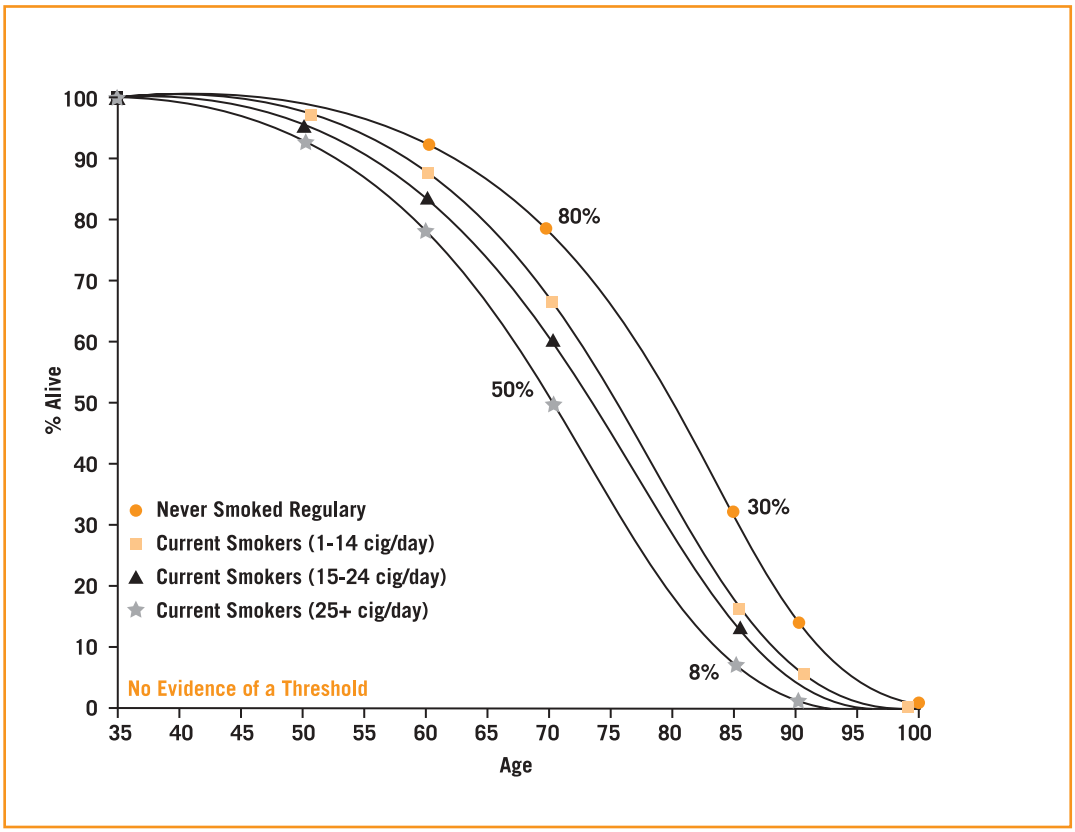
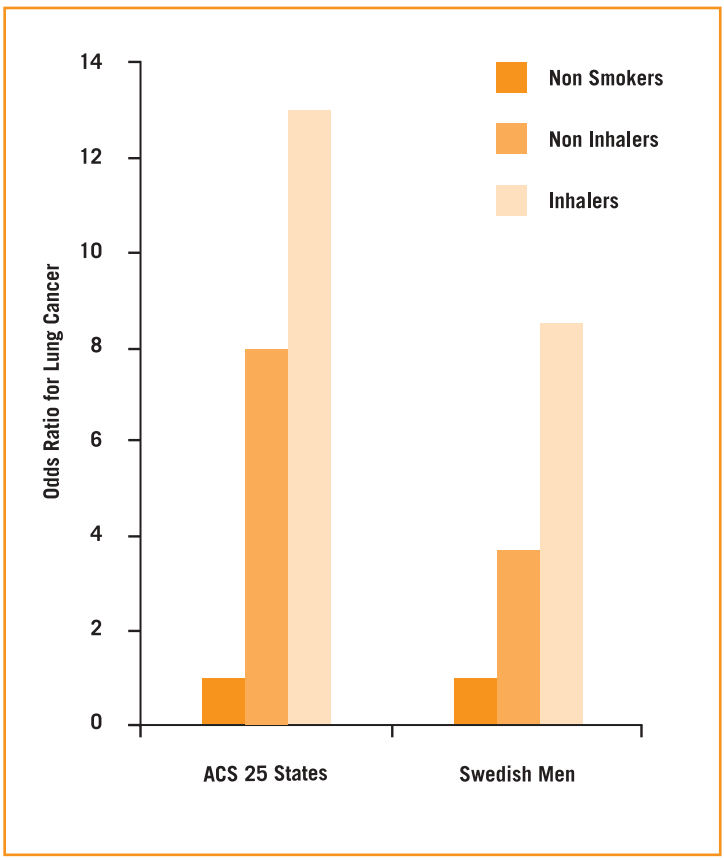
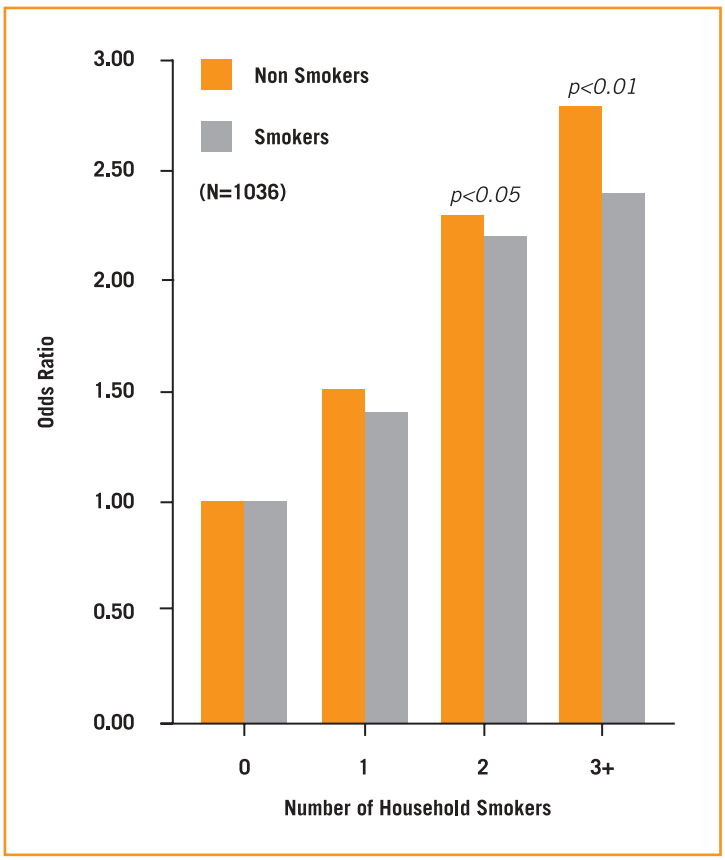


Figure 4 (left)
U.K. male doctors study: 40 yrs, 1951-1991. Effects of cigarette smoking on survival
This 40 year-long study of survival as a function of age shows that no matter how long a person lives, tobacco smoke exposure causes a loss of life expectancy.

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).





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).

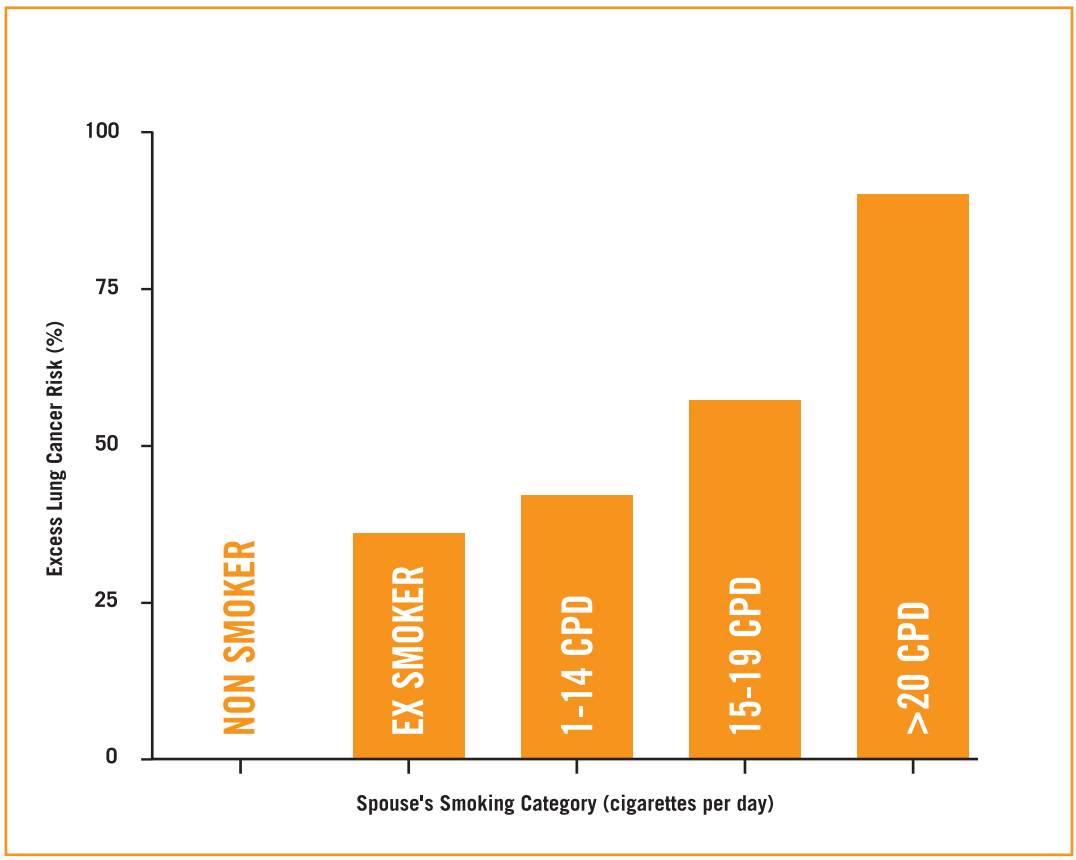


Figure 6
Exposure-response between lung cancer and spouse's smoking rate
(Hirayama T., Proc. 5th World Conf. Smoking & Health, 1983)

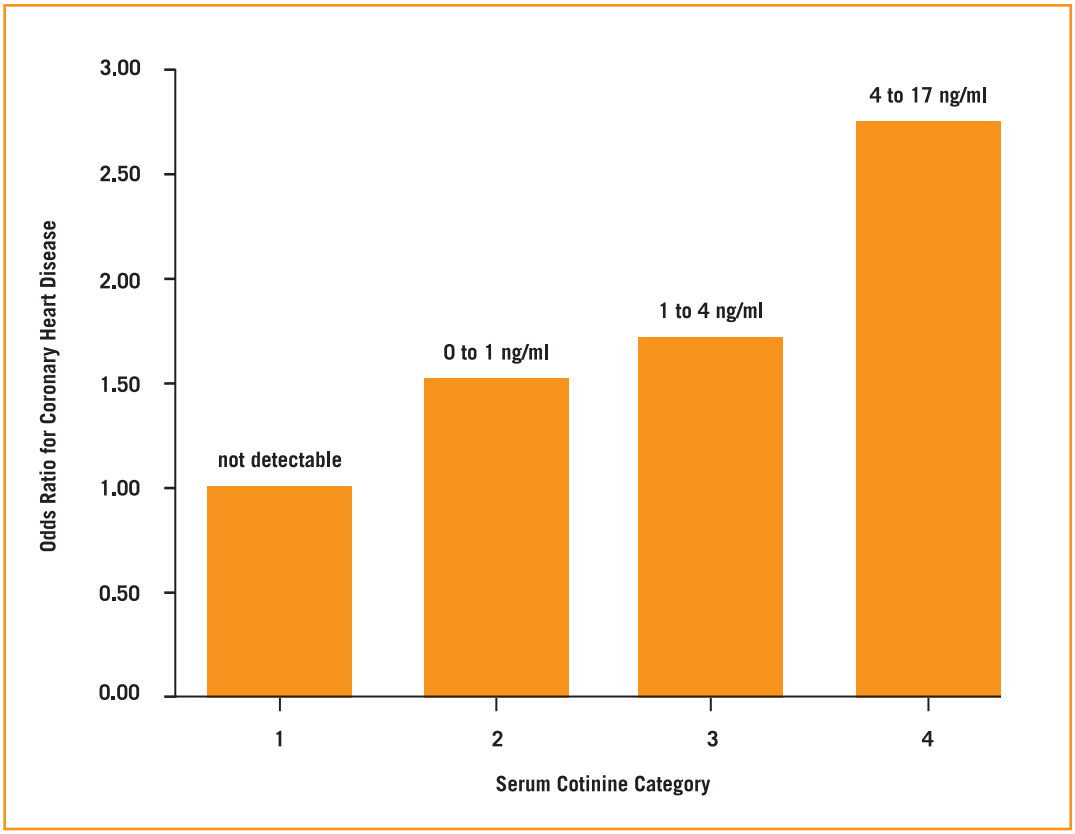


Figure 7
Dose-response for passive smoking in the Scottish Heart Study
(Tunstall-Pedoe, et al., J.Epidemiol and Comm Health 49: 139-143, 1995)

