ESTIMATED MORTALITY FROM SECONDHAND SMOKE AMONG CLUB, PUB, TAVERN, AND BAR WORKERS IN NEW SOUTH WALES, AUSTRALIA

James L. Repace, MSc.
Repace Associates, Inc.
Secondhand Smoke Consultants
April 7, 2004

A report commissioned by
The Cancer Council New South Wales
2004

The Cancer Council New South Wales

TABLE OF CONTENTS

BACKGROUND

The Cancer Council of New South Wales (NSW), Australia, has commissioned Repace Associates, Inc. to conduct a scientific assessment of the risks from exposure to secondhand smoke (SHS) for workers in NSW hotels, clubs and other licensed venues such as taverns, and casinos, relative to the absence of workplace exposure.

By way of background, the Government of the Australian Capital Territory (ACT) announced recently that smoking would be banned (with no exemptions) in every ACT club, pub and licensed venue by December 2006. This represents the first time an Australian jurisdiction has committed to total smoking bans in the whole of the hospitality industry.

In NSW, current legislation (the Smoke-free Environment Act NSW 2000) prohibits smoking in a wide range of enclosed public places, but provides exemptions for most areas of hotels, registered clubs and nightclubs.

AUTHOR

James Repace, MSc., is a health physicist, and president of the international secondhand smoke consulting firm, Repace Associates, Inc.. He has published 60 scientific papers on the hazard, exposure, dose, risk, and control of secondhand smoke. He holds an appointment as Visiting Assistant Clinical Professor at the Tufts University School of Medicine, Boston, Massachusetts. He has earned several national honors, including The Surgeon General's Medallion, the Flight Attendant Medical Research Institute Distinguished Professor Award, the Robert Wood Johnson Foundation Innovator Award, and a Lifetime Achievement Award from the American Public Health Association. He was a senior policy analyst and scientist with the U.S. Environmental Protection Agency, Office of Air and Radiation and 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. For many years he was a research physicist at the Naval Research Laboratory in the Ocean Sciences and Electronics Divisions. His curriculum vitae may be viewed at www.repace.com.

EXECUTIVE SUMMARY

Secondhand Smoke (SHS) contains numerous carcinogens and toxins regulated in the outdoors or the industrial workplace. National and international occupational health, environmental health, and public health authorities have widely condemned SHS exposure.

A new study of SHS air pollution in 17 licensed gaming clubs with smoking and nonsmoking areas in New South Wales (NSW) by Cains et al. (2004) showed that smoking caused 86% of inhalable particle air pollution (PM_{10}) in the smoking sections and caused 71% of the pollution in the nonsmoking sections.

An analysis of this data shows that SHS irritation thresholds for nonsmokers (the level of awareness of eye, nose or throat irritation) were exceeded by more than 90-fold in the smoking sections, and by nearly 35-fold in the nonsmoking sections due to smoke infiltration, despite these premises meeting recommended Australian Standard ventilation rates.

Cains et al. (2004) also measured airborne nicotine levels from SHS. Generalising these levels, using published SHS risk assessment techniques, I estimate that SHS exposure in the workplace causes more than 73 worker deaths per year among the 40,000 Club, Pub, Tavern, and Bar workers in New South Wales.

This estimates deaths from lung cancer and heart disease only and includes nonsmokers and smokers. The estimated range for nonsmoking workers only (never-smokers and ex-smokers) is 18% less (over 59 deaths per year).

These estimates compare to a total of 97 deaths for all occupational fatalities in NSW annually from all other causes.

The risk analysis method used in this report is consistent with the Environmental Tobacco Smoke Harm Index published in Australian Standard 1668.2-2002, Supplement 1, which enables ventilation engineers to estimate the risk from SHS exposure in workplaces as a function of smoking and ventilation rates.

SHS levels increase as ventilation rates decline, and decrease as smoking prevalence declines. In the past decade, Australian design ventilation rates have declined 65% faster than smoking prevalence.

I conclude that smoke-free workplace legislation, like that enacted in the ACT, is urgently needed in New South Wales.

James L. Repace MSc
Repace Associates Inc.
Secondhand Smoke Consultants
April 2004

1. INTRODUCTION

Secondhand smoke (SHS) [also known as environmental tobacco smoke (ETS)] is indoor air pollution from tobacco smoke exhaled by smokers and emitted from the burning ends of cigarettes, pipes and cigars. Secondhand smoke is the most significant source of respirable particulate (RSP) human air pollution exposure and appears, in general, at much higher concentrations indoors, and is far more toxic than outdoor RSP air pollution (Repace and Lowrey, 1980).

Tobacco smoke aerosol is a mixture of gases and particles, and contains more than 4000 chemicals, 500 of which are gaseous [Hoffman & Hoffmann, 1999]. Of these, 133 are known toxic substances, including 3 Criteria Air Pollutants and 33 Hazardous Air Pollutants regulated under the U.S. Clean Air Act, 47 that are classified as Hazardous Wastes whose disposal in solid or liquid form is regulated by the U.S. Resource Conservation and Recovery Act, and 68 that are known human or animal carcinogens, 3 of which are industrial chemicals regulated under the U.S. Occupational Health and Safety Act . Some chemicals fall into more than one category.

The widespread public exposure and high toxicity of SHS have concerned environmental, occupational and public health authorities, and numerous authoritative, peer-reviewed reports have been prepared by national and international bodies, including in the U.S. the Surgeon General (1986), the National Academy of Sciences (1986), the National Institute for Occupational Safety and Health (1991), the Environmental Protection Agency (EPA) (1992), the Occupational Safety & Health Administration (1994), the National Cancer Institute (1993, 1998, 1999), the California EPA (1997, 2004), and the National Toxicology Program (2000). In other countries reports have also been prepared by, the Australian National Health and Medical Research Council (1997), the U.K. Scientific Committee on Tobacco or Health (1998), and the International Agency for Research on Cancer (1987, 2004).

Summing up the conclusions of these various organizations to 2004, nonsmokers' exposure to SHS causes fatal heart disease, lung and nasal sinus cancer, asthma induction and aggravation, middle ear infection, sudden infant death syndrome, and respiratory impairment, as well as irritation of the mucous membranes of the eyes, nose, and throat. SHS exposures can be predicted and generalized by scientific methods.

Estimated total Australian mortality from SHS based on US data

Table 1 gives the estimated mortality from SHS in Australia in 1998 prior to many of the current smoking restrictions. The estimate of ~4200 nonsmokers' deaths per year, is based on scaling of U.S. data and compares to an estimated 60,000 deaths/year in the U.S. and 800 deaths/year in New Zealand, (where bars will go smoke-free in 2005).

This estimation includes 602 Australian deaths from breast cancer which is currently the subject of some discussion. While the State of California, in a draft report (CalEPA, 2004), has stated that SHS causes breast cancer, at the time of writing it remains in public review draft.

