Environmental Tobacco Smoke in Indoor and Outdoor Public Places

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Executive Summary

The objectives of this report were to (i) review the epidemiologic studies of Environmental Tobacco Smoke (ETS) exposures in the workplace and home settings to evaluate the extent to which these findings could be generalizable to outdoor and other settings where ETS exposure occurs, (ii) summarize the peer-reviewed scientific evidence on the health effects of ETS exposure in indoor and outdoor public spaces, and (iii) review and summarize the exposure levels and associated levels of risk.

The summary of epidemiologic studies of the health effects of ETS exposures in the workplace and home settings is based on reviews of reviews. Six independent government and national scientific reviews were completed in the 1990s in the US (USEPA[1], CalEPA[3], NTP[44]), Australia (NHMRC[42]), United Kingdom (SCOTH[43]) and internationally (WHO[41]). Each focused on a different number and types of health endpoints that are listed in Appendix Table A. The health effects of exposure to ETS in these epidemiologic studies are based on non-smoking spouses of smokers or non-smokers exposed in the workplace. ETS exposure in these settings is generally of long duration, and there is a latency effect associated with these health endpoints. These reviews concluded that exposure to ETS is causally related to the following health outcomes:

Developmental effects

- fetal growth (low birth-weight or small for gestational age),
- sudden infant death syndrome (SIDS),

Respiratory effects in children

- acute lower respiratory tract infections in children (e.g. bronchitis and pneumonia),
- asthma exacerbation in children
- chronic respiratory symptoms in children
- middle ear infection in children (otitis media),

Carcinogenic effects

- lung cancer,
- nasal sinus cancer

Cardiovascular effects

- coronary heart disease.

There are a number of health effects for which evidence is suggestive of a causal association. These include:

Reproductive and Developmental Effects

- spontaneous abortion
- adverse impact on cognition and behaviour
- decreased pulmonary function in children

Respiratory Effects

- Exacerbation of cystic fibrosis
- Asthma induction in children

Carcinogenic Effects

- Cervical Cancer

In 2004 the International Agency for Research on Cancer[26] (IARC) also concluded that there is a statistically significant and consistent association between lung cancer risk in spouses of smokers and exposure to secondhand smoke at home and in the workplace. Other important conclusions relating to carcinogenic effects were:

- The evidence linking ETS exposure to breast cancer is inconsistent,
- The evidence linking childhood cancer and ETS exposure from parental smoking is inconsistent.
- Data relating to the following cancers among adults were considered sparse, inconsistent and bias could not be ruled out: cancers of the naso-pharynx, nasal cavity, paranasal sinuses, cervix, gastrointestinal tract and cancers of all sites combined.

The following non-cancer health effects of ETS exposure were reached in the IARC (2004) review:

- Exposure to ETS increases the risk of a coronary heart disease event by 25 -30% for non smokers living with a smoking spouse compared to non-smokers living with a non-smoking spouse.
- Involuntary smoking has an adverse effect on the respiratory system. In adults, the strongest evidence for a causal relation exists for chronic respiratory symptoms.
- Full-term infants born to women who smoke weigh about 200 grams (g) less than those born to non-smokers. A smaller adverse effect is attributed to babies born to mothers who are exposed to secondhand smoke.

- In contrast to the findings for active smokers, women exposed to secondhand smoke do not appear to weigh less than women not exposed to secondhand smoke.

- No consistent association of maternal exposure to ETS with fertility or fecundity has been identified.

- There is no clear association of ETS exposure with age at menopause.

In addition, a more recent review[45] concludes that exposure to ETS in-utero has a greater effect on the lung function of young children than post-natal exposure. In-utero exposure to maternal smoking was independently associated with deficits in lung function that were larger for children with asthma. Boys and girls with a history of in-utero exposure to maternal smoking showed deficits in maximum midexpiratory flow (MMEF) and a decrease in FEV(1)/FVC ratio. Compared with children without asthma, boys with asthma had significantly larger deficits from in-utero exposure in FVC, MMEF, and FEV(1)/FVC, and girls with asthma had larger decreases in FEV(1)/FVC.

The health effects of ETS exposure from epidemiologic studies have led to many successful public health initiatives to protect non-smokers from ETS exposure. Smoking bans have been progressively introduced in indoor workplaces and enclosed public places where it was inferred that workers would be subject to the adverse health effects resulting from chronic ETS exposure. Hospitality industry premises were initially not covered under workplace legislation relating to ETS. Health effects of exposure to ETS in the hospitality industry have been the subjects of extensive research. A population based study demonstrated that mild asthmatics are at an increased risk of respiratory and sensory irritant sensory symptoms, and extra bronchodilator use, even at low ETS exposure levels of 0 – 0.05 $\mu g/m^3$ (nicotine)[55]. Another study in Helena[57], Montana suggests that smoke-free laws may have an effect on morbidity from heart disease. A higher exposure to ETS (measured by salivary cotinine concentrations) is related to a poorer respiratory and sensory symptom profile among non-smoking workers in hospitality premises[59,60,61,62,63,64,65,66,67,68,69,70,71,72]. The introduction of smoke-free policies in hospitality premises is accompanied by:

- a significant reduction in the number of hours spent exposed to ETS during work,

- a significant decline in the prevalence and number of different respiratory and sensory irritative symptoms reported,

- a significant decline in salivary cotinine concentrations and

- improvements in lung function among both smoking and non-smoking workers.