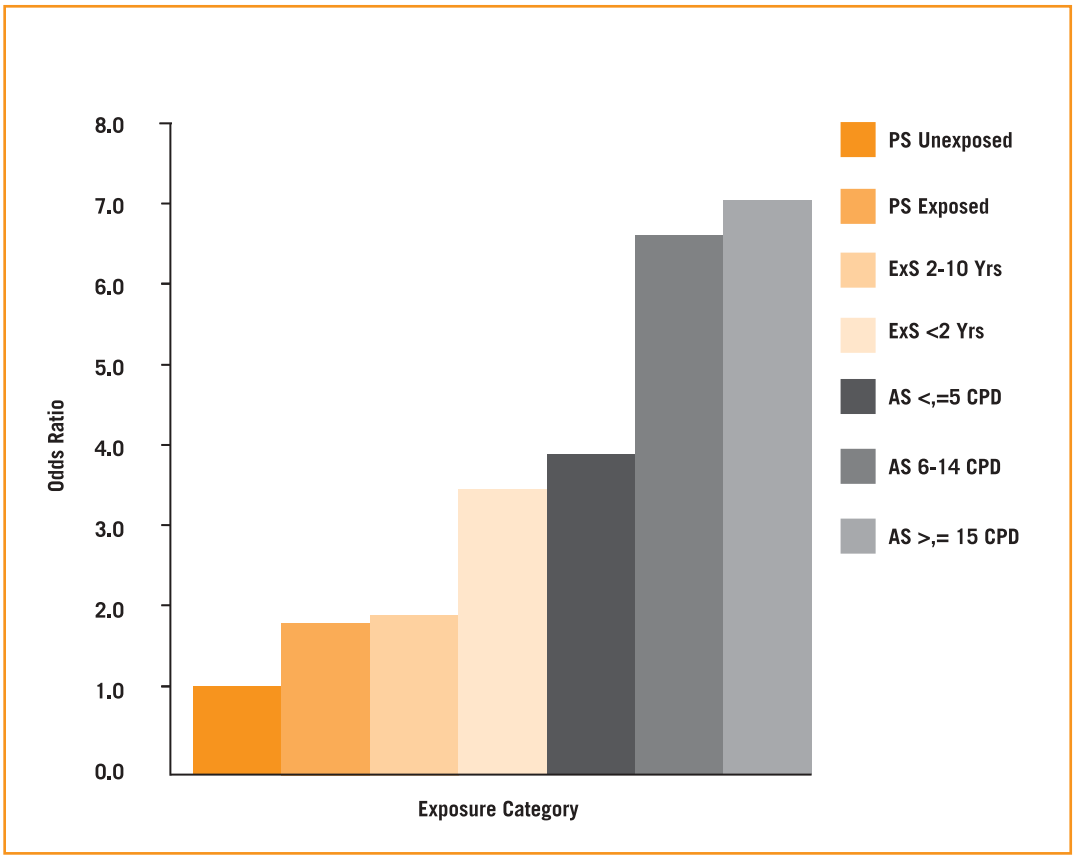


Figure 8
Relative risk for stroke increases as tobacco smoke exposure increases
(Bonita, et al., Tobacco Control 8:156-160, 1999)

PS = passive smoking category
ExS = ex-smoker category
AS = active smoker category.
Passive smoking increases risk of stroke by 82% on average.

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.

Table 1
Annual Deaths in the U.K. based on U.S. Estimates
Estimated Passive Smoking Deaths
(U.S. values from AJ Wells, Env. Internat. 25:515-519, 1999)
(U.K. deaths scaled from U.S. deaths by relative population J.L. Repace)

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

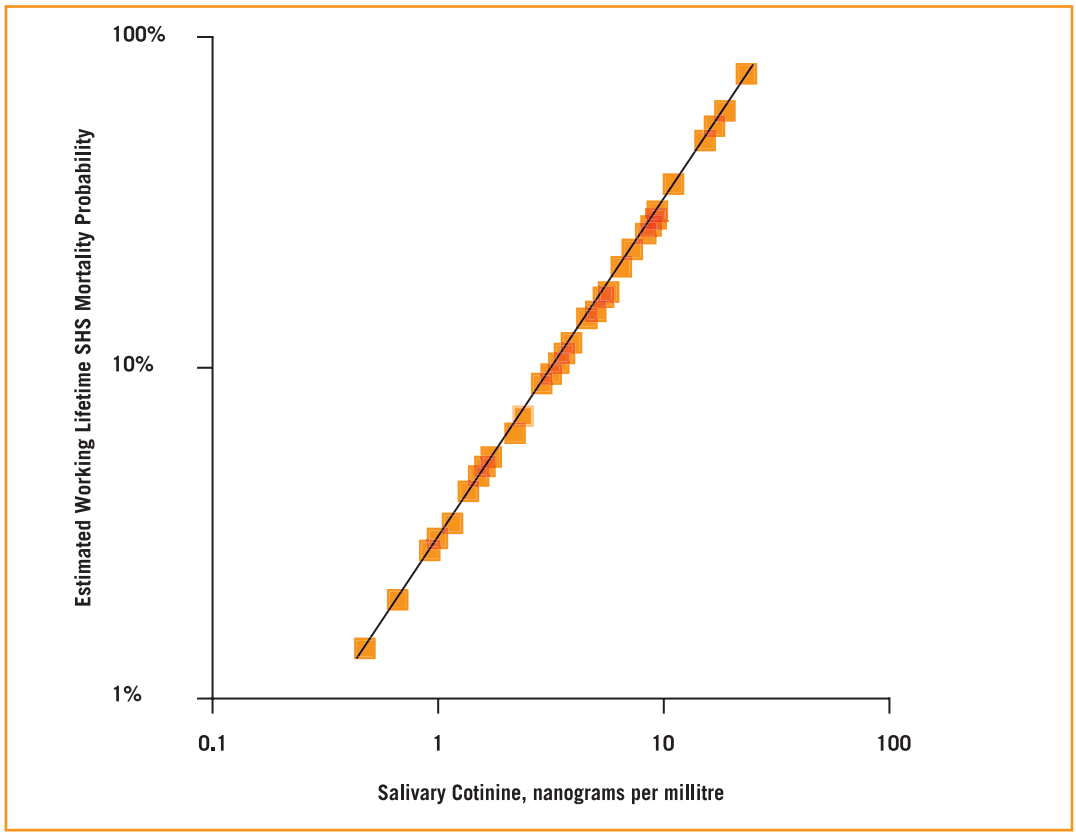


Figure 9
London Bar Staff:
Salivary Cotinine vs. SHS Risk
(Cotinine data: Jarvis, 2001)

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

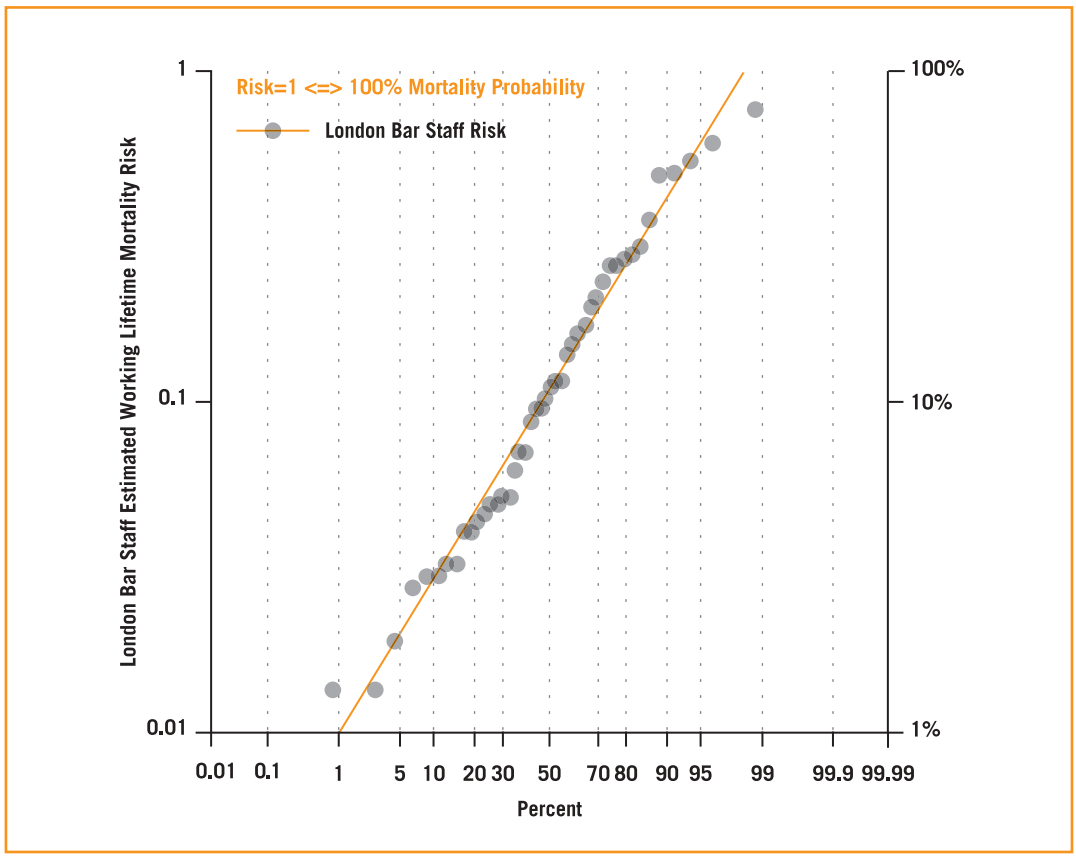


Figure 10
Estimated SHS risk of London Bar Staff by Percentile
(Data: Jarvis 2001)

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

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

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 (µg/m³) 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 (µg/m³).

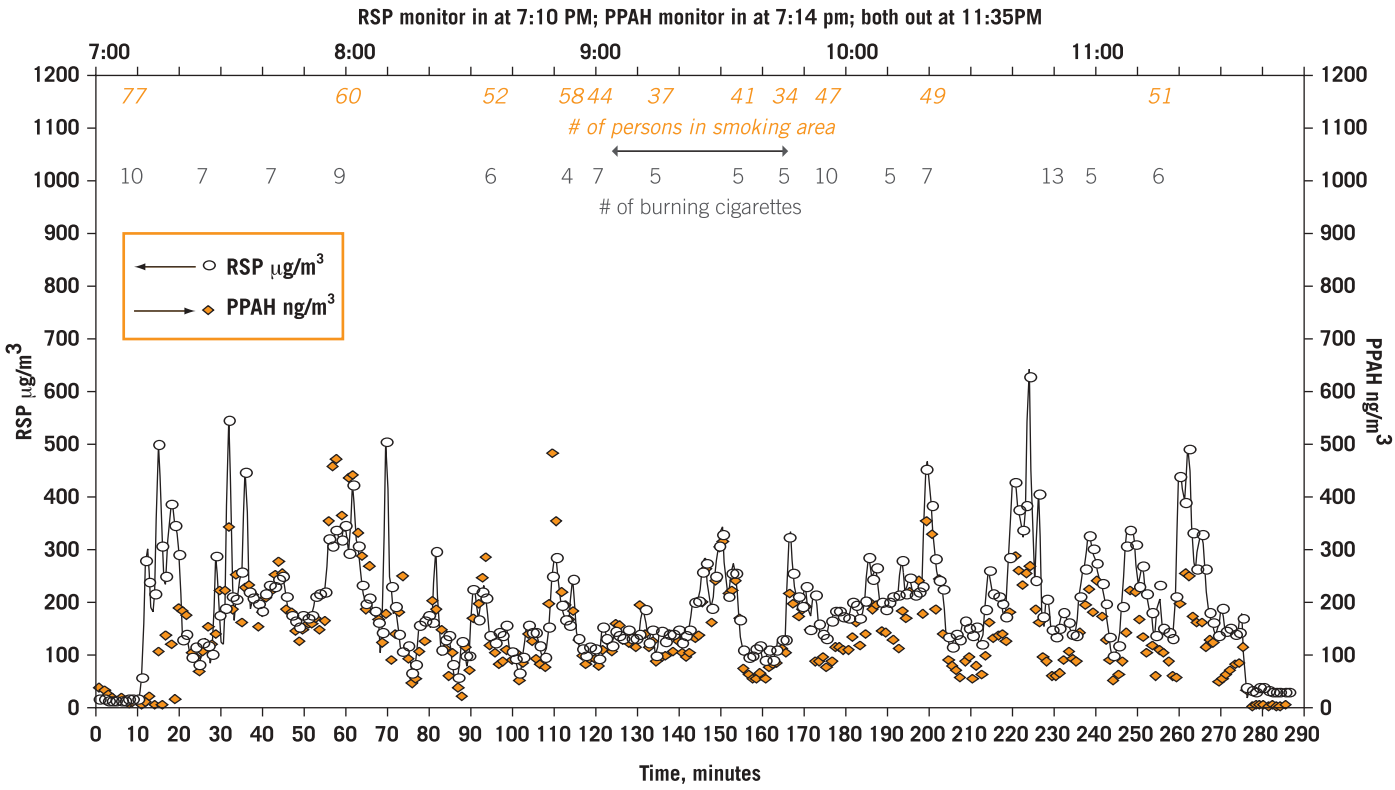
Thus, the estimated salivary cotinine level for bar staff in a Charter-ventilated bar would be S = (0.0057)(345) = 1.97 ng/ml. From Equation 2, this yields an estimated Risk = 0.0275 S = (0.0275)(1.97) = 5%. In other words, at the Charter-recommended ventilation rate for a pub at full occupancy, an estimated 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 µg/m³. The estimated level of 345 µg/m³ of RSP ($PM_{3.5}$) for an eight hour work shift averages out to (8/24)(345) = 115 µg/m³ over a 24-hour period. Assuming the outdoor background is in compliance with the annual NAQS of 40 µg/m³ 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 m³ Toronto pub ventilated at 8.6 h^{-1}, with a 2.9 m ceiling, with 46 persons per 100 m² occupancy, and a 42% smoking prevalence, conditions similar to a Charter-compliant U.K. pub. The average indoor RSP level was 199 µg/m³ 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/m³, while the outdoor averaged 8 ng/m³, 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 µg/m³ (11% of the indoor value) yielding 177 µg/m³. This is adjusted to the Charter defaults for smoking prevalence, occupancy, air exchange, and volume as follows: (177 µg/m³)(47%/42%)(100 P/46 P)(8.6 h^{-1}/12 h^{-1})(295 m³/250 m³) = 364 µg/m³, within 6% of the 345 µg/m³ 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.