Table 1. Passive smoking mortality

Est. Passive Smoking Deaths, 1998

(U.S. Values from AJ Wells, Env. Internat. 25:515-519, 1999)

Cause	U.S.A.	Australia	New Zeal.
Lung Cancer	3060	212	40
Heart Disease	47 000	3252	628
Breast Cancer	8700	602	116
Cervical Ca.	500	35	7
Nasal Sinus Ca.	200	14	3
Brain Cancer, Leukemia, & Lymphoma	1000	69	13
Total	**60 460**	***4184**	***807**
Adult Population, 15+	*205.2 million*	*14.2 million*	*2.74 million*

**[scaled from U.S. by relative population size. J.L. Repace, 1999]*

Eliminating all causes of death except lung cancer and heart disease - for which there is considerable evidence - reduces the estimates by 17.2% or 720 deaths for Australia, to an estimated 3464 deaths per year which still differs significantly from the extremely conservative estimates of 88 deaths per year by the Australian National Health and Medical Research Council (NHMRC, 1997). The difference in estimates can be accounted for by the difference in data sources. The NHMRC estimates of passive smoking were based on exposure to spousal smoking and did not take into account exposure outside the home, or deaths in nonsmokers who were ex-smokers.

Moreover, passive smoking exposure assessment based on reported spousal smoking alone leads to an underestimation of exposure and a further underestimation of risk. The U.S. Centers for Disease Control reported, in a national probability sample of U.S. nonsmokers that, although 88% had detectable levels of the nicotine metabolite, cotinine, in their body fluids, only 40% reported exposure to SHS (Pirkle et al., 1996). The U.S. Environmental Protection Agency's report on passive smoking (U.S. EPA, 1992), which accounted for cotinine dose in "unexposed" nonsmokers, estimated 3000 deaths per year, which when scaled to Australia yields an estimated 212 lung cancer deaths per year, nearly 20 times higher than NHMRC (1997).

2. METHODS

2.1 CALCULATING SECONDHAND SMOKE CONCENTRATIONS

SHS concentrations in commercial buildings are determined by the number of smokers present and their smoking rate, the volume of the space which contains the smokers, and the rate at which SHS pollution is removed by the air-handling systems. SHS concentrations can be measured (e.g., Repace and Lowrey, 1980), and can be estimated in buildings using mathematical models developed by Repace and Lowrey (1980), based on the tobacco product emissions, smoking rates, the size of the room, and the air exchange rates (SG, 1986; NRC, 1986; IARC, 1987).

Non-industrial air handling systems in their simplest form rely upon seepage of air through cracks around closed windows and doors (infiltration), and the opening of a window (natural ventilation). In their more complex forms, air handling systems use mechanical ventilation, which pumps outdoor air through a building, usually with recirculation, in order to dilute indoor contaminants, and to replace a portion of the contaminated indoor air with uncontaminated outdoor air according to the number of outdoor air changes supplied per hour.

Sometimes, air filtration is additionally supplied to lower the concentration of an indoor contaminant without bringing in additional outside air. Air filtration is not as effective as ventilation in removing indoor pollutants, especially for gaseous contaminants, but may be used as a supplement when the existing ventilation system cannot provide sufficient capacity to handle the indoor pollutant loads, for economic reasons, or when a pollutant is entering the building from outdoors, e.g., pollens.

2.1.1 Number of smokers

In general, the number of smokers present in a smoking-permitted bar, club, or casino, and their smoking rate will depend on a variety of factors. Table 2 shows that Australian smokers on average smoke at the rate of slightly less than 2 cigarettes per smoker per hour. It takes on average about 10 minutes to smoke a cigarette and, therefore, the greatest number of cigarettes any smoker can smoke is 6 cigarettes per hour; such persons are called “chain-smokers.”

Table 2 shows that the Australian smoking prevalence in 1995 was 26%, the same as in 1992 (Hill et al., 1998), while by 2001 (Table 3), the prevalence of daily smoking among people aged 14 years and over in New South Wales had declined to 18.1%, a drop of 30%. The prevalence of daily smoking was higher for males than females, whereas the proportions of never smokers were higher for females than males across the country.

For the ages of persons most likely to patronize bars, casinos and clubs, however, the proportion ranges as high as 24% in NSW. In any specific venue

on a given night, the proportion of smokers present, as well as their smoking rates, may vary.

Table 2. Australia cigarette smoking 1995 (CDC, 1999; CDHF, 1996)

- 26% Prevalence (28% Male; 24% Female)
- 3.7 Million Current Smokers **(aged ≥ 15 yrs)**
- 32.5 Billion Cigarettes Smoked/Y (1994)
- 8809 cigarettes per smoker per year
- ~24 cigarettes per smoker per 14 hr smoking day
- 1.7 cigarettes smoked per person per hour
- Smoking Deaths per year in 1995: 18,000

CDC = U.S. Centers for Disease Control, Atlanta, GA
CDHF = Central Data Handling Faciility

Table 3. NSW, Prevalence of daily smoking: proportion of the population aged 14 years and over, by age and sex, New South Wales, 2001 (AIHW, 2001)

Age group NSW	Per cent of Persons
14–19	**13.0**
20–29	**24.0**
30–39	**24.1**
40–49	**20.7**
50–59	**17.5**
60+	**8.4**
All ages	***18.1***

2.1.2 Ventilation rates

Ventilation rates in NSW are currently governed by Australian Ventilation Standard 1668.2—2002, which sets ventilation rates for enclosures in which smoking is not prohibited. The recommended ventilation rates of the previous edition of this standard 1668.2-1991 are compared with its counterparts in the U.S. and New Zealand in Table 4. Note that in 1991 (Table 4) the Australian design ventilation rate for bars was 20 L/s-person, while in 2002, it had declined by 50% to 10 L/s-person.

Table 4. Previous ventilation standards

Australian, New Zealand, and North American Design Ventilation Standards		
Ventilat. Stnd.	Pers./100 m^2	L/s.person
AS 1668.2	*(1991)*	*Australia*
Bar	100	20
Dining	70	15
NZ 4303	*(1990)*	*New Zealand*
Bar	100	15
Dining	70	10
ASHRAE 62	*(1989)*	*North America*
Bar	100	15
Dining	70	10

Standard 1668.2 states that its recommended rates are "based on the amenity effects of environmental tobacco smoke (ETS). The Standard does not address the health aspects of ETS exposure". The standard continues: "Health authorities advise that ETS is associated with serious adverse health effects, including ischaemic heart disease and lung cancer. Users of the Standard are advised to consult relevant Government authorities for details of legislation that deals with public health and occupational health aspects of ETS exposure. Users wanting to calculate an estimate of some of the health risks to occupants of an enclosure where smoking is not prohibited may also consult Appendix A of the Supplement to this Standard, AS 1668.2 Supp 1." AS 1668.2 Supplement 1, is discussed below in the context of risk assessment.