Smoking policy change in the hospitality industry has not been accompanied by the same decrease in exposure levels shown in workplaces outside the hospitality industry[73,74,76].

Overall, smoking restrictions reduce, but do not eliminate ETS exposure of workers in restaurant and hospitality industry premises. There is a 50% to 66% reduction in ETS marker concentrations when

the no-smoking area is located in a separate room. The reduction of ETS marker concentrations in the separate no-smoking room is greater than that in the no-smoking areas that are subsections of rooms where smoking otherwise occurs[80,81,82,83,84,85].

Ventilations systems reduce ETS marker concentrations, and mechanical ventilation systems are relatively more effective than extractor fans or natural ventilation[86,87]. However, residual mean and median air nicotine concentrations in both the smoking and non-smoking sections of hospitality premises, regardless of the type of ventilation system in place, may still present a significant risk to health.

With laws banning smoking in workplaces and indoor public places, outdoor gathering places are likely becoming relatively more important as sources of residual non-residential ETS exposure[55,81]. Pubs in Ireland that set up outdoor patios in response to a smoking ban in all workplaces, showed higher indoor nicotine concentrations compared with pubs that did not set up these outdoor patios[81]. Depending on the distance of these outdoor patios from building entrances and other openings, smoke may drift indoors.

Only two studies were identified that attempted to characterize outdoor ETS exposure levels[6,90]. Unlike indoor ETS marker concentrations that remain in the room long after a cigarette has been extinguished, secondhand smoke outdoors disperses more readily. Outdoor ETS concentrations are affected by source-receptor distance and wind conditions.

Repace (2005)[90] demonstrated that respirable suspended particulate (RSP) concentrations decline more slowly than cigarette smoke polycyclic aromatic hydrocarbons (PPAH) concentrations.

A study of 24 young healthy females[91] in an exposure chamber reported eye, nasal and throat irritation thresholds at ETS-PM2.5 concentrations of 4.4 $\mu g/m^3$ and odour thresholds at 1.1 $\mu g/m^3$. Extrapolating from these findings to his distance decay curves, Repace[90] estimates that ETS odour would be detectable as far as 7 meters (23 feet) from the source, and levels of irritation would begin at approximately 4 meters (13 feet) from the source.

1.0 Objectives

Scientific evidence links the effects of environmental tobacco smoke (ETS) exposure to adverse effects on health in non-smoking spouses of smokers or non-smokers exposed in the workplace. These adverse health outcomes include lung cancer, heart disease, respiratory disease and asthma among many others. Exposures to ETS in the home and in the workplace are generally of long duration and occur in indoor settings where ETS may be the primary contributor to indoor air pollution. Evidence of adverse health outcomes among non-smoking spouses of smokers and non-smokers exposed to ETS in the workplace have led to many successful public health initiatives to protect non-smokers from ETS exposure. Smoking restrictions have been progressively introduced in workplaces and enclosed public places such as restaurants, shopping malls, cinemas and theatres, and public transport and airlines.

Questions are now being raised about extending smoking bans to public places that are not covered by current regulations relating to ETS exposure. These may include indoor public places such as casinos and gaming venues as well as outdoor public places such as outdoor patios, school grounds, hospital grounds, institutional campuses and entrances and exits to public buildings. This has generated interest in the current availability of scientific evidence regarding the health consequences of exposure to ETS in settings other than in the home or workplace, i.e. in both indoor and outdoor public settings.

The purpose of this report is to:

1) review the studies of ETS exposures in workplace and home settings to evaluate the extent to which these findings can be generalized to other settings where ETS exposure occurs. Health endpoints include:

    a. all cancers,

    b. cardiac disease,

    c. stroke,

    d. asthma and other respiratory diseases and symptoms,

    e. other health conditions or endpoints identified in the published literature.

2) summarize the peer-reviewed scientific evidence in the epidemiologic, medical and environmental science literature on the health effects of environmental tobacco smoke exposure in indoor and outdoor public spaces. Public places can be defined as locations that are accessible to the public without much selection e.g. restaurants, bars, shops, offices, meeting places, schools, sport centers, as well as outdoor public places such as outdoor patios attached to restaurants and entrances and exits to office buildings.