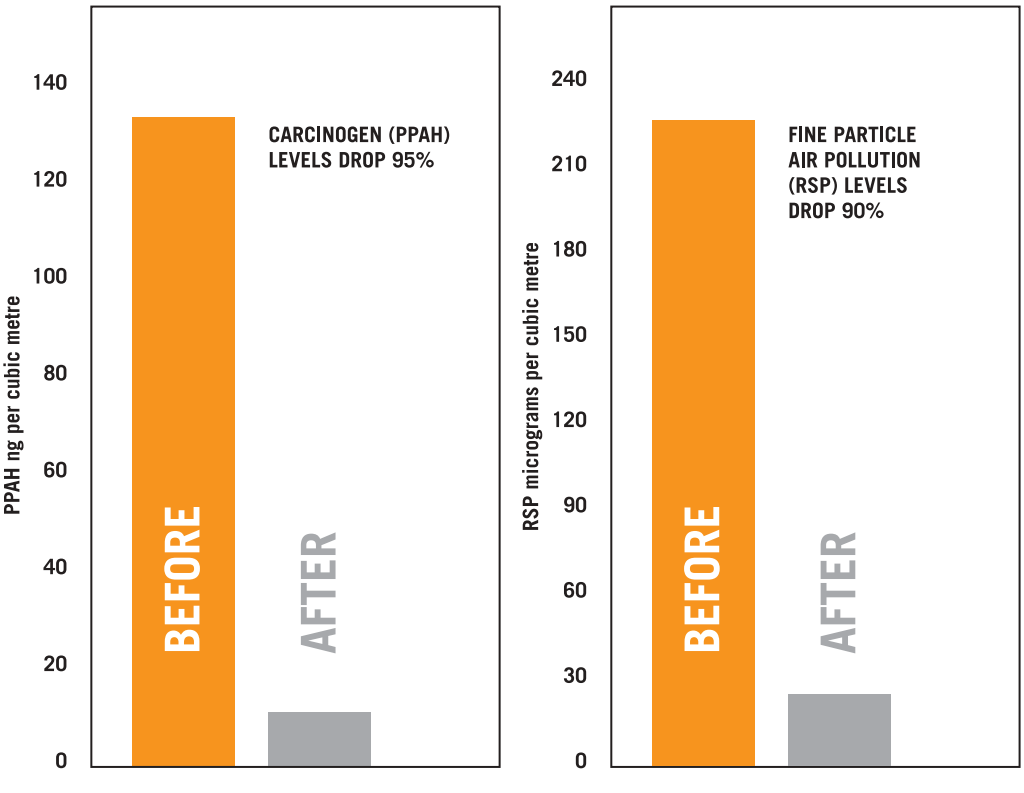


Figure 12 (above)
Metropolitan Toronto, Canada Pub: RSP & PPAH vs Time, Friday Dec 13 2002
(J.L. Repace unpublished)

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).

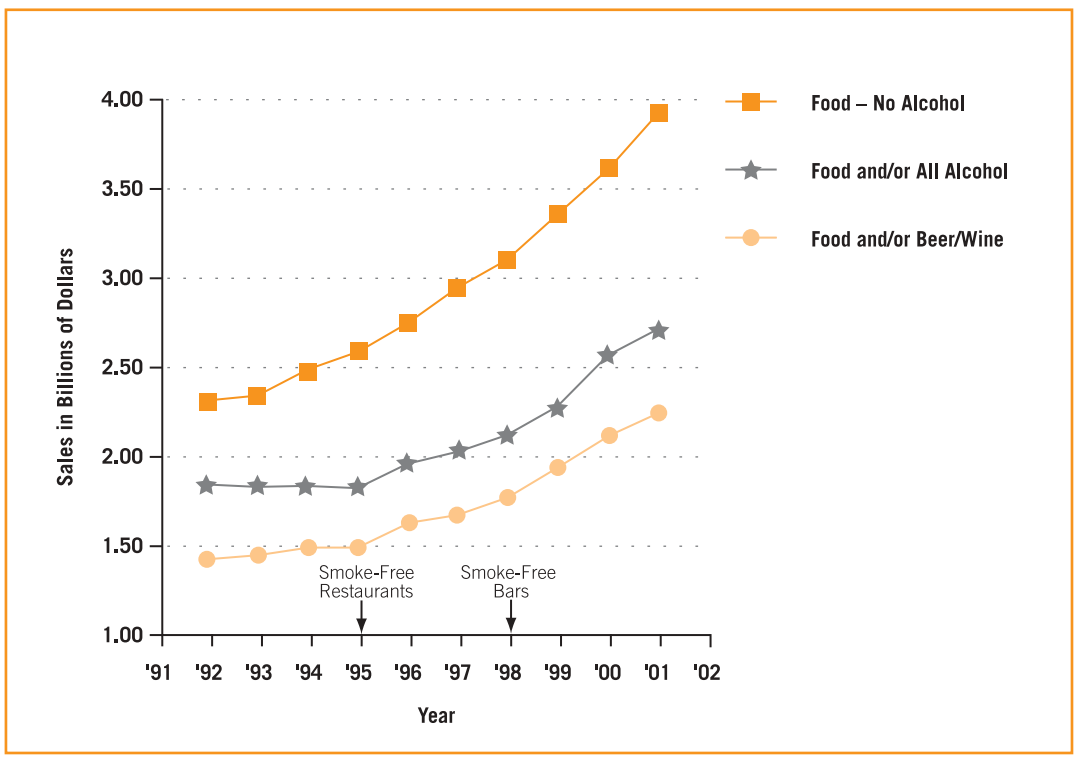


Figure 14
First quarter taxable sales figures for California restaurants and bars, State of California, '92-'01
(Source: California Dept. of Health; California Board of Equalization)

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, Cinncinati, 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 (l980).

27. Repace JL, and Lowrey AH. Tobacco Smoke, Ventilation, and Indoor Air Quality. ASHRAE TRANSACTIONS 88: Part I, 895 (l982).

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).

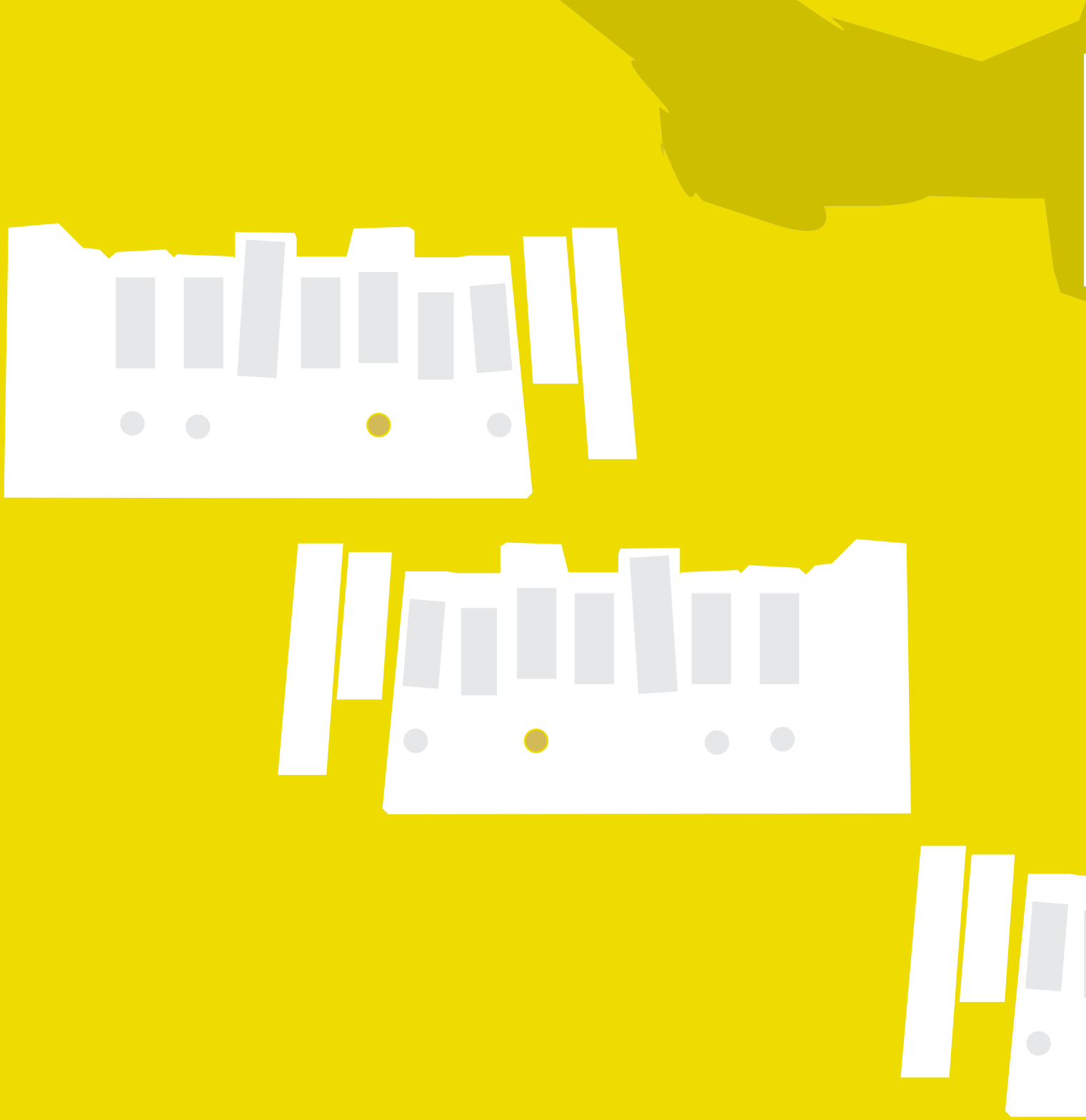
40. U.S. Department of Health and Human Services. A report of the Surgeon General. Reducing the health consequences of smoking. Public Health Service, Washington, DC, 1989.