2.1.3 Carbon dioxide (CO_2) levels as an index of ventilation rate

Appendix C of ASHRAE Standard 62-1999 specifies the following equation for C_s, the equilibrium CO_2 levels in parts per million (ppm) in a space:

$$C_s = \frac{N}{V_o} + C_o \qquad \textbf{(Eq. 1),}$$

where N is the CO_2 generation rate per person (N = 5000 ppm-Litres/sec per person, corresponding to office work), V_o is the outdoor air flow rate in Litres/sec per person (L/s-P), and C_o is the CO_2 concentration (ppm) in the outdoor air. Equation 1 is typically used to estimate the flow rate adequacy based upon indoor/outdoor CO_2 measurements.

Note that the flow rate of V_o = 20 L/s-P specified by AS 1668.2-2002 corresponds to a CO_2 level of C_s = (5000/20) + C_o = 250 ppm + C_o, or 250 ppm above outdoor background. For example, Cains et al.(2004), as part of an air quality and SHS study, measured mean CO_2 levels in 17 gaming clubs in the Sydney metro area,

reporting an average value for C_o = 600 ppm outdoors, and averaging about 860 ppm indoors, for a net indoor CO_2 level of 160 ppm. Assuming that these values are close to steady state, the clubs on the whole appear to be ventilated well within AS 1668.2's specifications. This has significant implications for this important air quality study, further discussed below.

2.2 CALCULATING WORKERS' SECONDHAND SMOKE DOSE

Dose from SHS is determined by multiplying the SHS exposure concentration, times a person's respiration rate, and duration of personal exposure. SHS dose is typically assessed by the use of a definitive biological marker called cotinine, which is a metabolic product produced by the body upon inhalation of nicotine in SHS-polluted air. Cotinine, which is oxidised nicotine, persists in body fluids such as blood, urine, or saliva with a 19-hour half-life, which means it is a good marker for SHS exposure within the previous one to two days.

Cotinine doses have been measured for the average nonsmoker and for worker groups, such as flight attendants, bartenders and restaurant workers. In comparing dosimetry in flight attendants to area measurements of nicotine and fine particle pollution reported for aircraft, Repace (2004) found that actual doses corresponded to exposures much higher than indicated by area air pollution monitors for nicotine or particles, because such monitors reflect the well-mixed SHS concentration, and did not take into account the respiration rates or proximity to cigarettes experienced by flight attendants who were enveloped by the concentrated cigarette plume while serving smoking passengers. This is likely to be the case for bartenders and wait staff serving smoking patrons as well.

2.3 CALCULATING WORKERS' MORTALITY RISK

Risk from SHS is addressed in AS 1668.2 Supp 1, which gives an Environmental Tobacco Smoke Harm Index (ETSHI) for the guidance of ventilation engineers and others who would use ventilation measures for indoor contaminant control under AS 1668.2. The ETSHI (¶ A14) bases the harm index on the exposure-response relationships derived in the NHMRC Report (1997), and estimates, as an example, the combined lung cancer and heart disease mortality risk for office workers in a typical smoking-permitted office whose occupancy and ventilation parameters are specified by AS 1668.2: occupancy, 100 persons (10 persons per 100 m^2 of occupiable space); ventilation rate, 10 litres per second per person; 33% smoking prevalence; 2 cigarettes per smoker per hour; 5 m^3/s return airflow with a 35% efficiency return air filter for particles, which reduces the lung cancer risk but not the ischaemic heart disease risk. An emission rate of 13.7 mg of RSP per Australian cigarette is assumed (¶ A10.1).

Under these conditions, assuming a normal 8-hr work-exposure day, a combined mortality rate from SHS is calculated for the nonsmoking office workers: ETSHI

= 225 deaths per million exposed Australian office workers per year.

Repace et al. (1998) estimated a similar harm index for U.S. office workers, using ASHRAE Standard 62-1989, the counterpart U.S. ventilation standard. The occupancy, was 10 persons per 1000 ft^2 (about 10 persons per 100 m^2 of occupiable space); ventilation rate, 10 litres per second per person; 29% smoking prevalence; 2 cigarettes per smoker per hour; no effective filtration for SHS aerosol. It can be shown that an emission rate of 13.7 mg of RSP per U.S. cigarette is consistent with this exposure model.

Under these conditions, assuming a 7-hr work/exposure day, 260 days per year, a combined mortality rate from SHS was calculated for the nonsmoking office workers: a 45-year working lifetime (WLT_{45}) risk of 11 deaths per 1000 workers, or on an annual basis, 244 deaths per million exposed U.S. office workers per year.

Thus using similar assumptions, Repace et al. (1998) and the ETSHI method of AS 1668.2 Supp 1 yield similar risk estimates. A 45-year working lifetime is a standard default assumption of the U.S. OSHA when calculating risk to workers (Repace et al., 1998).

Repace et al. (1993) estimated a WLT_{45} for workers exposed to an 8-hr time-weighted average (TWA) nicotine concentration of 6.7 micrograms of nicotine per cubic meter ($\mu g/m^3$) of 1 lung cancer death per 1000 workers; when coupled to the heart disease risk model of Repace et al. (1998), 6.7 $\mu g/m^3$ of nicotine 8-hr TWA for a WLT_{45} yields an estimated 11 deaths per 1000 workers, from heart disease and lung cancer mortality combined. This risk model, a mathematical way of estimating risk from exposure, permits estimates of risk based on a worker's average working lifetime exposure to SHS nicotine.

Expressed in terms of a unit nicotine concentration, this is 1100 deaths per 100,000 workers per WLT_{45} per 6.7 $\mu g/m^3$ nicotine, for an estimated exposure-response relationship of 164 deaths per 100,000 per 1 $\mu g/m^3$ nicotine per 8-hr TWA exposure day, or 6 hours per day, annualized average, taken over men and women workers, which is equivalent to 288 days per year exposure at 8 hr/day, and is based on actual multi-national sociological time-use studies of workers (Repace and Lowrey, 1985).

3. ESTIMATION OF HEALTH RISK IN 17 NSW VENUES

3.1 INHALABLE PARTICULATE AIR POLLUTION

Cains et al. (2004) measured air quality indoors and outdoors [inhalable particulate matter (PM_{10}), airborne nicotine, and carbon dioxide (CO_2)] for a representative sample of 17 social and gaming clubs in Sydney, NSW. All clubs

were licensed to serve alcohol, all provided gaming machines as a principle recreation for patrons, and smoking occurred in all.

Monitors were centrally located in the room with gaming machines. Outdoor measurements were made in an area adjacent to the club building, but remote from human or vehicular traffic and from building ventilation exhaust.

These clubs catered to diverse interests, including football, lawn bowling, golf, veterans, social and community improvement, and had both smoking and nonsmoking areas of various kinds. Measurements were undertaken at a time of maximal occupancy as advised by the club management; typically, a Friday evening.

Cains et al. (2004) concluded that designated "no-smoking" areas may provide some reduction in the level of exposure to SHS, but that such reduction may often be marginal or trivial. They also concluded that persons working in the smoking areas in such clubs, e.g., bartenders and waiters, may be heavily exposed to SHS.