3) review and summarize the exposure levels and associated levels of risk.

2.0 Environmental Tobacco Smoke

2.1 What is ETS

ETS is a term used synonymously with "second-hand smoke", "passive smoking" or "involuntary smoking". It refers to exposure not from your own smoking, but from being exposed to someone else's cigarette, cigar, or pipe smoke. ETS is composed of mainstream smoke (11%) exhaled by the smoker, sidestream smoke (85%) emitted from the burning end of the cigarette, and other contaminants that diffuse through the cigarette paper. These emissions contain both vapour phase and particulate contaminants, with sidestream smoke (SS) contributing nearly all of the vapor phase constituents and over half of the particulate matter[1]. One half to two thirds of smoke emanating from a burning cigarette are not inhaled by the smoker, but are released into the environment[2,3].

ETS is a dynamic complex mixture of thousands of compounds in particulate and vapour phases that cannot be measured as a whole. This complex mixture comprises more than 4,000 chemicals and more than 50 of these have been identified as known, probable or possible human cancer-causing agents (carcinogens)[1,4,5]. Table 1 presents a small list[6] of the identified gas phase components in ETS and ETS particulate matter with known health effects.

Accurate assessment of exposure to ETS is a challenge because of the dynamic complex composition of ETS and because exposure is not a direct consequence of actions of the "exposed" subjects[7]. Accurate measurement of exposure is important because it allows for more accurate estimation of the risk of disease that can be attributed to ETS, adds to further our understanding of the mechanisms and pathways of specific diseases, and assists in the decision-making process relating to public health interventions and health policy [8].

Table 1: Gas Phase Components in ETS and ETS Particulate Matter with Known Health Effects

Gas Phase Components in ETS with Known Health Effects			ETS Particulate Matter with Known Health effects		
Constituent	IARC Class[1/]	Non-Cancer Health Effects	Constituent	IARC Class[1/]	Non-Cancer Health Effects
1,3-Butadiene		irritant[2/], neurological effects	N-napthylamine	1	Irritant[2/], dizziness
Acetaldehyde	2B	irritant, dermatitis	2-Toluidine	2B	CNS[3/] depressant
Acetone		irritant, dizziness	4-Aminobiphenyl	1	Hematuria, lethargy
Acetonitrile		irritant, cause vomiting	Aniline	3	methemoglobinemia
Acrolein	3	Irritant, pulmonary edema	Arsenic (inorganic)	1	Hemolysis, neuropathy
Benzene	1	CNS[3/]depressant, nausea	Benz(a)anthracene	2A	
Carbon Monoxide		Headache, dizziness	Benzo(a)pyrene	2A	Dermatitis, irritant
Ethyl Benzene		Irritant, CNS depressant	Cadmium	2A	Bronchiolitis, irritant
Formaldehyde	2A	Irritant, induce asthma	Catechol	2B	methemoglobinemia
Hydrazine		Hepatotoxic, dermatitis	Chromium IV	2A	Renal toxicity, hemolysis
Methanol		Neurotoxicant, irritant	Dibenzo(a,i)pyrene	2B	
Methyl chloride		CNS depressant, fatigue	Dibenzo(a,l)pyrene	2B	
N-Nitrosodiethylamine	2A		Hydroquinone	3	CNS excitation, tinnitus
N-Nitrosodimethylamine	2A	Causes liver damage	Lead	2B/3[4/]	
N-Nitrosopyrrolidine	2B		N'-Nitrosonornicotine	2B	
Pyridine		Irritant, dizziness	Nickel	1	Immune alterations, irritant
Styrene	2B	CNS depressant, irritant	Nicotine[5/]		
Toluene		CNS depressant, irritant	N-Nitrosodiethanolamine	2B	
Source: California Air Resources Board (2005)			NNK[6/]	2B	
			Phenol	3	Cardiac arrthythmias
			Quinoline		Irritant, nausea, coma

1/ International Agency for Research on Cancer (IARC) Classification: 1-carcinogenic to humans; 2A-probably carcinogenic to humans with sufficient animal and inadequate or no human evidence; 2B-possible carcinogenic to humans with limited animal and no human evidence; 3-not classifiable as to its carcinogenicity to humans

2/ "Irritant" may be classified as an eye, respiratory, and/or skin irritant.