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.



ash.
action on smoking and health

C O S H

HONG KONG COUNCIL ON SMOKING AND HEALTH

Passive smoking and risks for heart disease and cancer in Hong Kong catering workers

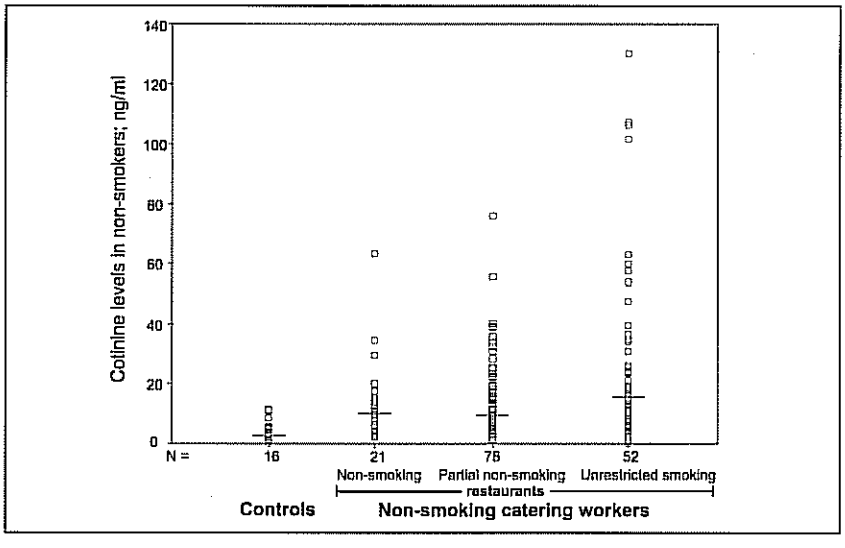
2 0 0 1

May 2001 Report No. 8

Second-hand smoke exposures and passive smoking in non-smoking catering workers in Hong Kong: the combined risks for heart disease and cancer

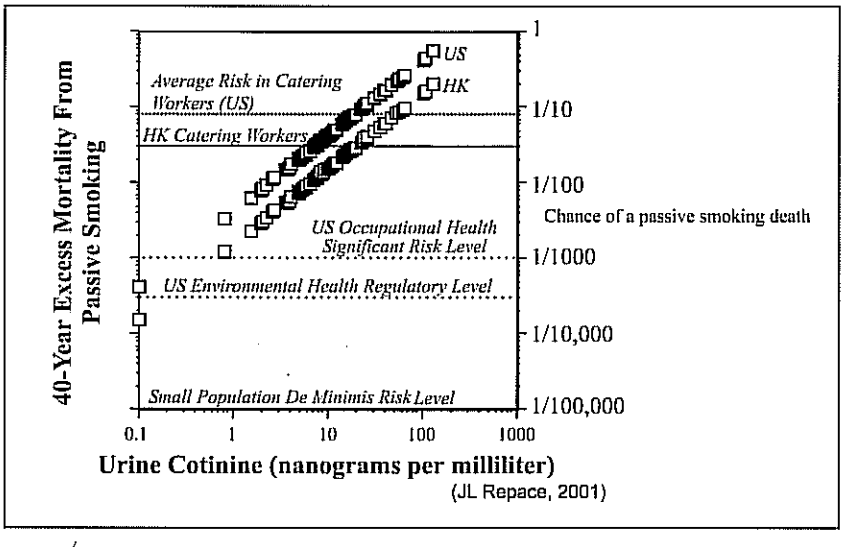
AJ Hedley, SM McGhee, J Repace, TW Wong, MYS Yu, AYW Chan, TH Lam,
PCK Lo, M Tsang, LC Wong, ALN Chan, ESL Ng, M Janghorbani
Hong Kong Council on Smoking and Health; Department of Community Medicine, University of Hong Kong;
Department of Community and Family Medicine, Chinese University of Hong Kong, and
Repace Associates Inc, Bowie MD (http://www.repace.com/), USA

Urine cotinine levels in non-smoking catering workers and controls



104 catering workers, exposed to second-hand smoke only at work

Working lifetime combined risk from fatal heart disease & lung cancer based on both US and Hong Kong mortality rates and Hong Kong exposures to passive smoking



1. Background

Second-hand smoke and passive smoking: Passive smoking results from non-smokers breathing air which is contaminated with second-hand smoke made up of *mainstream smoke* exhaled by smokers and *side-stream smoke* emitted from the tips of burning cigarettes and cigars. Second-hand smoke is extremely poisonous; it contains over 4000 chemicals in the form of particles and gases.

Health hazards: Exposures to second-hand smoke are the cause of many health problems in non-smokers. These include extreme irritation to mucous membranes in the eyes, nose and throat; chronic respiratory symptoms such as cough, phlegm and wheeze and exacerbations of asthma. Asthmatics experience a decline in lung function when exposed to second-hand smoke. Passive smoking also causes damage to blood vessels so that non-smokers are at increased risk of heart attacks and stroke. Passive smoking is a hazard to the health of pregnant women and the foetus. Children are extremely sensitive to second-hand smoke and those with passive smoking exposures have more health problems including middle ear disease, bronchitic symptoms, acute chest infections and emergency admissions to hospital.

Second-hand smoke contains a high concentration of carbon monoxide which is implicated as one cause of heart disease in smokers. Tobacco smoke also increases platelet aggregation and causes changes in blood clotting mechanisms. Cancer causing compounds in second-hand smoke are inhaled and pass into the circulation. Exposure of non-smokers to tobacco smoke leads to increased blood and urinary concentrations of tobacco-specific cancer causing substances.

The US Environmental Protection Agency (EPA) and the UK Government Department of Health Scientific Committee on Tobacco and Health (SCOTH) and many other national and international agencies accept the evidence that exposures to passive smoking cause lung cancer and conclude that second-hand smoke is a *proven human carcinogen.*

No safe threshold: In terms of its cancer inducing potential there is no known safe level of second-hand smoke. Neither simple measures designed to separate smokers from non-smokers nor ventilation engineering will prevent passive smoking when a common air space is contaminated with tobacco smoke.

In 1999 the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) eliminated all reference to any level of smoking being permissible from the ANSI/ASHRAE indoor air quality standards. This standard now makes it clear that the governing standard (ANSI/ASHRAE 62-1989) is based on a totally non-smoking environment.

The prevention of passive smoking: There is increasing awareness and acceptance in Hong Kong, on the part of the Government and the general public, that effective controls are needed to prevent involuntary passive smoking in all public places and in the workplace. At the present time very few indoor places meet the necessary criteria to ensure that the public and the workforce are protected against second-hand smoke exposures.

One major deficiency in the present legislation concerns the catering industry where there is no protection for most customers and none at all for workers. The requirement for restaurants with 200 or more seats to offer one third of seating in "smoke-free" sections is a token arrangement which cannot meet even the minimum criteria and public health requirements for a smoke-free indoor environment.

Two previous reports on Hong Kong public opinion by COSH, in 1995 and 2000, showed that the overwhelming majority of the public wanted smoke-free dining facilities and that patrons would eat out more often given assurances of smoke-free facilities; in other words it would be good for business. A large proportion of the customers in these surveys frequently experienced adverse exposures to second-hand smoke including foul odour, contamination of clothes and hair, irritation of eyes, nose and throat, and asthma/ wheezing or other respiratory problems. Over one third formed an unfavourable impression of the restaurants concerned and considered taking their patronage elsewhere.

Two previous studies in Hong Kong have shown that passive smoking in the workplace is a major cause of chronic respiratory problems in Hong Kong. This report examines the preliminary results of a new survey of non-smoking workers in the catering industry, which aimed to assess their passive smoking exposures in different work settings and their risks for heart disease and cancer.

2. Objectives

The objectives of this pilot study were to

- document workplace and other exposures to second-hand tobacco smoke in non-smoking catering workers
- collect and analyse urine samples for cotinine which is a breakdown product of nicotine and an indicator of passive smoking in non-smokers
- estimate the combined working-lifetime risks for heart disease and lung cancer in Hong Kong catering workers.

3. Subjects and Methods

Subjects: A total of one hundred and eighty four catering workers were recruited to the study and 165 provided complete data on exposures to second-hand smoke. All were volunteers, invited on the basis that they were non-smokers but any smokers who wished to participate were accepted. They received $100-$150 (including travel expenses) for their participation. They were asked to complete an interview schedule and give a 50 ml sample of urine. All subjects were tested using a monitor to detect carbon monoxide in their breath (expired air). Carbon monoxide levels in human breath are usually less than 10 parts per million (ppm) in non-smoking subjects. Fourteen subjects were found to be (or declared that they were) occasional or regular smokers and 170 (83 male and 87 female) were non-smokers. Seven subjects were found to be *regular smokers* either because of self-declaration or raised breath carbon monoxide (>9 ppm) and seven more admitted to being *occasional smokers*, defined as using less than 7 cigarettes per week. Their results are included in the findings for comparison with the other groups (Table 1). The majority (86%) of workers were employed in restaurants which permitted smoking. The remainder were from catering facilities which did not permit any smoking by customers (Table 2).

An additional sample group of 16 control subjects were recruited, being physicians, nurses or university researchers. All were non-smokers who worked in a smoke-free workplace and who generally avoided smoky environments.

Table 1: Catering workers and urinary cotinine levels by exposure to second-hand smoke at work, home and leisure activities

Subjects	Exposure outside work	Non-customer exposure	Mean	N	SD	Range
Controls						
	no exposure outside work	nil	3.3	13	3.5	0-11.2
	home or leisure exposure	nil	5.5	3	4.9	1.1-10.8
	Total	nil	3.7	16	3.7	
Worker in non-smoking restaurant						
	no exposure outside work	nil	6.4	3	6.6	2.6-14.0
		other staff or break	14.0	10	17.7	2.2-62.9
		Total	**12.3**	**13**	**15.9**	
	home or leisure exposure	nil	20.3	5	11.9	3.9-34.1
		other staff or break	9.9	3	3.9	5.8-13.6
		Total	**16.4**	**8**	**10.7**	
	Total	nil	15.1	8	12.0	
		other staff or break	13.1	13	15.5	
		Total	**13.8**	**21**	**14.0**	
Worker in partial smoking restaurant						
	no exposure outside work	nil	6.1	6	6.4	1.5-18.6
		other staff or break	14.3	50	10.8	2.0-55.3
		Total	13.4	56	10.7	
	home or leisure exposure	nil	7.1	1		7.1
		other staff or break	16.6	21	17.2	1.0-76.4
		Total	16.2	22	17.0	
	Total	nil	6.3	7	5.8	
		other staff or break	14.9	71	13.0	
		Total	14.2	78	12.7	
Workers in unrestricted smoking restaurant						
	no exposure outside work	nil	15.9	4	6.5	7.6-23.1
		other staff or break	28.7	34	33.9	0-129.4
		Total	**27.4**	**38**	**32.3**	
	home or leisure exposure	nil	26.5	3	10.5	14.7-34.6
		other staff or break	20.0	11	21.9	0.03-62.3
		Total	**21.4**	**14**	**19.8**	
	Total	nil	20.4	7	9.5	
		other staff or break	26.6	45	31.4	
		Total	**25.7**	**52**	**29.4**	
Occasional smoker						
	no exposure outside work	other staff or break	145.0	6	118.4	2.2-286.8
	home or leisure exposure	other staff or break	881.4	1		
	Total	**other staff or break**	**250.2**	**7**	**298.6**	
Regular smoker						
	no exposure outside work	other staff or break	2996.3	3	1695.0	1281-4671
	home or leisure exposure	other staff or break	4034.0	4	1274.1	
	Total	**other staff or break**	**3589.2**	**7**	**1441.2**	

Table 2: Number (%) of non-smoking workers by type of catering facility

Non-smoking restaurants			24
Fast-food	22		
Western/Eastern	1		
Canteen	1		
Smoking restaurants			146
Chinese restaurants	70	(41.2)	
Cha Charn Ting	31	(18.2)	
Fast food shop	6	(3.5)	
Western/Eastern	8	(4.7)	
Club/canteen/caf	31	(18.2)	
Total			170

Cotinine: When nicotine in tobacco smoke is absorbed into the circulation it undergoes metabolic breakdown in the liver into other compounds, including *cotinine* which can be measured in blood, saliva and urine. In this way it can be used as a marker of exposure to the toxic components of second-hand smoke in non-smokers who become passive smokers. The urinary cotinine levels of all workers and the controls in this survey were measured by the MetLife Laboratory in New York (Dr N Haley). The cotinine values are expressed as nanograms (ng) per milliliter of urine.

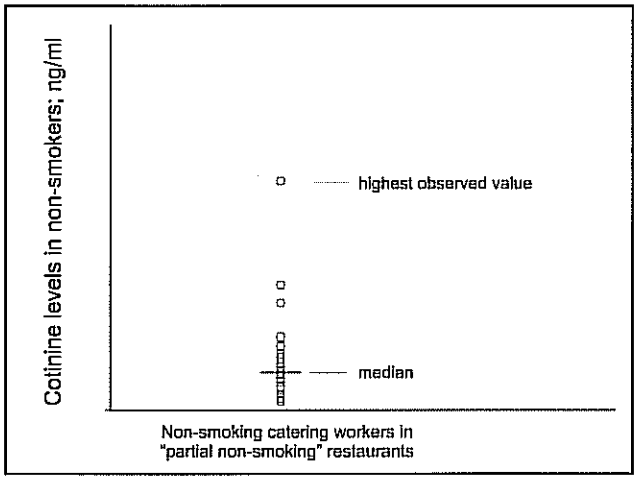
Interview: In addition to basic demographic information, workers were asked about workplace, home and leisure exposures to tobacco smoke. The numbers and proximity to them of smokers in their workplace were documented whenever possible. The workers' past active smoking history was recorded when relevant and the time since quitting was recorded. Finally questions about respiratory and cardiovascular health, including diagnoses and current symptoms were included.

Analyses: *Urinary cotinine levels* were analysed by main groups and sub-groups, defined by their worker or control status, workplace type and reported exposures to tobacco smoke from any source.

The classification of subjects has initially been carried out on an *a priori* basis using their criteria for selection (ie "control", or "catering worker") or their place of work (ie "non-smoking" or "smoking" catering facilities).

These findings have been further explored by subgroups, including "non-waiter" (eg accounts clerks, housekeepers, chefs, others), and "waiter" (anyone serving tables as waiter or senior restaurant supervisors). Exposures have been examined by the workers' declarations of "other exposures" including staff smoking, exposure during rest times, home and leisure activities.

The graphics for the urinary cotinine values are presented as dot charts as shown in the example below. Each dot represents an individual cotinine value within the group tested; the lowest and highest dot indicate the range and the horizontal bar is the median or middle value. The cotinine values are measured as nanograms per milliliter (ng/ml).