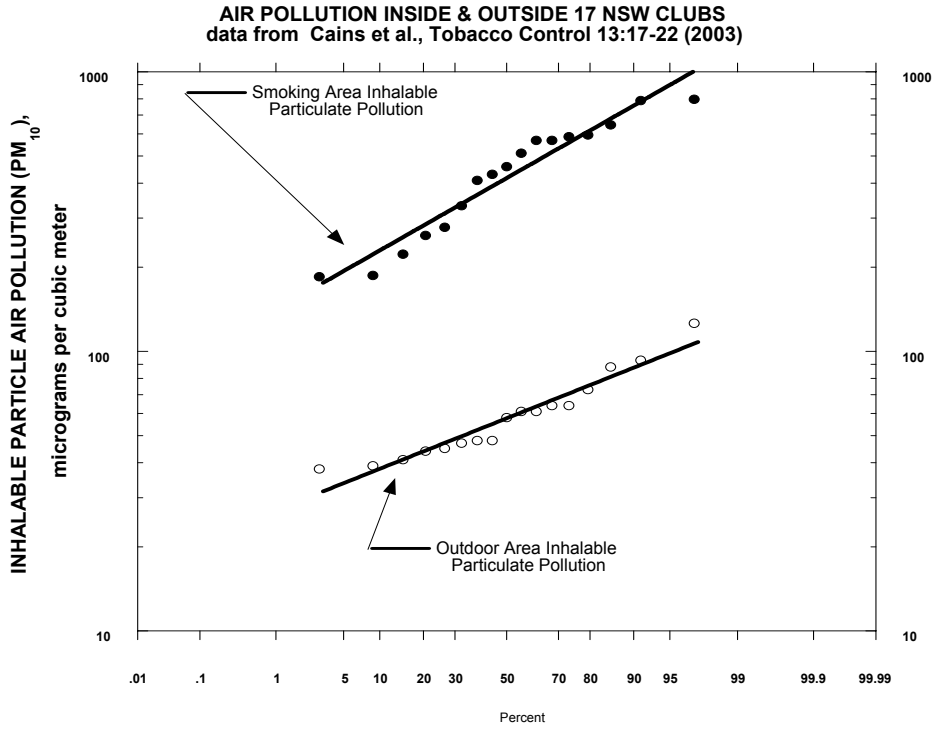

Figure 1. Inhalable particle pollution (particulate matter 10 microns in aerodynamic diameter or less, called PM_{10}) in 17 Sydney metro area gaming clubs in the smoking area on a night of maximal occupancy. as measured by a Dust-Trak Aerosol monitor; each data point represents 10 minute samples taken in the smoking area (solid circles) or outdoors (open circles).

Using the Cains et al. (2004) data, I constructed log-probability plots to ascertain the probability of encountering a given concentration of particulate air pollution, indoors and outdoors (Figure 1). Log-probability plots are useful because atmospheric pollutants are log-normally distributed, and when plotted on such a graph, the data will lie along a straight line. This method permits displaying every data point with the corresponding probability of such a concentration occurring, conveying more statistical information than a simple mean plus standard deviation. For example, Figure 1, at the 90th percentile of the smoking area data, shows that 10% of the workers are exposed to a smoking-area air pollution level greater than 800 $\mu g/m^3$, and that 100% of the workers are exposed to air pollution levels exceeding 185 $\mu g/m^3$, whereas if the indoor air pollution level were no higher than outdoors, e.g., as with a smoking ban, then 100% of the workers would be exposed to concentrations less than 126 $\mu g/m^3$.

PM_{10} levels in the 17 Clubs averaged 460 (SD 196) $\mu g/m^3$ in smoking areas and 210 (SD 210) $\mu g/m^3$ in designated nonsmoking areas, compared to 61 (SD 23) $\mu g/m^3$ outdoors. How do these levels compare with RSP levels measured in other hospitality venues? The U.S. EPA (Fig. 3-8, 1992) reported a range of 40 $\mu g/m^3$ to 986 $\mu g/m^3$ for average values of RSP in restaurants; the measurements of Cains et al. (2004) lie comfortably within this range.

Comparing indoor/outdoor means shows that smoking caused 86% of inhalable particle air pollution (PM_{10}) in the smoking sections and 71% of PM_{10} in the nonsmoking sections. The air in these 17 smoking-permitted clubs is heavily polluted, placing both workers and patrons at risk of air-pollution induced disease. Moreover, SHS is a well-established sensory irritant, variously producing itching, tearing, burning, swelling of eyes, sneezing, blocking, running, itching of nose, headache, cough, wheezing, sore throat, nausea and dizziness, and respiratory discomfort (SG, 1986; NRC, 1986; EPA, 1992; Speer, 1968; Savel, 1970).

A Swiss study by Junker et al. (2001) reported an odor acceptability threshold of 1 $\mu g/m^3$ SHS-RSP, and a SHS-RSP irritation threshold level of 4.4 $\mu g/m^3$. Even at that 4.4 $\mu g/m^3$ SHS-RSP level, only 33% of nonsmoking test subjects found the air quality acceptable, as many nonsmokers find the odor of SHS to be foul, and the SHS odor detection threshold in this study was 1 $\mu g/m^3$. The average smoking section SHS-RSP level of (460 – 61) 399 $\mu g/m^3$ is more than 90 times the irritation threshold, and in the *non-smoking* section, the SHS-RSP pollution level of (210 – 61) 149 $\mu g/m^3$ for the 17-Club average is about 34 times the Swiss study's eye, nose, and throat irritation threshold. Peak SHS-RSP pollution levels are more than 6-fold higher than the outdoor mean, as illustrated in Figure 1.

3.2 ATMOSPHERIC NICOTINE

Cains et al. (2004) reported that the atmospheric nicotine levels in areas where smoking occurred ranged from 37 $\mu g/m^3$ to 199 $\mu g/m^3$ in smoking areas and from 23 $\mu g/m^3$ to 71 $\mu g/m^3$ in contiguous nonsmoking areas, and had mean nicotine levels of 100.5 (SD 45.3) micrograms per cubic meter ($\mu g/m^3$), and 41.3 (16.1) respectively in designated "smoking" and "no smoking" areas. Cains et al. (2004) concluded that at best only partial protection from SHS is achieved by designated "no smoking" areas, and this is in no way comparable to the protection afforded by smoking bans on premises.

How do the area nicotine measurements of Cains et al. (2004), which ranged from 23 $\mu g/m^3$ to 199 $\mu g/m^3$ for licensed clubs compare with those of other studies? Hammond (1999) reported a range of 1 $\mu g/m^3$ to 65 $\mu g/m^3$ for nicotine average measurements in a wider variety of establishments, including 28 bars, restaurants, taverns, cocktail lounges nightclubs, and gaming facilities. However, it should be noted that a number of the studies reviewed were funded by the tobacco industry. Industry-funded studies often report conclusions such as "bartenders are not exposed to levels as high as estimated by OSHA" (Jenkins and Counts 1999), or "ETS exposures are extremely low" (Oldaker, et al., 1990). Schorp and Leyden (2002) more recently reviewed 33 studies which measured nicotine in the hospitality industry around the world. These studies report a similar range. However, an examination of the references shows that (19/33) or 58% were recognizable by this observer as performed by the tobacco industry or its consultants.