3/ CNS – central nervous system

4/ Inorganic lead – 2B; organolead – 3

5/ also found in gaseous form

6/ NNK: 4-(N-methyl-N-nitrosamino)-1-(3-pyridyl)-1-butanone

The main approaches used by researchers to determine this exposure include biomarkers, measurements of ETS components in air and exposure history. There are several biomarkers of environmental tobacco smoke exposure. The National Research Council proposed four criteria for a valid marker of ETS in the air as follows: the marker (a) should be unique or nearly unique to ETS, (b) should be easily detectable at low smoking rates, (c) should be emitted at similar rates for a variety of tobacco products, and (d) should have a fairly constant ratio to other ETS components of interest (e.g. respirable suspended particulates (RSP))[9]. Based on these criteria, researchers have concluded that cotinine (the primary metabolite of nicotine) in blood, saliva, or urine appears to be the most specific and most sensitive biomarker of exposure to ETS[10,11]. Cotinine assays are sensitive enough to reliably distinguish between non-ETS-exposed persons and ETS exposed non-smokers with low, moderate and high levels of ETS exposure[12]. It is the marker of choice in most epidemiologic studies, including the National Health and Nutrition Examination Survey (NHANES) carried out in the US between 1988-1994[13].

Cotinine in biologic fluids (measured in urine/saliva/serum) is not a valid marker of past or long- term ETS exposure because it has a half-life of approximately 17 hours. Cotinine measures in biologic fluids reflect several days of past exposure to ETS. It has been shown to vary among individuals depending on the rate at which nicotine is metabolized and cleared. Ethnicity, age and sex are factors shown to influence the rate at which different people convert and metabolize nicotine to cotinine, as well as the rate at which cotinine is cleared[12]. The difficulty associated with collecting and testing bodily fluids and cotinine's limitation as a measure of long-term ETS exposure, has led researchers to examine nicotine in hair as a marker of ETS exposure. Unlike cotinine in biologic fluid, hair as a biomarker is less susceptible to measurement error associated with the timing of sample collection[9]. It also integrates ETS exposure from all sources. However, reviews of hair nicotine as a biomarker suggest the need for larger studies that can demonstrate viability, and account for confounding factors specific to hair, such as irregular hair growth, hair colour and hair treatment using bleaches and dyes that affect nicotine binding[12].

ETS components that are monitored in air include nicotine, particulate matter (PM_{10}, $PM_{2.5}$) and other indicators or markers such as ultra-violet particulate matter (UVPM), fluorescent particulate matter (FPM), solanesol particulate matter (SolPM), carbon monoxide (CO) and 3-ethenylpyridine.

Interview questionnaires or self reported questionnaires are the most commonly used method of exposure assessment in epidemiological studies of health effects of ETS. Commonly, for non-smoking spouses of smokers the husband's smoking status is the exposure surrogate and exposure has been assessed in the home. Similarly, where exposure is assessed in the workplace the smoking status of co-workers is the exposure surrogate[8].

Accurate assessment of ETS exposure in epidemiologic studies is a challenge and is often raised in the discussion section of research papers to explain variation in risk estimates of an adverse health effect. Misclassification is an important consideration and is possible based on whether: (i) cases and controls are accurately classified as non-smokers, and (ii) epidemiologic studies can accurately classify ETS exposure[8].

The assessment of ETS exposure is especially an issue in case-control and cohort studies because there is not a valid and reliable long term biological marker of ETS exposure. These studies have therefore relied on interviews and questionnaires to assess ETS exposure status among cases and controls. All information relating to past exposure relies on estimates that may vary in their accuracy (recall bias). Self-report measures such as hours per day exposed are likely to be imprecise indicators of exposure because of variations in the number of cigarettes smoked, proximity of smokers to non-smokers, ventilation and other environmental characteristics as well as individual sensitivity to ETS. This issue of accurately measuring ETS exposure in epidemiologic studies has given rise to methodological studies in three areas: (i) validation studies comparing cotinine concentrations to current ETS exposure; (ii) test-re-test studies among cases and controls; and (iii) studies of the accuracy of spousal smoking histories[10]. Data on the extent of misclassification in epidemiological studies, based on comparing urine cotinine/creatinine values and questionnaire-reported smoking status among cases and controls, indicate that questionnaires provide accurate ETS exposure[15,16]. Spousal smoking histories by cases and controls tended to strongly agree with results of interviews with the spouses or next –of –kin[17,18,19]. Test –re-test studies also indicate that questionnaires are reliable in assessing ETS exposure[20,21,22]. However, the Canadian study by Pron et al[20] suggests that respondents more reliably reported residential exposure to spouse's passive smoke than the passive smoke of others at home or occupational ETS exposure. Additionally, there was low reliance of self-reported duration of ETS exposure (number and duration), which these authors argue is consistent with the inability of several studies to detect a dose-response relationship with lung cancer, especially where these studies relied solely on self-reported measures of duration.