The risk of heart disease and lung cancer in this sample of Hong Kong catering workers who are exposed to second-hand smoke has been estimated using a pharmacokinetic risk model developed by Repace and his co-workers. This enables cotinine levels in urine, saliva and plasma to be related to lung and heart disease in passive smokers. The risk is calculated for a 40 year working life time (WLT_{40}). Using this model Repace and Lowrey associated an average plasma cotinine of 0.4 ng/ml with a WLT_{40} increased mortality for lung cancer of 1 in 1000. The model of estimated mortality associated with salivary cotinine level indicates that the risk for heart disease rises from 1 in 3000 to about 1 in 100 with a gradient of salivary cotinine of 0.1 up to 1 nanogram/milliliter. This risk model successfully predicted the risk observed in the American Cancer Society Cohort Study of passive smoking and lung cancer in non-smokers.

4. Findings

Overall, our control subjects with declarations of low exposures had the lowest cotinine levels. The lowest risk group in this survey, were doctors, nurses and members of a university department of public health who were non-smokers, working in a totally smoke free environment and

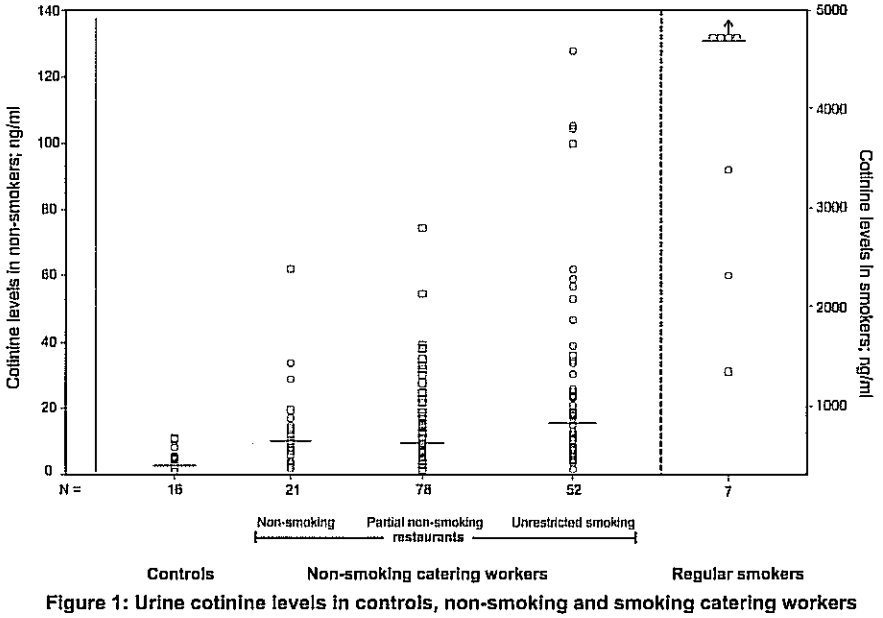


Figure 1: Urine cotinine levels in controls, non-smoking and smoking catering workers

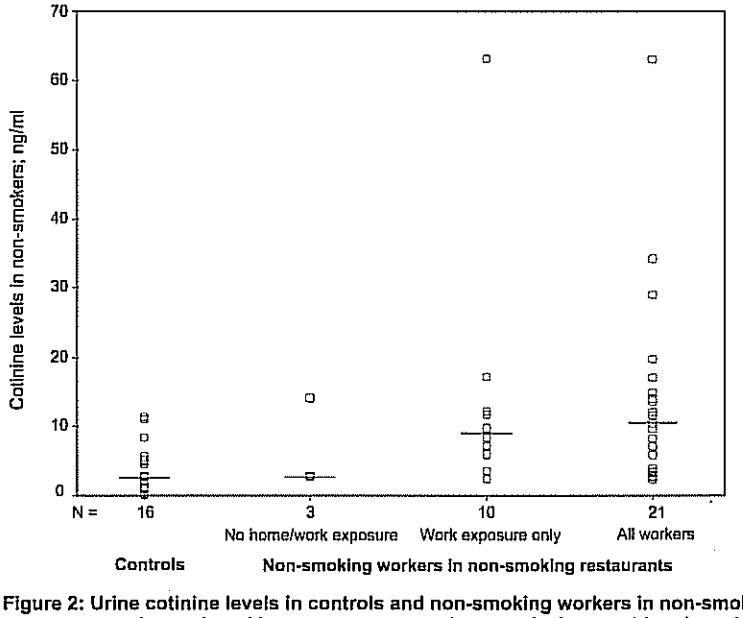


Figure 2: Urine cotinine levels in controls and non-smoking workers in non-smoking restaurants by work and home exposure to other peoples' second-hand smoke

who generally took action to avoid second-hand smoke exposures. At the high end of the non-smoking subjects were waiters and other staff in catering facilities with either partial smoke-free areas or no restrictions on smoking. The small group of regular smokers in the sample showed the expected very high levels of urinary cotinine which were several thousand percent higher than the controls and non-smoking workers (Figure 1).

The data are heterogeneous and show important variations in cotinine levels in catering workers by exposures to tobacco smoke from both customers and other staff, as well as home and leisure exposures. The following brief description is based on the data in the summary Table.

Controls: A total of 16 subjects were tested. Thirteen control subjects with no work or other exposures had a mean of 3.3 (median 2.6, range 0-11.2). In an additional three subjects who declared that they had exposures outside of work the mean cotinine was 67% higher at 5.5 (median 4.5, range 1.1-10.8) (Table 1).

Workers in "non-smoking" restaurants: There was considerable variation in cotinine levels in workers in those restaurants which were designated as "non-smoking" for the purpose of their catering services to the public. Overall, the 3 workers with no exposures *outside of work* who declared that they avoided or did not receive *non-customer exposures at work* had the lowest mean cotinine level at 6.4 (median 2.7, range 2.6-14.0). However a majority of staff (13/21; 62%) were in fact exposed to *non-customer second-hand smoke* because of other staff smoking at break times. Their mean cotinine levels range from 9.9 (median 10.3, range 5.8-13.6) to 14.0 (median 9.0, range 2.2-62.9), that is 50% to 118% higher than workers not exposed to this source and 200% to 324% higher than the lowest risk controls (Figure 2).

Because of exposure to staff smoking at work the cotinine levels in many workers in "non-smoking" restaurants were as high as those in workers in "partial non-smoking" restaurants.

Workers in "partial-non-smoking" restaurants: These findings relate to any worker employed in an organisation which permitted smoking but had various forms of smoke-free areas or seating. Those workers with no exposure *outside of work* and no *non-customer exposure* at work had the lowest cotinine at 6.1 (median 4.2, range 1.5-18.6); a figure which is 85% higher than the value for the lowest risk controls in this study.

Those with any other additional exposures to tobacco smoke had higher mean levels ranging from 7.1 in one subject associated with *home and leisure exposure* only, to 14.3 (median 9.6, range 2-55.3) in those with *other staff and/or break time* exposures, and a mean of 16.6 (median 12.0, range 1-75.4) in 21 workers with both *home/leisure* and *staff/break time* exposures. These mean values are 333% to 403% higher than the control group (Table and Figure 1).

Workers in "unrestricted smoking" restaurants: Overall the mean cotinine levels and the ranges of values in all subgroups of workers in unrestricted smoking establishments were higher than those in workers who had lower declared exposures. In 4 workers with no exposures *outside of work*, and no *non-customer exposures*, the mean was 15.9 (median 16.5, range 7.6-23.1) compared with 28.7 (median 17.3, range 0-129.4) in 34 workers with non-customer workplace exposure. For those with *home/leisure* and/or *non-customer exposures* the mean cotinines ranged from 20.0 to 26.5 (medians 10.4, 30.2, range 0.03-62.3). Overall for this group of 52 workers in unrestricted smoking establishments the mean for those who did not have exposures from other staff was 20.4 (median 18.2, range 7.6-34.6), and 26.6 (median 14.8, range 0-129.4) for those with staff/ break exposures in addition to customer exposures (Table and Figure 1).

Cotinine levels in waiters and non-waiters: When workers were classified into subgroups relating to their job description, no significant differences were found in the mean cotinine values between waiters and workers in other departments in the same establishment.

However some individual waiters had the highest cotinine values observed in the survey. For example the mean cotinine for non-waiters in partial-smoking restaurants was 14.0 (median 12.1, range 1.0-35.0) compared with 14.2 (median 9.4, range 1.4-75.4) for waiters. In the restaurants with unrestricted smoking the mean cotinine for non-waiter staff was 23.0 (median 18.6, range 0.03-57.3) compared with 26.9 (median 14.7, range 0-129.4) for waiters. Lower cotinine values were observed in 3 catering workers who worked in either partial-smoking or unrestricted smoking restaurants. Two of these were non-waiters.

Variations by work exposure and gender: The average restaurant worker, who had second-hand smoke exposures at work only, had a urinary cotinine which was 464% higher than the control subjects. These 104 workers, with work exposure only, had a mean cotinine of 18.6 (median 11.1, range 0-129.4) compared with a slightly lower mean 17.0 (median 10.9, range 0-129.4) in the whole group of 170 workers (Figure 3). There is therefore no evidence that the high cotinine values observed in workers are mainly due to second-hand smoke exposures outside of their work (Figure 3). There was no significant difference in cotinine levels between male and female workers.

Ventilation and cotinine levels: The majority (98/105; 93%) of catering workers who were exposed to tobacco smoke only at work, stated that air conditioning units operated in their workplace. In general cotinine levels in these workers were as high or higher than the levels in workers without air conditioning.

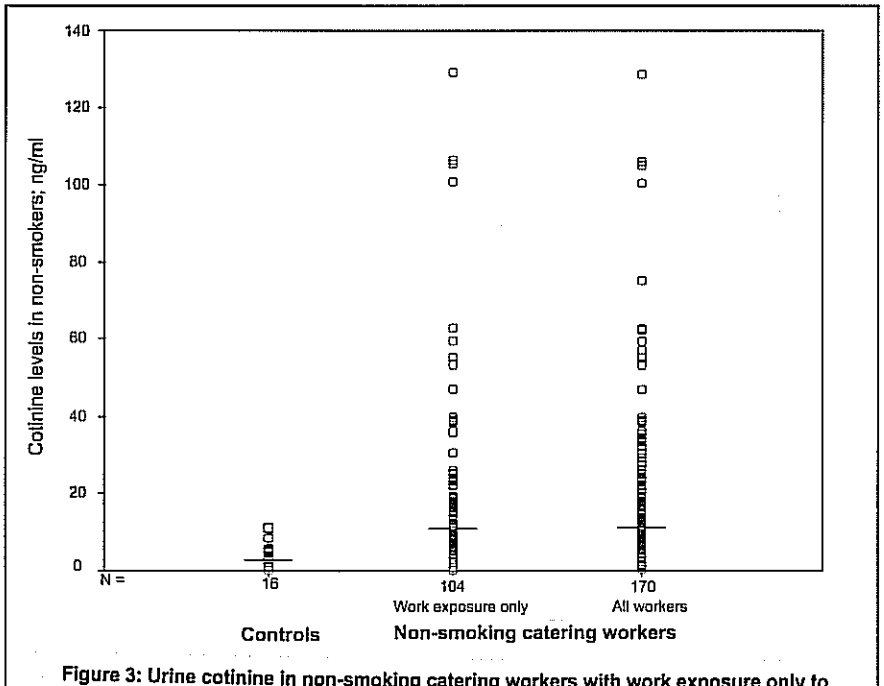


Figure 3: Urine cotinine in non-smoking catering workers with work exposure only to second-hand smoke compared with all workers with (declared) both work and home/leisure

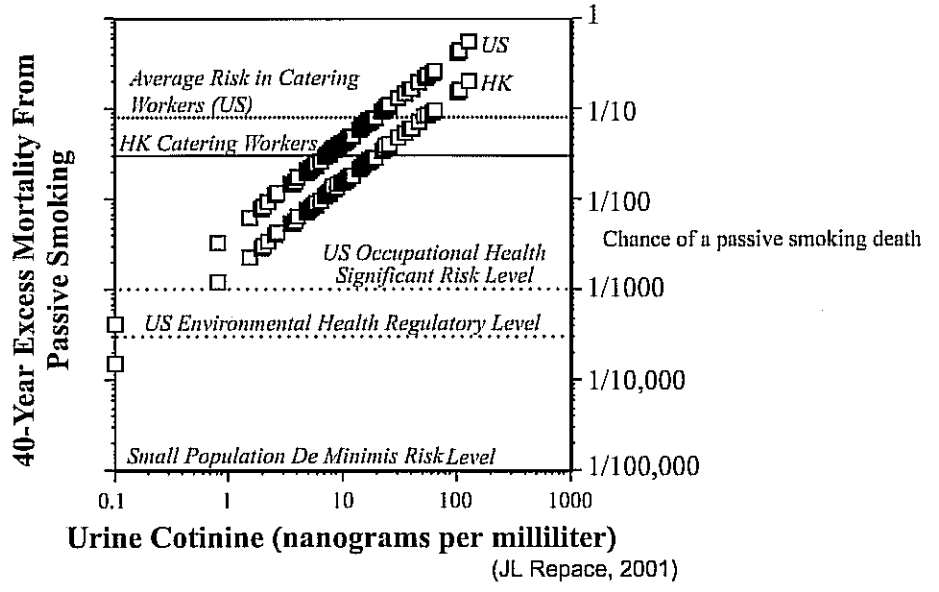


Figure 4: 104 catering workers, exposed to second-hand smoke only at work
Working lifetime combined risk from fatal heart disease & lung cancer based on both US and Hong Kong mortality rates and Hong Kong exposures to passive smoking

The cotinine levels were lower in workers who had their last shift more than 12 hours previously, compared with those who had worked more recently or were at work during the survey (Table 3). This reflects the exposure levels and the biological half-life of cotinine in body fluids.