From the Cains et al. (2004) study, I also plotted the probability of encountering a given concentration of SHS nicotine in a smoking section (Figure 2), and estimated the commensurate risk to workers exposed for an 8-hr work shift for a working lifetime of 45 years. The exposure-response relationship used is 1 $\mu g /m^3$ nicotine $\Leftrightarrow$ 164 deaths per 100,000 Workers per WLT_{45}.

For workers who work full-time in smoking areas, their mean lifetime risk is (100 $\mu g/m^3$ nicotine)(164 deaths per 100,000 workers per 1 $\mu g/m^3$ nicotine per WLT_{45}) = 164 deaths per 1000 workers per 45 years, or 3.64 deaths per 1000 workers per year. Similarly, for club workers exposed to SHS in the nonsmoking areas of these clubs, the risk is (41.3 $\mu g/m^3$ nicotine)(164 deaths per 100,000 workers per 1 $\mu g/m^3$ nicotine per WLT_{45}) = 68 deaths per 1000 workers per 45 years, or 1.5 deaths per 1000 workers per year.

There were an average of 90 persons in the smoking sections of the Clubs, and 38 persons in the nonsmoking areas. Calculating a weighted mean, assuming that workers are distributed between smoking and nonsmoking areas according to the number of patrons, we get [(3.64)(90) + (1.5)(38)]/(128) = 3 SHS deaths per 1000 workers per year. These risks would accrue to all workers, whether smokers or nonsmokers.

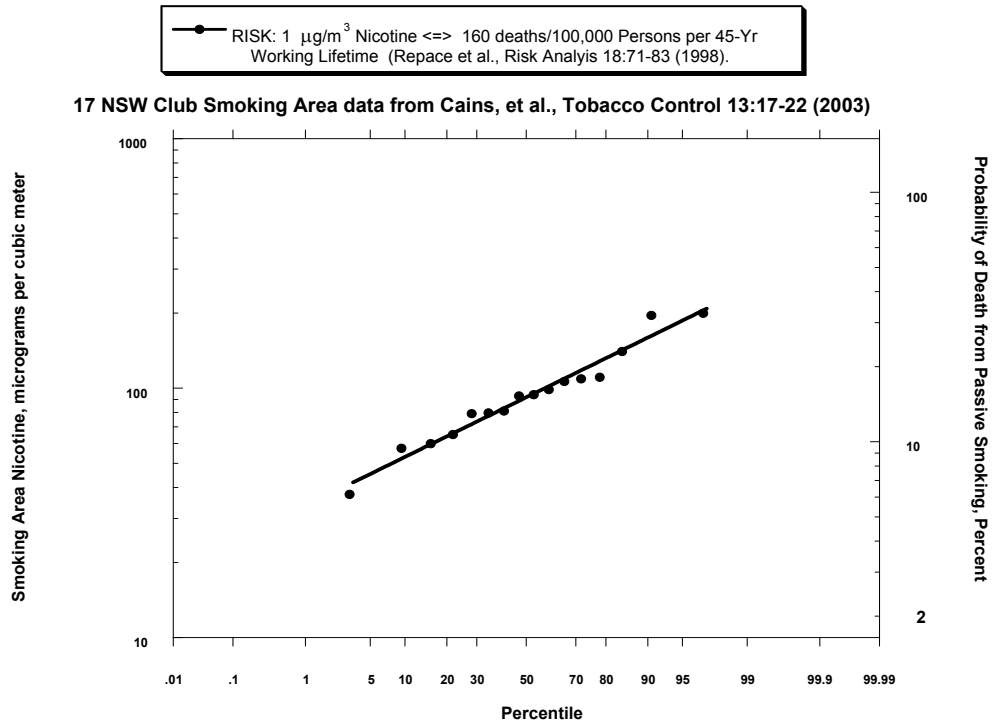

Figure 2. 6-hour ave. nicotine concentrations measured in 16 Sydney metro area gaming clubs in the smoking area on a night of maximal occupancy (one datum is missing). Concentrations are shown on the left vertical axis, and estimated risk (i.e., lifetime probability of a passive-smoking-related death) for Club workers on the right vertical axis. The horizontal axis gives the percent of workers below each level of exposure and risk. Workers at the 95th percentile have a 30% chance of a SHS-caused death.

4. GENERALISING THE 17 CLUB STUDY RESULTS TO OTHER VENUES

AS 1668.2-2002 specifies that for all smoking not prohibited establishments, a recommended outdoor air supply rate is 10 L/s per occupant for a single enclosure, or 15 L/s per occupant for a multiple enclosure, and under section 4.8 prescribes that the minimum flow rate of outdoor air supplied shall be given by the maximum occupancy times the recommended flow rate per occupant. Note that the current version of AS1668.2 has lower ventilation rates than the 1991 version (Table 4). Increases in the cost of fuel have caused a decline in ventilation rates (Repace, 2004).

Use of AS 1668.2's prescriptive procedure results in an equal or higher minimum outdoor requirement than the alternative engineered procedure, which permits recirculation of air if recycle air cleaning is provided. Since the ventilation rates are on a per-occupant basis, although lower design occupancies will have a lower outdoor air supply rate, they will also have a lower pollution generation rate, assuming a constant smoking rate. Standard 1668.2 posits a smoking rate of 1 cigarette per person-hour (smokers and nonsmokers combined); thus, an

occupancy-based ventilation rate should in principle result in an identical smoke concentration for all venues irrespective of design occupancy. This means that the pollution results for the clubs are generalisable to other venues permitting smoking, such as pubs, taverns, bars, and the like.

5. ESTIMATION OF MORTALITY RISK FOR BAR WORKERS ACROSS NSW

We now turn to the number of workers in the NSW hospitality industry. Table 5 gives the number of workers in the NSW club, pub, tavern, and bar industries. Of these, it appears that there are about 40,000 workers, if one includes bar workers in the Star City Casino. If we assume that all workers are distributed during work in smoking and nonsmoking zones to the same extent as in the 17 clubs, and that the nicotine concentrations measured apply for the entire work-week, applying the risk model from section 3.2 yields: (3 deaths per 1000 workers per year) (40,000 workers) = 120 deaths per year from SHS, assuming exposure at the same level for 40 hrs/week.

Table 5. Workers in the NSW club, pub, tavern, and bar industries (ABS 2001)

Cafes and restaurants	62,040 persons	(not affected by passive smoking as smoking is banned in eating areas)
Star City Casino	2,252	(moderately affected – smoking is banned in table gaming areas but not fully in electronic gaming or bars)
Clubs	25,598	(affected – few smoke free)
Pubs, taverns & bars	14,168	(affected – few smoke free)
Total employment NSW (all persons, all industries)	6,371,750	

However, from Table 6, Australian Accommodation, Café and Restaurant workers averaged 32.23 hours per week, and assuming this is valid for NSW, this adjusts the estimated deaths by (32.23/40)(120) = 97 estimated deaths per year in NSW among the 40,000 club, pub, tavern, and bar workers; this includes never-smokers, ex-smokers, and smokers. At the current smoking prevalence of 18.1%, this reduces to 79 deaths per year among nonsmokers only.