2.2 Toxicity of Sidestream Smoke vs. Mainstream Smoke

The chemical composition of mainstream smoke has been more extensively characterized but many of the same chemical constituents are found in both mainstream and sidestream smoke. Both are produced by the same fundamental processes[1,7]. However, because of chemical and physical differences during their generation, there are important differences in the rates at which different constituents are emitted into the air. SS is generated in a lower temperature (around 600 °C) between the puffs, whereas MS is formed in a higher temperature (around 800–900 °C) during the puffs. The major components of sidestream smoke include tar particles, carbon monoxide, nicotine, nitrogen oxides and volatile hydrocarbons (ethane, propene, 1,3-butadiene and isoprene (2-methyl-1,3-butadiene)), aromatic hydrocarbons (polycyclic aromatic hydrocarbons) and several aromatic amines (including the carcinogens o-toluidine, 2-naphthylamine and 4-aminobiphenyl), chlorinated compounds, nitrosamines and radioactive isotopes[23].

Different cigarette brands using different ingredient blends, changes in cigarette design and manufacturing methods can influence the combustion efficiency of cigarettes and thus the mainstream and sidestream smoke delivery of different contaminants. The following polycyclic aromatic hydrocarbons (PAHs) were detected in 14 cigarette brands sold in Italy in 2001-2002: fluoranthaene, pyrene, benzo(a)anthracene, chrysene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene and dibenzo(a,h)anthracene[24]. Measured in a glass chamber, total PAH levels in sidestream smoke were approximately tenfold higher compared to

levels found in mainstream smoke. Other research on rates at which some of the other known compounds are released in sidestream compared to mainstream smoke also indicate that sidestream smoke may be more toxic per unit mass as compared to mainstream smoke[1,7,25].

Once emitted into the air, sidestream smoke released from the tip of the cigarette undergoes further physical and chemical changes through dilution, chemical reactions, deposition and ageing[13]. These processes may decrease the concentration of the airborne constituents of ETS, alter the size distribution of suspended particles, and chemically modify some of the more reactive constituents of ETS. The USEPA report (1992 pages 3-8 to 3-9) tabulates data on sidestream emission rates from filtered and commercial cigarettes for many compounds of public health interest. It showed that sidestream emissions are similar across different cigarette brands varying only by a factor of 2-3 when measured under standard smoking conditions. These differences are primarily related to the weight of the tobacco and paper burned during smoldering, with cigarette design having little effect[1].

Thus non-smokers who breathe other people's smoke inhale the same components as active smokers, though at much lower doses[26]. Differences in chemical composition and the concentration at which sidestream smoke is inhaled are used by different groups to either deny or to argue against the magnitude of estimated adverse health risk effects from ETS exposure. However, 21-day in-vivo inhalation toxicological studies comparing mainstream and sidestream smoke by Philip Morris at its laboratory in Germany during the 1980s demonstrated the following: inhaled fresh sidestream smoke is approximately four times more toxic per gram total particulate matter (TPM) than mainstream cigarette smoke, there was necrosis of the epithelial lining of the nasal cavity when the concentration of sidestream cigarette smoke was 23% that of mainstream smoke, atopy of the olfactory epithelium when the concentration of sidestream smoke was one tenth that of mainstream smoke, and squamous metaplasia of the nasal epithelium when the concentration was one-third that of mainstream smoke. The gas/vapour phase of sidestream smoke is responsible for most of the sensory irritation and respiratory tract epithelium damage, furthermore, damage increases with longer exposures. In addition to the 21-day in-vivo inhalation studies, Philip Morris also carried out eighty-day dermal exposure studies on mice. Results showed that sidestream condensate is approximately three times more toxic per gram and two to six times more tumourigenic per gram than mainstream condensate[27].

The Tobacco Testing and Disclosure Regulations in British Columbia require the major tobacco manufacturers to report on 44 toxic chemicals emitted in mainstream and sidestream smoke under two different smoking conditions defined by the International Organization for Standardization standards[28]. The same compounds are listed for both mainstream and sidestream smoke. The toxic compounds for which there are mandatory reporting requirements in BC are listed in Table 2.

Table 2: Mandatory Reporting : Tobacco & Testing Disclosure Regulations in BC

Mainstream Smoke

1. Ammonia
2. 1-aminonapthalene, 2-aminonapthalene, 3-aminobiphenyl and 4-aminobiphenyl
3. benzo[a]pyrene
4. formaldehyde, acetaldehyde, acetone, acrolein, propionaldehyde, crotonaldehyde, methyl ethyl ketone and butyraldehyde
5. hydrogen cyanide
6. mercury
7. nickel, lead, cadmium, chromium, arsenic and selenium
8. nitric oxide
9. N-nitrosonornicotine (NNN), 4-(N-nitrosomethylamino)-1-(3-pyridyl)-1-butanone (NNK), N-nitrosoanatabine (NAT) and N-nitrosoanabasine (NAB)
10. pyridine, quinoline and styrene
11. hydroquinone, resorcinol, catechol, phenol, m+p-cresol, and o-cresol
12. tar, nicotine and carbon monoxide
13. 1,3-butadiene, isoprene, acrylonitrile, benzene and toluene