Table 3: Relationship between shift work and cotinine level

Restaurant type \ Last shift	More than 12 hours ago		Less than 12 hours ago	
	Mean (SD)	N	Mean (SD)	N
Non-smoking	4.9 (1.3)	2	14.8 (14.4)	19
Partial smoking	11.7 (9.2)	26	15.4 (14.1)	52
Unrestricted smoking	21.7 (36.3)	11	26.8 (27.7)	41

Declared smokers: The mean cotinine level for those who were classified as *occasional smokers* was 145.0 (median 167.9, range 2.2-286.8) for those with no exposure *outside of work*. The overall mean for this subgroup of smokers was 250.2 (median 213.9, range 2.2-881.4). The use of tobacco in this group was variable and very low in some subjects. Four out of seven had cotinine >200, the other 3 ranged from 2.2 to 121.8.

For *regular smokers* the mean cotinine was 3589 (median 4671, range 1282-4671) ng/ml. Variations within this group are likely to reflect mainly individual smoking pattern and amount rather than passive smoking exposure.

Combined heart disease and lung cancer risks: The risk calculations based on urinary cotinine levels were carried out on a selected subgroup of 104 non-smoking workers who only had work exposure to second-hand smoke. In this series the mean urinary cotinine concentration is 18.6 ng/ml and the median 11.1 ng/ml. The 90th percentile is 39.1 ng/ml and many workers have cotinine in excess of 40 ng/ml.

The 40 year working lifetime combined excess risk for heart disease and lung cancer is 7.8% (that is 1 in every 13 persons at risk) based on the US population mortality for heart disease and lung cancer (Figure 4). However, in Hong Kong, the present population mortality rates for heart disease are lower than in the US by a factor of about 2.6. The working lifetime excess risk for Hong Kong is 3% (that is 1 in 33 workers at risk) (Figure 4). This means that in the current population of catering workers (about 200,000), we predict 150 deaths per year of exposure from passive smoking, or 6,000 in a working lifetime. Of these 6,000 deaths, 3,840 (64%) will be in workers who have never smoked. Also marked on the graph in Figure 4 is the *de minimis* risk level, which corresponds to an excess lifetime mortality risk of one death in a million persons at risk and is considered acceptable from a regulatory point of view. An estimated risk level as high as 3 in 10000, marked on the graph as the *US Environmental Health Regulatory Level*, would be considered so unsafe that US Federal regulatory agencies almost always act to reduce them.

The aim of interventions and control of second-hand smoke would be to reduce the risk level to zero or at least to the *de minimis* level.

5. Comment

Based on the findings of this sample we can conclude that the majority of catering workers in Hong Kong, both waiters and other staff, have high levels of exposure to second-hand smoke in their workplace with a major risk to their current and future health.

None of the groups of workers examined had mean levels of cotinine as low as that of the control subjects and most were more than double this value. Tobacco smoke from other staff smoking (ie the *non-customer exposures*) within the workplace were apparently important sources of second-hand smoke for all catering workers. This was a major source of tobacco smoke exposure in those workers supposedly working in smoke-free restaurants. The mean levels of those exposed to non-customer smoking were more than twice the levels of those not exposed. Non-customer smoking in all restaurants is clearly a hazard to both workers and patrons, as would be expected from the well established parameters of smoke dispersion in all indoor environments.

Questions will be raised about the validity of the findings in this survey, and particularly about the possibility of misclassification of occasional smokers as non-smokers. Occasional smokers are relatively uncommon and overall

we believe that smokers have been effectively excluded from this sample by the questionnaire and breath carbon monoxide screening. Very high cotinine values (>85) have been found in other surveys, eg in non-smoking bar tenders in Buffalo, New York (Repace 2001). In our survey there were four cotinine values greater than 75 in non-smoking restaurant workers (3 female, one male; 101.1, 105.4, 106.5 and 129.4). All of these subjects worked as waiters in restaurants with unrestricted smoking; all stated that several co-workers smoked near to them and all were at work during the survey and had been at work the previous day. All stated that they had no exposure outside of work; we believe they are passive smokers. Exclusion of these four high values would only reduce the mean cotinine for all restaurant workers with work exposure from 18.6 to 15.0 ng/ml and would not affect the conclusions of the survey.

The data also show the importance of home and leisure exposures to second-hand smoke in non-smokers in Hong Kong. All of the subgroups in this pilot survey showed a marked tendency to have raised cotinine levels if they were exposed to smoke in their leisure venues or at home.

General exposures to second-hand smoke in Hong Kong are clearly widespread as only 2 (13%) out of the 16 "low-risk" control subjects had zero cotinine levels. This contrasts with a recent population survey by the US Center for Disease Control which showed that, as a result of countrywide smoking bans in public and indoor places in the United States, 50% of the sample had *undetectable* levels of cotinine.

The mean urinary cotinine in our lowest risk group in Hong Kong (those without any known home or leisure exposure) was 3.3 ng/ml, a finding which is totally unacceptable given that in the US it indicates a lifetime excess risk for coronary heart disease mortality of greater than 1 in a 100 compared with the normative *de minimis* standard of acceptable risk of 1 in 1,000,000.

The Government should increase the resources available to inform the public of the serious health hazards of second-hand smoke, including those associated with smoking in the home.

A recent study in New Zealand showed that the exposure of bar and restaurant staff to tobacco smoke can be as high as the exposure of active smokers. The hair nicotine levels of non-smoking workers in workplaces with no restrictions on smoking were as high as those in smokers.

Previous studies of non-smoking workers exposed to second-hand smoke in Hong Kong have demonstrated an increased frequency of chronic respiratory complaints (cough, phlegm and wheeze), increased health care utilization and costs and sickness absence from work.

Passive smoking is increasingly recognized as an occupational health risk world-wide. For example:

- In October 1997, 60,000 US flight attendants won a major settlement in a class action against transnational tobacco companies. The action was initiated by a non-smoking flight attendant who contracted lung cancer. The tobacco industry did not admit liability.

- In the Netherlands a court ruled in May 2000 that employers must guarantee that non-smoking staff have a working environment completely free of tobacco smoke. It upheld a postal worker's complaint that her exposure to tobacco smoke at work *infringed her right to work in a smoke-free environment.* The court ruled that her employers were bound by the constitutional rights of citizens, to protection of "physical integrity and "health", to provide such conditions. The employers failed to satisfy this right under employment law.

- In May 2001 an Australian barmaid, a non-smoker, was awarded US$235,000 for cancer caused by working for 11 years in a smoky bar. Most Australian states have already banned smoking in pubs, clubs and restaurants and a similar ban will come into force in New South Wales in September 2001.

Cotinine levels in this survey are consistently higher in establishments with partial or unrestricted smoking. Increasing smoker density in designated smoking areas increases the hazard to workers who have to service these areas. In separately ventilated smoking lounges and cigar divans the concentrations of second-hand smoke particulates and gases, including cardiovascular toxins and cancer causing substances, will predictably be very high. The contamination persists after smoking ceases and part of this comes from off-gasing from deposits on furniture and fittings. The risks to both patrons and staff are currently being ignored.

It is clear that ventilation technology cannot control and reduce the risk from second-hand smoke to minimal safety standards (1 in a million) *without massively impractical increases in ventilation and intolerable levels of air changes of "typhoon strength"* (JL Repace: Repace@erols.com).

However damage to the health of catering workers from passive smoking is wholly preventable. The establishment of smoke-free bars and taverns in California was followed by a rapid improvement in the respiratory health of the workers. The present survey confirms that workers in Hong Kong who are forced to breathe second-hand tobacco smoke in their workplace have markedly raised levels of nicotine metabolites in their circulation. We know that this is also an indicator of toxic exposures to substances which cause *heart disease* and *cancer* in addition to chronic *respiratory health problems.*

On the other hand the tobacco industry and many sectors of the hospitality industry continue to (i) deny that second-hand smoke is a poison, (ii) deny that both workers and customers are injured by breathing second-hand smoke, (iii) oppose the introduction of environmental and public health measures to prevent passive smoking in the workplace and public places. This is in spite of the fact that no *bone fide* economic analyses have shown any adverse impact on catering business or tourism. Tobacco industry propaganda has generated unjustified concern about loss of business and jobs. There is no reason why Hong Kong workers should not now be protected against the risks of passive smoking.

Legislation to provide and ensure totally smoke-free indoor workplaces is the only satisfactory solution to this widespread problem and it is urgently needed as a public health and occupational health measure in Hong Kong.

Voluntary agreements and codes of practice will not work and create many problems of monitoring and enforcement. Legislation on smoking bans in all public places is the only cost-effective and reliable means of protecting non-smokers. No workers, whether smokers or non-smokers should be obliged to work in a smoke contaminated workplace. *The principle on which Hong Kong's future workplace smoking controls must be based is that no worker should be required to work in an environment where tobacco products are burning.*

Adherence to this principle will ***not*** *permit smoking in outdoor catering facilities.* Partial smoking restrictions of all kinds leave non-smokers exposed to the risk of passive smoking.

Summary conclusions and recommendations

1 The world's best scientific literature on health risks from passive smoking clearly demonstrates that second-hand smoke is extremely poisonous and the cause of many health problems including chronic respiratory disease, coronary heart disease and cancers.

2 *The majority of catering workers in Hong Kong are exposed to second-hand smoke in their workplace and most of them have markedly raised urinary cotinine concentrations which indicate markedly raised health risks for chest and heart disease and cancer in addition to many other health problems caused by passive smoking.*

3 Most of the non-smoking subjects in this new survey have raised working lifetime excess risks for heart disease and lung cancer as a result of passive smoking. In catering workers the *average* excess risk was 3% or about 1 in 33. We estimate that among 200,000 catering workers, 6,000 will die from passive smoking due to heart disease and lung cancer; 3,800 (64%) of these deaths will be in never smokers.

4 *In a group of "low risk" control subjects from smoke-free workplaces, many had detectable cotinine levels indicating that for many of them the airspaces of their home, leisure activities or other worksites visited by them are contaminated by tobacco smoke. All non-smokers in Hong Kong should have no detectable cotinine in body fluids.*

5 There is no practical solution from ventilation engineering to the problem of second-hand smoke exposures; the only safe and most cost-effective strategy is to introduce smoke-free regulations in all catering facilities and other workplaces. **The principle must be that no worker should have to work in air contaminated with tobacco smoke in order to hold a job.**

6 *There is an urgent need for effective and enforceable legislation which will ensure that all workers in all workplaces in Hong Kong do not have to breathe second-hand smoke.*

7 There should be no exceptions to, or trade-offs in, smoke-free regulations which will lead to the health of workers being placed at risk.

8 *There should be an urgent review by Government of designated smoking areas including smoking lounges which are separately ventilated, and particularly those which are continuously staffed such as cigar divans. The health implications for all workers who service any type of smoking lounges or other designated areas should be examined and re-assessed.*

9 The catering and hospitality industry should take the lead now in implementing comprehensive smoke-free policies in all facilities to protect both staff and customers.

10 *The public, the media, legislators and particularly the catering industry should be aware that the tobacco industry has for many years consistently denied and obfuscated the findings of research into second-hand smoke and passive smoking.*

11 We fully expect that the tobacco industry will also attempt to discredit the findings of this latest investigation in Hong Kong, but there are incontrovertible reasons why Government policy to eradicate passive smoking should be fully implemented without further delay.

References

Repace Associates Inc. http://www.repace.com/.

Repace JL and Lowrey AH. An enforceable indoor air quality standard for environmental tobacco smoke in the workplace. Risk Analysis 1993; 13:463-475.

Lam TH, Ho LM, Hedley AJ, Adab P, Fielding R, McGhee SM, Aharonson-Daniel L. Environmental tobacco smoke exposure among police officers in Hong Kong. *JAMA* 2000; 284: 756-763.

McGhee SM, Adab P, Hedley AJ, Lam TH, Ho LM, Fielding R, Wong CM. Passive smoking at work: the short-term cost. *Journal of Epidemiology and Community Health* 2000; 54: 673-676.

Al-Delaimy W, Fraser T, Woodward A. Nicotine in hair of bar and restaurant workers. *N Z Med J* 2001;114:80-3.

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

BBC News Asia Pacific. Barmaids passive smoking payout. http://news.bbc.co.uk.

Dr N Haley. MetLife Laboratory, 4 West Chester Plaza, Elmsford, NY 10523, USA. Email: njhaley@MetLife.com.

Sheldon T. Dutch workers entitled to smoke-free conditions, court rules. bma news online. *BMJ* 2000; 320:1227 (6 May).