Table 6. ABS Labour Force Vol 6203.0
Nov 2002 (hours worked by industry)

Accommodation, café and restaurant group - AUSTRALIA wide figures

Average weekly hours worked: 32.23 hours

Numbers of people working:

0 hours	17,500
1 - 15 hours	101,300
16-29	84,600
30-34	42,300
35-39	47,300
40	46,500
40-44	15,500
45-48	22,900
49 and over	83,100

This estimate does not take into account industry turnover. According to the Australian Bureau of Labour Statistics, for persons employed in Accommodation, Cafés, and Restaurants, 20.0% left the industry in 2001, and 26.6% left the industry in 2002 (ABS, 2004, Table 6.22, changes in employment). If this applied to all workers in these hospitality industries, it would appear to signify a complete workforce turnover every 4 to 5 years, compared to turnover for all industries of 12.4%, implying a complete turnover every 8 years.

However, an examination of statistics by age group shows that there is a strong age-related component to job hospitality industry turnover, with those in age groups 15-34 accounting for the bulk of the turnover, at rates of 19% to 26%, compared to 6% to 13% among ages 35 to 69, with all age groups averaging 15% (ABS, 2004, Duration of current job). These statistics could be interpreted as signifying that the high turnover rates in the hospitality industry are limited to the younger age groups, and it is likely that the very high turnover rates in the hospitality industry are confined to the age groups 15-34.

Thus, if this assumption is made, the estimated annual mortality rates should be reduced by about 25% to account for the loss of the younger ages, who seek temporary employment while in school or for other reasons. Reducing the estimate of 97 deaths by 25% yields an estimated 73 deaths per year for all hospitality workers regardless of smoking status, or 59 deaths per year in nonsmokers (never-smokers plus ex-smokers) only.

In summary, I estimate the annual mortality from passive smoking for all 40,000 NSW hospitality workers regardless of smoking status, to range from 73 to 97

deaths per year, or 18% less for nonsmokers (never-smokers plus ex-smokers) only, at 59 to 79 deaths per year.

6. COMPARISON WITH OTHER AUSTRALIAN INDUSTRIES

To place these estimates into perspective, 97 deaths per year is equal to the total mortality for NSW workers from all occupational causes in the workplace for all industries (Table 7). Thus the range of 73-97 estimated annual deaths from SHS is 75% to 100% of all NSW occupational mortality, and is also larger than any individual cause of occupational death, including such common ones as being hit by moving objects, falls, vehicle accidents, and electrocution. By comparison, the incidence of mesothelioma from asbestos in all of Australia in the retail trade in Accommodation, Cafés and Restaurants is only 4 cases over a 3-year period 1998-2000, or 1.25 cases per year (NOHSC, 2003). Thus annual estimated mortality from SHS in the hospitality sector ranges from (73/1.25) 58 to (97/1.25) 78 times the number of mesothelioma cases in all of Australia in this sector. In 2002 there were 461,100 people employed in the accommodation, café and restaurant sector across Australia.

Table 7. Mechanism of fatal incident – workplace and working deaths. Number, NSW, 1989 to 1992, workplace only (Table 10, NOHSC, 1999).

Mechanism	**Average number per year (rounded)**
Being hit by moving objects	37
Falls	14.25
Contact with electricity	10.25
Vehicle accident	12.25
Weapons	6.5
Rollover	4.25
Drowning	4
Chemicals and other substances	3.25
Contact with heat or cold	1.75
Hitting objects with part of body	1.5
Slide/cave-in	1.25
Explosion	0.75
Other and multiple	0.25
All	***97.25***
Secondhand Smoke	**73 - 97 (estimated, this work)**

7. DISCUSSION OF UNCERTAINTY

7.1 NICOTINE LEVELS

I assumed that the measured nicotine concentrations at a time of maximal occupancy apply to the remainder of the week. This is a prudent measure for public health purposes when estimating risk. Nevertheless, this assumption may actually be conservative. The RSP and nicotine concentrations reported by Cains et al. (2004) are measured by area monitors remote from the club attendants' breathing zones, where they encounter more concentrated cigarette plumes as they serve club members. Servers and bartenders may be exposed to tobacco smoke of members at distances approaching 0.5 m. Several studies have shown that increasing proximity to a pollution source increases exposure – Repace and Lowrey (1982), McBride, et al., (1999, 2002) and Klepeis, et al. (2004) all found that the concentration of RSP increased by 2 to 5 times as the exposure distance decreased from $\geq$2 m to 0.5 m while the pollution source was on. These effects were also observed for carbon monoxide and carcinogens (McBride et al., 1999).

Nevertheless, even if the average weekly concentration in the typical club, pub, tavern or bar venue were half of that measured by Cains et al. (2004) during the high occupancy period, the estimated annual mortality at 30-49 deaths per year would be larger than any other single cause of worker mortality, and still even at the lowest level (30/1.25) 24-fold the number of mesothelioma cases annually in these hospitality workers all over Australia.

7.2 MORTALITY FROM SHS IN SMOKERS

The estimates of mortality from SHS for smokers is justified by 3 complementary lines of evidence: lung cancer rates for active smokers who do not inhale (both cigarette and cigar smokers) that are substantial fractions of those who report inhaling (SG, 1978), and exposure-response relationships reported for active smokers who live with smokers relative to those who do not (Sandler, et al., 1985). The predictions of the risk models of Repace et al. (1985, 1993, 1998) of 440 lung cancer deaths/year and 4400 heart disease deaths per year for U.S. workers, compared with the midpoint of U.S. OSHA's published estimates for U.S. workers, differ by 2% with OSHA's lung cancer estimates, and are 42% below OSHA's midrange heart disease estimates, a result of the more conservative model used by Repace et al. (1998).

7.3 AVERAGE WORKING LIFE ESTIMATES

Although the lower bound risk estimate is intended to reflect the effect of hospitality industry turnover, even transient employment in the hospitality industry might impact heart disease morbidity and mortality. Sargent et al. (2004) in a very recent study, investigated whether there was a change in hospital admissions for acute myocardial infarction while a local law banning smoking in public places and in workplaces was in effect. The study recorded hospital

admissions from December 1997 through November 2003, in Helena, Montana, a geographically isolated community with one hospital serving a population of 68 140.

Sargent et al. (2004) found that during the six months the law was in effect, a statistically significant decrease of 16 admissions [95% confidence interval (CI): -31.7 to -0.3] occurred, from an average of 40 admissions during the same months in the years **before and after** the law to a total of 24 admissions during the six months the law was in effect. There was a non-significant increase of 5.6 in the number of admissions from outside Helena during the same period, from 12.4 in the years before and after the law to 18.0 while the law was in effect. Sargent et al. (2004) concluded that smoke-free workplaces and public places may effect heart disease morbidity.