Sidestream smoke:

1. Ammonia
2. 1-aminonapthalene, 2-aminonapthalene, 3-aminobiphenyl and 4-aminobiphenyl
3. benzo[a]pyrene
4. formaldehyde, acetaldehyde, acetone, acrolein, propionaldehyde, crotonaldehyde, methyl ethyl ketone and butyraldehyde
5. hydrogen cyanide
6. mercury
7. nickel, lead, cadmium, chromium, arsenic and selenium
8. nitric oxide
9. N-nitrosonornicotine (NNN), 4-(N-nitrosomethylamino)-1-(3-pyridyl)-1-butanone (NNK), N-nitrosoanatabine (NAT) and N-nitrosoanabasine (NAB)
10. pyridine and quinoline
11. hydroquinone, resorcinol, catechol, phenol, m+p-cresol and o-cresol
12. tar and nicotine
13. 1,3-butadiene, isoprene, acrylonitrile, benzene, toluene and styrene
14. Carbon monoxide.

Source: www.qp.gov.bc.ca/statreg/reg/T/TobaccoSales/282_98.htm

2.3 Summary

ETS is a dynamic complex mixture of more than 4,000 chemicals, and more than 50 of these have been identified as known, probable or possible human cancer-causing agents.

The main approaches used by researchers to determine exposure to ETS include biomarkers, measurements of ETS components in air and exposure history.

Research on rates at which some of the known compounds are released in sidestream compared to mainstream smoke indicate that sidestream smoke may be more toxic per unit mass as compared to mainstream smoke.

3.0 Health Effects of Exposure to Environmental Tobacco Smoke

3.1 Carcinogenicity

Several government scientific committees had declared environmental tobacco smoke a carcinogen in the 1980s and 1990s. It was the 1982 Report of the US Surgeon General[29] that first focused concern on the possibility that involuntary smoking may cause lung cancer. A large number of epidemiological studies followed the publication of that report. In their review of the evidence in these epidemiological studies the 1986 Report of the US Surgeon General[30], the National Research Council[31], the US Environmental Protection Agency[1], and the California Office of Environment Health Hazard Assessment[3] concluded that ETS exposure causes lung cancer. The National Toxicology Program (NTP) of the US Department of Health and Human Services in its 9th, 10th and 11th Report on Carcinogens5 (2000, 2002, and 2005) confirmed that secondhand smoke is a known human carcinogen.

In 2002, a scientific committee of twenty-nine experts from twelve countries convened by the International Agency for Research on Cancer (IARC) reviewed the accumulation of all significant published evidence relating to tobacco smoking and cancer, both active and involuntary[26]. It concluded that tobacco smoke is a carcinogen and causes cancer in many different human organs, a conclusion considered in an earlier IARC Monograph published in 1986[32]. In addition, this IARC scientific committee concluded that there is a statistically significant and consistent association between lung cancer risk in spouses of smokers and exposure to secondhand smoke from spouses who smoke[33].

IARC's conclusion was based on an evaluation of more than 50 studies which compared the lung cancer risk for non-smoking spouses of smokers with risks for non-smoking spouses of non-smokers. Meta-analysis of these studies showed that non-smokers have a greater risk of lung cancer if their spouses are smokers than if their spouses are non-smokers. The risk is approximately 25% greater for women (based on 46 studies) and 35% greater than expected for men (based on 11 studies). These excess risks remain even after controlling for potential sources of bias and confounding. Meta-analysis of 19 studies of non-smoking women exposed to secondhand smoke in the workplace showed that risk of lung cancer was approximately 20% greater than expected. These results are consistent with five other meta-analyses published 1997- 2002, where the pooled relative risks varied between 19% and 29% for the non smoking women exposed to secondhand smoke compared to their unexposed counterparts[34, 35,36,37].

Lung tumours of non-smokers exposed to ETS contain TP53 and KRAS mutations that are similar to those found in tumours from smokers[26]. The urine of non-smokers exposed to ETS contains metabolites of the tobacco-specific carcinogen, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone. The excess risk of lung cancer in non-smoking women exposed to ETS is 1-2% of that in smokers. Levels of these metabolites in ETS exposed women were ~ 5.6 % that of their smoking partners, indicating

consistency between dose and excess risk for lung cancer[38,39]. IARC concluded that exposure to secondhand smoke is a cause of lung cancer in never-smokers.

The 2004 review by IARC[26] also concluded that the evidence linking ETS exposure to breast cancer is inconsistent. Four of ten case-control studies showed statistically significant increases in risk, but prospective studies, particularly two large cohort studies in the US, did not provide evidence of a causal link between exposure to ETS and breast cancer. The data did not show a positive dose-response and there was a lack of association between breast cancer and active smoking, indicating that it is unlikely there is an association between breast cancer and passive smoking.