National Institutes of Health, National Cancer Institute. Smoking and Tobacco Control. Monograph 9. Cigars: Health Effects and Trends, 1999.

Glantz S. Smoke-free restaurant ordinances do not affect restaurant business. Period. *Journal of Public Health Management and Practice* 1999; 5:vi-x.

Glantz SA, Charlesworth A. Tourism and hotel revenues before and after passage of smoke-free ordinances. *JAMA* 1999; 281:1911-1918.

Acknowledgements

This project was prompted by the Research Committee of COSH (Chair: Professor Edith Lau). We thank the management of all of the catering establishments and the catering workers and control subjects who completed the interview schedule and donated a urine sample. We are very grateful to Dr Sidney Tam, Consultant and Head of the Division of Clinical Biochemistry Unit, Queen Mary Hospital for his advice and help with the management of the urine samples. We thank Ms Marie Chi for clerical assistance and the typing of this report.

Controlling Tobacco Smoke Pollution

By James Repace, Associate Member ASHRAE

Secondhand smoke (SHS) is a dangerous, often unregulated, environmental pollutant that causes cancer and heart disease in adults and respiratory disease in children. Smoking bans eliminate these risks. However, some groups insist that ventilation, which inevitably leaves residual smoke in the air, can provide acceptable indoor air quality.

How does ventilation compare to smoking bans in controlling SHS in hospitality venues? On Nov. 27, 2002, Delaware banned smoking in all restaurants, bars and casinos, with the intent of giving hospitality workers the same occupational health protection that other workers had enjoyed since 1994.

This afforded an opportunity to investigate contemporary levels of SHS in the hospitality industry. I conducted an indoor/outdoor air quality study in the Wilmington, Del., metropolitan area before and after the enactment of Delaware's clean indoor air law.[1] *Table 1* describes the venues investigated, including a casino, six bars, and a pool hall.

The pollutants measured were respirable particulate matter (RSP) and particulate polycyclic aromatic hydrocarbons (PPAH), which are emitted by cigarettes, pipes, and cigars. These pollutants are also known to be involved in the induction of cancer, respiratory disease, heart disease, and stroke. RSP is also a regulated outdoor air pollutant, while PPAH contains 10 known carcinogens, and causes arterial wall damage.[1,13,14]

Equipment & Methods

I deployed concealed continuous real-time monitors for RSP, i.e., airborne particulate matter in the combustion range below 3.5 microns in diameter ($PM_{3.5}$), and PPAH, as well as carbon dioxide, carbon monoxide, temperature, and relative humidity. All indoor venues visited were selected by personnel of the American Lung Association of Delaware to represent a cross-section of the spectrum of area hospitality venues.

Visits averaged ~30 minutes. For comparison, I sampled outdoor pollutants on city streets, on Interstate 95 in heavy traffic during rush hour, and in a nonsmoking hotel room.

Monitoring was conducted on Nov. 15, 2002, prior to the smoking ban, and again on Jan. 24, 2003, two months after the ban. All monitoring equipment was synchronized to an atomic clock signal via computer; venue visiting times were recorded in a diary. The area, volume, number of persons, and average number of active smokers were recorded for each venue to generalize the results.

Predicting SHS Concentrations

Respirable particulate air pollution concentrations from SHS (SHS-RSP) are directly proportional to the smoker density and inversely proportional to the air-exchange rate, and can be quantified using the time-averaged mass-balance model, or Habitual Smoker Model (HSM).[2–5] A habitual smoker is defined as smoking two cigarettes per hour at 10 minutes per cigarette.[3,4] Thus, for every three habitual smokers, one cigarette burns constantly on average.

Equation 1 gives SHS-RSP in units of micrograms of RSP per cubic meter of air ($\mu g/m^3$), from the ratio of the active smoker density D_s, in units of average number of burning cigarettes per hundred cubic meters ($BC/100\ m^3$) in the space, to the air exchange rate Q_v, in air changes per hour (h^{-1}), where the constant 650 incorporates a 30% default RSP surface deposition term, and assumes 14 mg SHS-RSP per cigarette.[2,12]

$$SHSRSP = 650\ \frac{D_s}{Q_v} \quad (\mu g/m^3) \qquad (1)$$

Since Equation 1 predicts the time-averaged value of the SHS concentration, it does not require that the concentration be constant during the observation period for accurate predictions but assumes that the initial and final conditions are the same.

When used to analyze actual measured data, a "trend correction term" $\Delta X/Q_v T$ may be required if this quantity is significant compared to the time-averaged value of SHS-RSP, where ΔX is the difference between the initial and final SHS concentrations, and T is the observation time.[5] However, the trend correction term disappears when ΔX is zero, or can be

Venue	Description
A. Casino	Large volume slot machine-only casino with restaurant/bar areas, all smoking; one relatively small nonsmoking area prior to the ban. Monitors circulated around periphery of central salon during smoking tour; during nonsmoking tour, monitors located in outer portion of coat-check room open to surrounding air through large window.
B. Bar/Restaurant	Stand-up/sit-down smoking bar area with adjacent dining table area; located in a midsize shopping mall with an outdoor entrance. Monitors on both smoking and nonsmoking tours located in same location at end of bar area.
C. Bar/Restaurant	Large volume nonsmoking restaurant with entertainment section; caters to families, but with a fenced-off bar area (the only smoking area prior to the ban). Monitors located inside bar area at periphery at same location on both visits.
D. Bar/Restaurant	Sit-down smoking bar; open passage to dining area; genteel sports-bar-like atmosphere. Monitors located at same spot ~6 ft from vestibule at one end of bar area on both visits.
E. Bar/Restaurant	Large sit-down upscale smoking bar surrounded by smoking dining tables with adjacent dance floor; no cover charge; serves singles, couples, and parties. Monitors located between bar stools in proximate locations on each visit.
F. Bar/Restaurant	Sit-down smoking bar with large adjacent nonsmoking restaurant area for dining. Monitors located on opposite sides of one end of bar area on each visit.
G. Stand-up Bar	Stand-up smoking bar with adjacent dance floor primarily catering to college or college-age singles; very crowded. Cover charge was requested of all patrons. Monitors located ~6 ft from front door and on opposite sides for each visit. Door was frequently opened as persons entered or left premises. Several patrons smoked outside the door during the nonsmoking tour.
H. Pool Hall	Stand-up/sit-down smoking bar contiguous to adjacent smoking pool hall; mostly working class adult patrons. Monitors located on periphery of pool table area during smoking tour; at a nearby pool table during the nonsmoking tour.

Table 1: Eight Wilmington, Del., hospitality venues in which air quality measurements were made; areas described as "smoking" were smoking on Nov. 15, 2002, and nonsmoking on Jan. 24, 2003, after the ban. These venues were chosen from across the spectrum of available hospitality types.

neglected when T is very large compared to $\tau = 1/Q_v$, where τ is the residence time for smoke in the air.[5]

In many practical cases, SHS-RSP over the observation time is approximately constant in a space with many smokers, and the trend correction term can be neglected.

The HSM is used to predict SHS-RSP for a bar as follows: the Delaware smoking prevalence is 23%. For a bar with a default occupancy of 100 persons per 1,000 ft^2* and a 10 ft* ceiling, the metric volume is 283 m^3, and the habitual smoker density D_{hs} = (0.23 smokers/person)(100 persons)/283 m^3 = 8 habitual smokers per 100 m^3,* of whom an average of one-third are assumed to be actively smoking during any 10 minute period. Thus, the density of active smokers expected to be observed in a Delaware field survey is given by $D_s = D_{hs}/3$ = 2.7 burning cigarettes per 100 m^3.

The default air-exchange rate is estimated from ANSI/ASHRAE Standard 62-1989, *Ventilation for Acceptable Indoor Air Quality*, which prescribed 30 ft^3 of outdoor air per minute per occupant (ft^3/min-occ) for smoking bars. Thus Q_v = (30 ft^3/min-occ)(100 occ/10,000 ft^3)(60 min/hr) = 18 air changes per hour.

Using Equation 1, the estimated respirable smoke particulate (RSP) concentration ($PM_{3.5}$) for a Delaware bar under the ASHRAE default assumptions for smoking occupancy and ventilation, is: $SHS\text{-}RSP_{pub} = 650\ D_s/Q_v = 650(2.7)/(18) = 98\ \mu g/m^3$.

If we add the expected outdoor background RSP level of 16.6 µg/m^3 to this value (the 2003 annual average from the New Castle County, Del., outdoor air quality monitoring network), since outdoor RSP easily penetrates indoors, we would expect to find a typical total RSP level of (98 µg/m^3 + 17 µg/m^3) = 115 µg/m^3 in a Delaware bar ventilated according to Standard 62-1989.

Applied to the analysis of a specific bar whose indoor/outdoor RSP concentrations and smoker density have been measured, the HSM can be used to estimate the air-exchange rate.

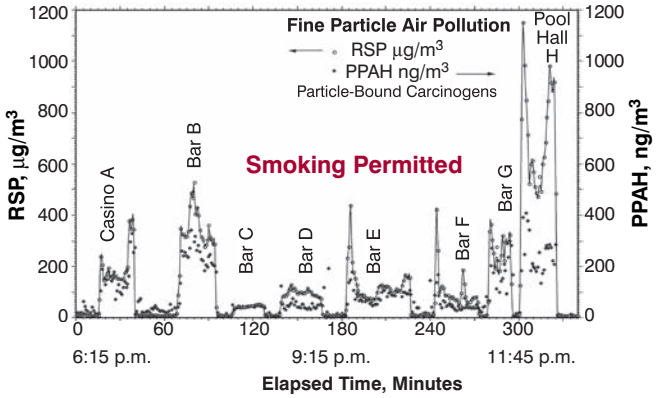
Field Measurements and Results

Figure 1 shows the real-time measurements performed on Nov. 15, 2002, before the smoking ban. The large peaks from the indoor smoke-filled venues loom far above the much cleaner outdoor air. Measurements of total RSP, averaged over the six bars in *Figure 1*, yield a mean of 160 µg/m^3 (standard deviation [SD] = 111 µg/m^3), with a median value of 115 µg/m^3. Thus, the median default prediction (above) and the global median value are in good agreement because both the measured six-bar median smoker density and estimated air exchange rate were 10% of expected: The expected D_s was 2.7, the actual values ranged from 0.02 to 1.4, and averaged 0.47 (SD = 0.56), and the median was 0.24. The expected Q_v was 18 h^{-1}, the estimated values for the six bars using the HSM ranged from 0.3 to 3 h^{-1}, with a mean of 1.5 h^{-1} (SD = 1), and a median of 1.5 h^{-1}.

Figure 2 shows the corresponding measurements in the same venues performed on Jan. 24, 2003, after the smoking ban, with dramatically lower pollution levels. Post-ban, it is nearly impossible to distinguish between indoors and outdoors except for the pool hall, which has another indoor source, possibly chalk dust from the pool cues.

Figure 3 shows that both RSP and PPAH increase markedly with smoker density, as the model predicts. PPAH does not show as strong a variation with air exchange rates as RSP, because controlled experiments show that due to enhanced

* 100 persons/100 m^2; 10 ft = 3.05 m; 30 cfm/occ = 15 L/s.



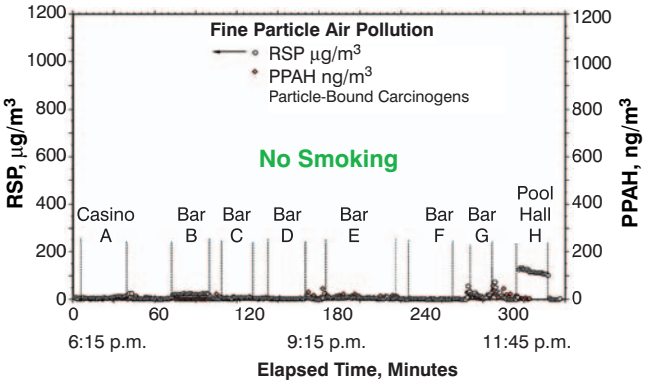


Figure 1 (left): Real-time RSP air pollution and PPAH outdoors and in a casino, six bars and a pool hall before a smoking ban.[1] For comparison, the NAAQS for fine-particle air pollution ($PM_{2.5}$) is 15 $\mu g/m^3$, the annual average level defining clean air. Figure 2 (right): RSP air pollution and PPAH in the same venues after the smoking ban.[1]

surface deposition, SHS-PPAH decays twice as fast as SHS-RSP.[1] The ratio of RSP/PPAH was found in controlled experiments to be 2,000:1.

Prior to the smoking ban, all venues were heavily polluted, with indoor RSP levels averaging 20 times outdoor background. For workers, these levels violated the annual National Ambient Air Quality Standard (NAAQS) for fine particles ($PM_{2.5}$) by a factor of 4.6. Wilmington hospitality workers were exposed to RSP levels 2.6 times higher than on Boston city streets heavily polluted by truck and bus traffic.

Wilmington pre-ban indoor carcinogenic PPAH averaged five times higher than outdoor background levels, tripling workers' daily exposure, and exceeding PPAH measured at an I-95 tollbooth at the Baltimore Harbor Tunnel.