8. CONCLUSIONS

1. Secondhand Smoke (SHS) contains 133 known toxic substances, among which are a number of substances regulated by law in other settings, including: 33 Hazardous Air Pollutants (pollutants which can cause cancer), 47 that are classified as Hazardous Wastes whose disposal is restricted, and 68 others that are known human or animal carcinogens.
2. SHS exposure has been widely condemned as a hazardous substance by national and international occupational health, environmental health and public health authorities.
3. Australian design ventilation rates for bars have declined by 50% since 1991, while smoking prevalence has declined by 30% since 1992. Thus ventilation has declined >65% faster than smoking.
4. Australian ventilation engineers have issued an Environmental Tobacco Smoke Harm Index to predict the risk of SHS exposure as a function of smoking and ventilation rates. Although derived differently, its predictions closely match those of a published SHS risk model used by the author of this report.
5. A New South Wales (NSW) study by Cains et al.(2004) reported SHS particulate pollution levels which, by my analysis, exceeded SHS irritation thresholds by >90 fold in the smoking sections, and by ~35 fold in the nonsmoking sections of 17 gaming clubs due to smoke infiltration, despite meeting recommended ventilation rates as indicated by measured carbon dioxide levels.
6. Without restrictions on smoking in public places, an estimated 3500 nonsmoking Australians (never-smokers and ex-smokers) would die annually from SHS exposure at home at work and in other venues, much higher than the very conservative NHMRC estimates, which are based on spousal smoking and apply to never-smokers only.
7. A risk assessment generalizing the nicotine levels reported in the Cains et al.(2004) 17 Club study shows that, at the measured level of SHS exposure, an estimated 73-97 deaths per year occur among the 40,000 NSW Club, Pub, Tavern, and Bar workers for all nonsmokers and smokers combined. This range is reduced 18% for nonsmokers only. 97 deaths per year is an amount is equal to that of all annual occupational fatalities in NSW.
8. Even if the average nicotine levels in all NSW hospitality venues were half of those measured by Cains et al. (2004), SHS would still cause ~60 to 80 times the annual mesothelioma mortality caused by asbestos exposure among all Australian accommodation, café, and restaurant workers averaged for the period 1998-2000.
9. New York City reported that a workplace smoking ban improved air quality in bars and restaurants, decreased worker SHS dose by 85%, and that both business receipts and employment increased in bars and restaurants.
10. A total smoking ban extending to hotels, registered clubs, and nightclubs, like that enacted in the ACT is justified in NSW.

REFERENCES

1. ABS 2001. Australian Bureau of Statistics. Census of Population and Housing - (industry of employment, persons, NSW)

2. ABS 2004. Australian Bureau of Statistics. Labour mobility; 6.21, Job mobility by age group and duration of current job.

3. AIHW. Smoking prevalence from the Australian Institute of Health and Welfare publication 2001 National Drug Strategy Household Survey - State and Territory Supplement (page 3) - http://www.aihw.gov.au/publications/phe/ndshs01sts/ndshs01sts.pdf

4. American Society of Heating, Refrigerating, and Air Conditioning Engineers) *Ventilation for Acceptable Indoor Air Quality, ASHRAE Standard 62-1989,* Atlanta, GA, 1989.

5. 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)

6. AS 1668.2—2002. (Incorporating Amendment Nos 1 and 2) Australian Standard™ The use of ventilation and air conditioning in buildings Part 2: Ventilation design for indoor air contaminant control (excluding requirements for the health aspects of tobacco smoke exposure).

7. Australian Bureau of Statistics. ABS statistics on clubs, pubs, taverns and bars in Australia http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/227FDC4C7EAAF19FCA2568A9001393F4

8. Australian Institute of Health and Welfare. 2001 National Drug Strategy Household Survey State and Territory supplement, DRUG STATISTICS SERIES Number 10, August 2002. Canberra, AIHW cat. no. PHE 37.

9. Australian National Health and Medical Research Council. The health effects of passive smoking – A scientific information paper. <http://www.nhmrc.gov.au/advice/nhmrc/foreword.htm>

10. Bernert JT, McGuffey JE, Morrison MA, Pirkle JL. Comparison of serum cotinine and salivary cotinine measurements by sensitive high-performance liquid chromatography-tandem mass spectrometry method as an indicator of exposure to tobacco smoke among smokers and nonsmokers. J Analytical Toxicology 24: 333-339 (20002).

11. Cains T, Cannata S, Poulos R, Ferson MJ, Stewart BW. Designated ‘‘no smoking’’ areas provide from partial to no protection from environmental tobacco smoke. Tobacco Control 13:17–22 (2004).

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

13. CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY. (2004). *Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant.* Office of Environmental Health Hazard Assessment. Public Review Draft. www.arb.ca.gov/toxics/ets/dreport/dreport.htm

14. CDC: National report on human exposure to environmental chemicals. CDC environmental health. NCEH Pub. No. 01-0164, March 2001.

15. CDC: Tobacco or Health, A Global Status Report. Country Profiles, 1999; CDC Atlanta GA, U.S.A.

16. Commonwealth Dept of Health & Family Services, Annual Report 1995-1996.

17. Hammond SK. Exposure of US workers to environmental tobacco smoke. Environmental Health Perspectives 107(suppl 2):329-340 (1999).

18. Hill DJ,White VM, Scollo MM. Smoking behaviours of Australian adults in 1995: trends and concerns. MJA 1998; 168: 209-213.

19. Hoffmann D and Hoffmann I. Significance of exposure to sidestream tobacco smoke. Ch. 1, in *IARC Scientific Publications no.81, Environmental Carcinogens--Selected Methods of Analysis--Volume 9 Passive Smoking; O'Neill I, Brunnemann K, Dodet B, and Hoffmann D.* International Agency for Research on Cancer, World, Health Organization, United Nations Environment Programme, Lyon, France; 1987.

20. *IARC Scientific Publications no.81, Environmental Carcinogens--Selected Methods of Analysis--Volume 9 Passive Smoking; O'Neill I, Brunnemann K, Dodet B, and Hoffmann D.* International Agency for Research on Cancer, World, Health Organization, United Nations Environment Programme, Lyon, France; 1987.

21. Jenkins RA, Counts RW. Occupational exposure to environmental tobacco smoke: results of two personal exposure studies. Environmental Health Perspectives 107 (suppl 2): 341-348 (1999).

22. Junker MH, Danuser B, Monn C, Koller T. Acute sensory response of nonsmokers at very low environmental tobacco smoke concentrations in controlled laboratory settings. Environmental Health Perspectives 109: 1045-1052 (2001).

23. Klepeis NE, Ott WR, Switzer P. Real-time monitoring of outdoor environmental tobacco smoke concentrations, a pilot study. Stanford University Dept. of Statistics, March 1, 2004.

24. McBride SJ, Ferro AR, Ott WR, Switzer P, :Hildemann LM. Investigations of the proximity effect for pollutants in the indoor environment. J Exposure Analysis & Environmental Epidemiology (1999) 602-621.