IARC states that the evidence linking childhood cancer and ETS exposure from parental smoking is inconsistent. The data suggests a modest association between exposure to maternal smoking during pregnancy and childhood cancer for all sites combined, but the data failed to show association for individual cancer sites. The review concludes that more studies are needed to rule out bias and confounding.

Data relating to the following cancers among adults were considered sparse, inconsistent and bias could not be ruled out: cancers of the naso-pharynx, nasal cavity, paranasal sinuses, cervix, gastrointestinal tract and cancers of all sites combined[40]. Further research is needed in all those areas where the data have been inconsistent, sparse and bias could not be ruled out.

Important conclusions reached by IARC (2004) for non-cancer health effects of ETS exposure included the following:

- Meta-analysis shows that exposure to ETS increases the risk of a coronary heart disease event by 25 -30% for non smokers living with a smoking spouse compared to non-smokers living with a non-smoking spouse.
- Involuntary smoking has an adverse effect on the respiratory system. In adults, the strongest evidence for a causal relation exists for chronic respiratory symptoms.
- Full-term infants born to women who smoke weigh about 200 g less than those born to non-smokers. A smaller adverse effect is attributed to babies born to mothers who are exposed to secondhand smoke.
- Data on the hormonal and metabolic effects of involuntary smoking are sparse. In contrast to the findings for active smokers, women exposed to secondhand smoke do not appear to weigh less than women not exposed to secondhand smoke.
- No consistent association of maternal exposure to ETS with fertility or fecundity has been identified.
- There is no clear association of ETS exposure with age at menopause.

3.2 Previous Scientific Reviews

The conclusions by six government and national scientific reviews[1,3,41,42,43,44] completed independently in the United States[1,3,44], Australia[42], United Kingdom[43], and internationally[41] show a remarkable degree of consensus where they focused on the same disease outcomes. Each of these reviews focused on a different number and types of health endpoints that are listed in Appendix Table A[a].

The review by the United States Environmental Protection Agency (USEPA)[1] published in 1992 focused on respiratory disorders. It was based on the total weight of scientific evidence available up to the time of its publication. The California Environmental Protection Agency (CalEPA)[3] published its review in 1997. It reviewed the total weight of scientific evidence for the following health effects: developmental effects, respiratory effects, carcinogenic effects and cardiovascular effects. In addition input was solicited from the tobacco industry and several public consultation workshops were held. Prior to publication the report was peer reviewed by the California Scientific Review Panel – a body created under California law to provide an independent peer review of the scientific aspects of its toxic air contaminants and air pollution program. The National Toxicology Program in its Ninth Report on Carcinogens[44] added environmental tobacco smoke to its list of 41 known human carcinogens. It based this conclusion on the reviews of evidence completed by USEPA[1] and CalEPA[3] indicating a causal relationship between exposure to ETS and human lung cancer.

The Australian report[42] examined the public health impact of ETS exposure in the home. Disease outcomes examined included respiratory illness and asthma among children, lung cancer and coronary heart disease among adults. The UK Report[43] examined the health effects of active smoking and exposure to second hand smoke. Its major focus on was the health effects of exposure of non-smokers to ETS, in particular the evaluation of possible increased risks of lung cancer, ischaemic heart disease, and respiratory and other diseases in childhood. The WHO Report[41] brought together experts from developed and developing countries to examine the effects of ETS on child health and to recommend interventions to reduce children's exposure. Health endpoints considered included: lower respiratory tract infections such as pneumonia and bronchitis, coughing and wheezing, worsening of asthma, and middle ear disease (otitis media), sudden infant death syndrome (SIDS), reduced birth weight, decreased lung function, cardiovascular disease in adulthood and neurobehavioural impairment.

a Table sourced from Ontario Tobacco Research Unit (2001). Protection from second-hand tobacco smoke in Ontario. A report of the Ontario Tobacco Research Unity, University of Toronto.

Three of these reviews concluded that exposure to ETS is causally[b] related to the following health outcomes[c]:

Developmental effects

- fetal growth (low birth-weight or small for gestational age)
- sudden infant death syndrome (SIDS)

Respiratory effects in children

- acute lower respiratory tract infections in children (e.g. bronchitis and pneumonia)
- asthma exacerbation in children
- chronic respiratory symptoms in children
- middle ear infection in children (otitis media)

Carcinogenic effects

- lung cancer
- nasal sinus cancer
- cervical cancer

Cardiovascular effects

- coronary heart disease.

b To evaluate whether a positive association denotes a causal relationship epidemiologists evaluate the evidence using standard criteria. Five criteria commonly used to make inferences about causality include: (i) consistency of the association between the exposure and the health endpoint (similar association is repeated in different populations), (ii) strength of the association (the magnitude of the risk estimate in the exposed compared to the unexposed population is positive and chance, bias and confounding can be ruled out with reasonable confidence), (iii) dose-response (does the health effect under study increase in a predictable way as exposure (concentration x duration) increases – how much, how often and for how long), (iv) temporality (whether the health effect appears after the exposure) and (v) biological plausibility (is the association plausible given the basic science and pathways of the disease).

c Also summarized in Ontario Tobacco Research Unit (2001). Protection from second-hand tobacco smoke in Ontario. A report of the Ontario Tobacco Research Unity, University of Toronto.
Mah S & Keefe A (2001) Proposed amendments to the environmental tobacco smoke (ETS) sections of the occupational health and safety regulation: summary of review of the health effects literature. Worksafe BC.