Comparing the indoor and outdoor data in *Figure 1*, and the data in *Figure 1* to *Figure 2*, SHS contributed 90% to 95% of the RSP air pollution during smoking, and 85% to 95% of the carcinogenic PPAH. This occurred despite a smoking prevalence 35% lower than the statewide average.

This air quality survey has demonstrated conclusively that the health of hospitality workers and patrons was endangered by SHS pollution. The Delaware Clean Indoor Air Act's ban on smoking in hospitality workplaces eliminated that hazard. As *Figure 3* shows, and CO_2 measurements support, there is substantial under-ventilation of all venues. For the four bar venues (B–E) for which reliable pre- and post-ban air exchange rate comparisons could be made (pre-ban calculated from the data by the model and post-ban from CO_2), the median pre-ban rate was 1.85 ACH vs. a post-ban median of 1.34 ACH, which was far below the 18 ACH expected. While the smoker densities are lower than expected, so are the air-exchange rates, and the model applied to the data allows us to understand why the concentrations are what they are, and, therefore, generalizes the results.

This raises two important questions: if these venues had actually been ventilated according to Standard 62-1989, would it have been enough to provide acceptable indoor air quality? And since no cognizant authority has actually defined an acceptable level for SHS, can we estimate what level of SHS might be acceptable?

Guidance on these questions can be derived from American and Australian ventilation standards, and from the air quality standards, practices, and proposed rules of U.S. regulatory agencies.

Minimum Ventilation Rates for SHS Control

After 30 years of recommending ventilation rates for the control of tobacco smoke odor, Standard 62.1-2004 revised the Minimum Ventilation Rate Table to apply only to no-smoking spaces, recognizing the mortal hazard of SHS as defined by cognizant authorities.[16] However, Standard 62.1-2004 requires additional (but unspecified) ventilation in excess of the table rates for engineers designing for smoking venues.

For a given level of smoking, is it possible to estimate how much additional dilution ventilation might be required to attain acceptable indoor air quality? This can be approached in two ways, both of which use the indoor air quality procedure of Standard 62.

Particulate Phase Control

First, consider SHS as just simple particulate pollution. One guideline recommended by Standard 62.1-2004[16] for assessing indoor air quality is the U.S. NAAQS. The NAAQS for $PM_{2.5}$ is designed to protect against respirable particle health effects such as premature death, increased hospital admissions, and emergency room visits, primarily among the elderly and individuals with cardiopulmonary disease; increased respiratory symptoms and disease in children and individuals with cardiopulmonary disease; decreased lung function particularly in children and individuals with asthma; and against alterations in lung tissue and structure and in respiratory tract defense mechanisms in all persons.

How much ventilation would it take to satisfy NAAQS? To satisfy NAAQS *de facto*, a worker's weighted annual average exposure needs to be ≤ 15 $\mu g/m^3$. Suppose the outdoor annual average RSP level were 10 $\mu g/m^3$, at the low end for all U.S. counties. The modeled SHS-RSP concentration for a bar is 98 $\mu g/m^3$. Then, a calculation of the time-weighted annual average exposure for bar staff, assuming an eight-hour workday and

a 250 day work-year, yields a maximum permissible indoor SHS-RSP concentration of 22 μg/m^3.

Using the HSM, it is easily calculated[1] that the minimum necessary air exchange rate would have to be ≥ 80 air changes per hour (ACH), equivalent to 133 cfm/occ (a ~15-fold increase over the 9 cfm/occ recommendation for bars from Standard 62-2004).

Suppose the outdoor air level were to average 14 μg/m^3. In that case, the required bar air-exchange rate Q_v increases to 400 ACH or 665 cfm per occupant (occ). At the actual 16.6 μg/m^3 outdoor air average, NAAQS can never be attained unless the outdoor air supply is cleaned with a fine particle filter.

However, even if NAAQS could be met, how could the practitioner be assured that the residual SHS concentration was safe for occupants to breathe from a carcinogenic and toxic standpoint? This leads us to the Australian approach.

SHS Carcinogen and Toxin Control

Australian ventilation engineers developed informative guidance called the Environmental Tobacco Smoke Harm Index (ETSHI) (AS 1668.2 Supplement 1—2002),[6] based on a scientific report of the Australian National Health and Medical Research Council.[7] The Australian methodology is equivalent to applying the Indoor Air Quality Procedure of Standard 62-2004.

The ETSHI is used to estimate the mortality risk associated with a specified exposure to SHS in an environment that is ventilated and that may be fitted with an air cleaner. Appendix A of the ETSHI guidance estimates the combined lung cancer and heart disease mortality risk for office workers in a typical smoking-permitted office as: ETSHI = 225 deaths per million exposed Australian office workers per year. This is similar to an estimate for U.S. office workers of 244 deaths per million per year, made using the defaults of the old Standard 62-1989, which recommended ventilation rates for smoking venues.[8] The default assumptions for both Australian and U.S. office workers are the same: 10 persons per 100 m^2 of occupiable space and a ventilation rate of 10 L/s-occ. The smoking prevalence for the Australian case was 33%, and in the U.S. case was 29%. Normalized for smoking prevalence, these risk estimates[6,8] differ by less than 15%, and are likely due to the use of particulate air filtration in the ETSHI calculation.

The ETSHI for office workers is readily scaled to bar workers. As the calculation under Equation 1 showed for the default bar, a concentration of 98 μg/m^3 resulted for a smoking prevalence of 23%. Scaling that to the 33% of the Australian office assumption, that increases to (0.33/0.23)(98) = 141 μg/m^3. For the default Australian office, the smoker density is $D_s = D_{hs}/3 = 0.39$. The default air exchange rate is 1.2 ACH, neglecting any additional air cleaning as the tobacco aerosol is submicron in size.

Using Equation 1, the predicted respirable smoke particulate (RSP) concentration ($PM_{3.5}$) for an Australian office is calculated as: $SHS-RSP = 650(0.39)/(1.2) = 211$ μg/m^3.

Thus, assuming a 33% smoking prevalence, the ETSHI for the default U.S. bar is scaled as (141/211)(225) = 150 deaths per million per year, or in a 45-year working lifetime, an estimated 6,750 deaths per million persons at risk, or a working lifetime mortality rate of 7 per 1,000.

By comparison, U.S. Occupational Safety and Health Administration (OSHA) estimated in 1994 a working lifetime risk to U.S. workers from SHS ranging from 7.4 per 1,000 to 17 per 1,000.[8,10]

How big are these risks? OSHA defines a risk of 1 per 1,000 as a "significant risk of material impairment of health." OSHA, a cognizant authority,[15] stated that, for mortality rates of this magnitude, "the significance of risk is very great."[10] Risks in excess of 3 per 10,000 are invariably regulated.[9]

Although no cognizant authority has set an acceptable level for SHS *per se*, we can ask if there is some level of mortality *risk* that federal regulatory agencies have viewed as acceptable? For guidance on this issue,[8] we turn to a Harvard University review of 133 U.S. regulatory decisions. The risk management decision rule employed by federal regulatory agencies such as OSHA, Environmental Protection Agency (EPA), and Food and Drug Administration for carcinogens and toxins in air, water, or food is called *de minimis risk*, i.e., a lifetime risk "beneath regulatory concern."[9] This level is typically one death per million persons per lifetime.[8,9]

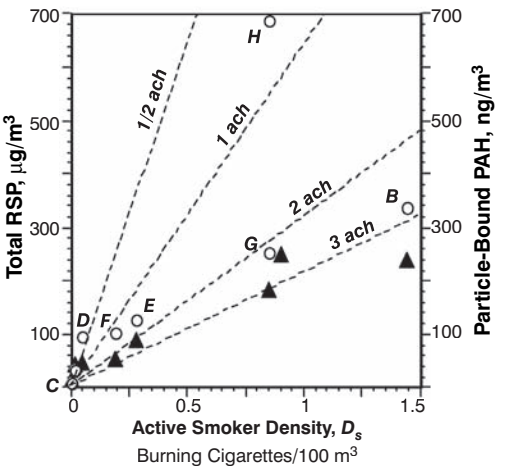


Figure 3: Air pollution in seven of eight hospitality venues where smoking occurred (smoker density not recorded for Casino A), and a Delaware nonsmoking hotel room on Nov. 15, 2002.[1] Both RSP and PPAH increase with increasing D_s. Data points B-G are the six bar venues. Circles represent RSP. Triangles represent PPAH.

OSHA failed to promulgate a rule governing the private sector, eventually withdrawing its proposed rule in 2001 (66 FR 64946) due to heavy Congressional pressure to leave SHS regulation up to the states.[1] Nevertheless, all federal workplaces have become smoke-free, and Congress itself legislated smoking out of airlines, which were not under OSHA's jurisdiction.[17]

However, states have been slow to act. To date, only nine—California, Connecticut, Delaware, Maine, Massachusetts, Montana (delayed until 2009) New York, Rhode Island, and Vermont (effective in Fall 2005)—have adopted smoke-free workplace laws that protect all workers. In 1997, the California EPA estimated total U.S. mortality from SHS at 38,000 to 65,000 per year.[11] By comparison, drunk driving-related deaths in 1997 totaled 16,000.

How much additional ventilation would be required to attain *de minimis risk* from SHS in the default U.S. bar described previously? If this risk reduction is to be achieved by ventilation alone, since risk is inversely proportional to ventilation rate, to reduce the risk to acceptable levels for bar workers, the ventilation rate would have to be increased by the ratio of the number of estimated deaths to the *de minimis risk*: a factor of 6,750:1, or to 6,750 × 30 cfm/occ = 202,500 cfm/occ, based on Standard 62-1989.[15] However, the default ventilation rate for a smoke-free bar under Standard 62.1-2004 is 9 cfm/occ (equivalent to 5.4 ACH). Thus, the amount that the ventilation rate would have to be increased over the smoke-free case is (202,500/9) = 22,500 times, and the corresponding estimated air-change rate required for acceptable indoor air quality would be 22,500 × 5.4 = 121,500 ACH, which would require a veritable indoor tornado. Even greater airflow rates would apply for air cleaning, which inefficiently removes SHS gases.

The conclusion is that ventilation technology cannot possibly achieve acceptable indoor air quality in the presence of smoking, leaving smoking bans as the only alternative.

Acknowledgment

This work was supported by the Flight Attendant Medical Research Institute (FAMRI).

References

1. Repace, J. 2004. "Respirable particles and carcinogens in the air of delaware hospitality venues before and after a smoking ban." *Journal of Occupational and Environmental Medicine* 46:887–905.

2. Repace, J. Human Exposure to Secondhand Smoke. In: Human Exposure Analysis, Part II. Inhalation – Occurrence, Sources, and Quantitative Levels of Exposure, W. Ott, L. Wallace and A. Steinemann (Eds.), (in press).

3. Repace, J. and A. Lowrey. 1980. "Indoor air pollution, tobacco smoke, and public health." *Science* 208:464–474.

4. Repace, J. and A. Lowrey. 1982. "Tobacco smoke, ventilation, and indoor air quality." *ASHRAE Transactions* 88(1):895–914.

5. Ott, W. 1999. "Mathematical models for predicting indoor air quality from smoking activity." *Environmental Health Perspectives* 107(2):375–381.

6. Standards Australia International. AS 1668.2 Supplement 1—2002, The use of ventilation and air conditioning in buildings— Ventilation design for indoor air contaminant control (Supplement 1 to AS 1668.2—2002) Standards Australia International Ltd, GPO Box 5420, Sydney, NSW 2001, Australia.

7. Australian National Health and Medical Research Council. The Health Effects of Passive Smoking—A Scientific Information Paper. www.nhmrc.gov.au/advice/nhmrc/forward.htm.

8. Repace, J., et al. 1998. "Air nicotine and saliva cotinine as indicators of passive smoking exposure and risk." *Risk Analysis* 18: 71–83.

9. Travis, C.C., et al. "Cancer risk management." *Environmental Science and Technology* 21:415–420.

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

11. California Environmental Protection Agency. 1997. "Health Effects of Exposure to Environmental Tobacco Smoke, Final Report." Office of Environmental Health Hazard Assessment. 1999. In: Smoking and Tobacco Control Monograph 10: Health Effects of Exposure to Environmental Tobacco Smoke, National Cancer Institute, NIH Publication 99-4645, August.

12. Klepeis N.E., W.R. Ott and P. Switzer. 1996. "A multiple-smoker model for predicting indoor air quality in public lounges." *Environ. Sci. Technol.* 30(9):2813–2820.

13. Glantz S. and W. Parmley. 1991. "Passive smoking and heart disease—epidemiology, physiology, and biochemistry." *Circulation* 83:1–12.

14. Barnoya, J. and S. Glantz. 2005. "Cardiovascular effects of secondhand smoke nearly as large as smoking." *Circulation* 111:2684–2698.

15. ANSI/ASHRAE Standard 62-1989, *Ventilation for Acceptable Indoor Air Quality*.

16. ANSI/ASHRAE Standard 62.1-2004, *Ventilation for Acceptable Indoor Air Quality*.

17. Repace, J. 2004. "Flying the smoky skies: Secondhand smoke exposure of flight attendants." *Tobacco Control* 13(Suppl 1):i8-i19.

James Repace is a scientific consultant with Repace Associates in Bowie, Md.