25. McBride SJ. A marked point process model for the source proximity effect in the indoor environment. J. American Statistical Assn. 97 (2002) 683-691.

26. National Academy of Sciences. *Environmental tobacco smoke -- measuring exposures and assessing health effects.* National Academy Press, Washington, DC. 1986(b).

27. 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.

28. National Cancer Institute. *Changes in Cigarette-Related Disease Risks and their Implication for Prevention and Control.* National Cancer Institute Monograph 8, NIH Publication # 97-1213, National Institutes of Health, Bethesda, MD., February 1997.

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

30. National Occupational Health and Safety Commission, The Incidence of Mesotheliioma in Australia 1998 to 2000, June 2003, Australian Mesothelioma Register Report 2003, Canberra, ACT, 2601.

31. National Occupational Health and Safety Commission, Work-related traumatic fatalities in New South Wales, 1989 to 1992 Information from the second work-related fatalities study, 1989 to 1992, December 1999, National Occupational Health and Safety Commission Sydney.

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

33. 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.

34. *New York City Departments of Finance, Health & Mental Hygiene, Small Business Services, Economic Development Corporation.* THE STATE OF SMOKE-FREE NEW YORK CITY: A ONE-YEAR REVIEW *March* 2004.

35. NHMRC. Australian National Health and Medical Research Council The health effects of passive smoking - A Scientific Information Paper November 1997

36. 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.

37. NOHSC. Australian Government National Occupational Health and Safety Commission. http://www.nohsc.gov.au/statistics/wrf2ncis.htm

38. NOHSC. Australian Government National Occupational Health and Safety Commission. The incidence of mesothelioma in Australia 1998-2000. Australian Mesothelioma Register Report 2003.

39. Oldaker GB, Ogden MW, Maiolo KC, et al. Results from surveys of environmental tobacco smoke in restaurants in Winston-Salem, North Carolina. Indoor Air '90, Proc. of the 5th International Conference on Indoor Air Quality and Climate, Vol 2, 281-285, Toronto, 29 July - 3 August 1990.

40. OSHA. 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.

41. Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the populati environmental tobacco smoke: the third national health and nutrition examination survey, 1 1991. JAMA 275: 1233- 1240 (1996).

42. 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 1998; 18: 71-83.

43. Repace JL, Lowrey AH. Indoor Air Pollution, Tobacco Smoke, and Public Health, SCIENCE 208: 464-474 (l980).

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

45. Repace JL. Flying the Smoky Skies: Secondhand Smoke Exposure of Flight Attendants. *Tobacco Control* 13(Suppl 1) i8-i19 (2004).

46. Sandler DP, Everson RB, Wilcox AJ. Passive smoking in adulthood and cancer risk. Am J. Epidemiol. 121:37-48 (1985).

47. Sargent RP, Shepard RM, Glantz SA.Reduced incidence of admissions for myocardial infarction associated with public smoking ban: before and after study. BMJ, doi:10.1136/bmj.38055.715683.55, published 5 April, 2004.

48. Savel H. Clinical hypersensitivity to cigarette smoke. Archives of Environmental Health 21: 146-148 (1970).

49. Schorp MK, Leyden DE. Distribution analysis of airborne nicotine concentrations in hospitality facilities. Environment International 27:567-578 (2002).

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

51. Speer F. Tobacco and the nonsmoker. A study of subjective symptoms. Archives of Environmental Health 16: 443-446 (1968).

52. Surgeon General. *Smoking and Health A Report of the Surgeon General.* U.S. Dept. of Health Education, and Welfare, Washington, DC (1979).

53. 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).

54. 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).

APPENDIX 1: THE NEW YORK CITY EXPERIENCE

When the Smoke-Free Air Act went into effect in new York on March 30, 2003, questions were raised about the impact of the smoking ban on staff health as well as how the law would affect the City's restaurants and bars. One year later, New York City issued the following statistics (NYC, 2004):

- **97% of restaurants and bars are smoke-free**
- **New Yorkers overwhelmingly support the law**
- **Air quality in bars and restaurants has improved dramatically**
- **Levels of cotinine, a by-product of tobacco, decreased by 85% in nonsmoking workers in bars and restaurants**
- **150,000 fewer New Yorkers are exposed to second-hand smoke on the job**
- **Business tax receipts in restaurants and bars are up 8.7%**
- **Employment in restaurants and bars has increased by 10,600 jobs (about 2,800 seasonally adjusted jobs) since the law's enactment**

SHS-RSP levels were measured by the New York City Health Department, which found that post-ban RSP ($PM_{2.5}$) levels declined to 1/6th of pre-ban levels. The level of cotinine in the saliva of nonsmoking bar and restaurant workers declined dramatically: the results of the cotinine dosimetry study are shown in Figure 3. To place these results into further perspective, in 2001, the 90th percentile of serum cotinine for the U.S. population was 0.52 nanograms per millilitre (ng/mL) [95% confidence interval 0.38-1.01], where saliva cotinine is roughly equal to serum cotinine (CDC, 2001; Bernert et al., 2000). Thus, before the ban, New York State bar and restaurant workers had SHS exposures about (6.6-1)/0.52) 10 times higher than 90% of the U.S. population. Given the similarity between U.S. and Australian air exchange rates and smoking prevalence, this is almost certainly to be the case in Australia as well.

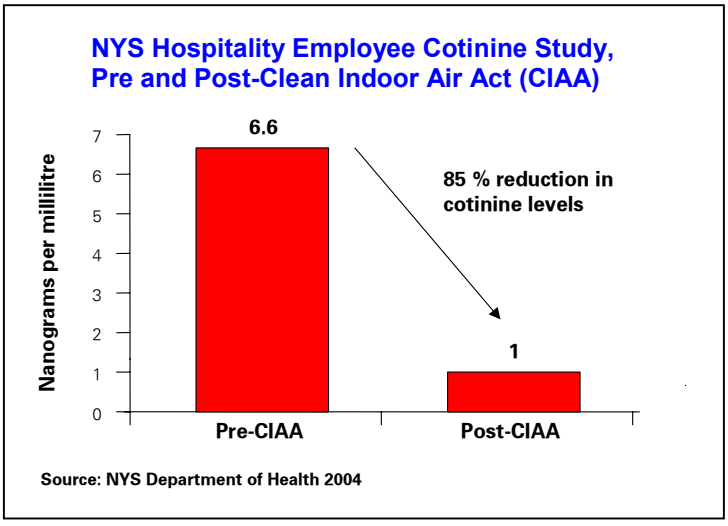

Figure 3. Cotinine, a nicotine by-product, is found in people who have inhaled tobacco smoke, and is used to determine nonsmokers' exposure to second-hand smoke. Researchers collected saliva cotinine from nonsmoking bar and restaurant employees during the month before the New York State Clean Indoor Air Act (CIAA) went into effect in July 2003, and again three months later. They found that cotinine levels declined by 85% after the state law went into effect (NYC, 2004).