There are a number of health effects for which evidence is suggestive of a causal association. These include:

Reproductive and Developmental Effects

- spontaneous abortion
- adverse impact on cognition and behaviour
- decreased pulmonary function in children

Respiratory Effects

- Exacerbation of cystic fibrosis
- Asthma induction in children

Carcinogenic Effects

- Cervical cancer

Because of their higher ventilation rates, the lungs of young children are more heavily exposed to ETS compared to adults. The lungs of growing young children may be more sensitive to ETS. A recent review[45] provided additional support for the conclusions reached by the six independent review committees:

- Exposure to ETS leads to reduced lung function in children. Three different windows of exposure may be biologically relevant: (i) in utero, (ii) the first 2 years of life when maternal smoking has the greatest impact, and (iii) subsequent years. The evidence suggests that exposure to ETS in-utero has a greater effect on lung function than postnatal exposure. Additionally, in-utero exposure to maternal smoking was independently associated with deficits in lung function that were larger for children with asthma. Boys and girls with a history of in-utero exposure to maternal smoking showed deficits in maximum midexpiratory flow (MMEF) and a decrease in FEV(1)/FVC ratio. Compared with children without asthma, boys with asthma had significantly larger deficits from in-utero exposure in FVC, MMEF, and FEV(1)/FVC, and girls with asthma had larger decreases in FEV(1)/FVC[46,47].

- Lower respiratory tract illnesses in early childhood (such as croup, bronchitis, bronchiolitis and pneumonia) are causally related to ETS exposure from parental smoking.

- Exposure to ETS from maternal smoking leads to acute exacerbation of asthma.

- The prevalence of asthma, wheeze and chronic cough is causally related to ETS exposure from parental smoking. The incidence of asthma and wheezing illness in children is increased if there is smoking in the household up to age 6 years, but the influence of smoking is less strong thereafter.

- ETS exposure is also associated with increased risk for sudden infant death syndrome (SIDS), and otitis media

3.3 Recent Research

New research on ETS exposure has continued to be published. They re-confirm the conclusions on the health effects of ETS exposure reached above. Although the association between ETS and the list of health endpoints mentioned above is not disputed, the magnitude of its impact still is, as demonstrated in the discussion that follows.

Lung cancer: Vineis et al[39] lists the relative risks[d] of 38 studies that examined the lung cancer risks of non smoking women with exposure to ETS from a spouse who smoked. Twenty of these 38 studies showed relative risk of lung cancer ranging from 1.2 to 3.4 among women who did not smoke but who had the highest exposure to secondhand smoke from a spouse who smoked (# of cigarettes smoked per day by smoking spouse ≥ 16 in 19 studies and > 10 in 17 study) compared with women who did not smoke and who had spouses who did not smoke. Similarly, eighteen studies with exposure assessment based on the number of years of marriage to a smoker showed relative risk of lung cancer ranging from 0.9 to 3.3 among women who did not smoke, but who had the highest exposure to secondhand smoke from a spouse who smoked (based on number of years married) compared with women who did not smoke and who had spouses who did not smoke.

Coronary Heart Disease: While there is general agreement on a link between exposure to involuntary smoking and coronary heart disease, there is continued debate over the magnitude of the risk. Meta-analysis of case control and cohort studies have shown that a non-smoker living with a smoker has a 25% -30% (range: 15% - 30%) increase in the risk of coronary heat disease compared with non-smokers living with a non-smoking spouse[48,49,50]. A recent study by Whincup et al[51] concludes that previous studies, defining exposure in terms of living with a smoking spouse, underestimate the risk of exposure to passive smoke. Surrogate measures of ETS exposure, using indicators such as number of years living with a smoking spouse, introduce the potential of misclassification which attenuates risk estimates. This 20-year prospective study measured exposure to secondhand smoke based on serum cotinine levels, a biomarker which is a specific nicotine by-product that measures ETS exposure from all sources. These researchers discovered that higher concentrations of serum cotinine among non-smokers are associated with an excess risk of coronary heart disease of about 50-60%.

Ongoing research: New research is also being published pointing to health effects that were previously not recognized as resulting from ETS exposure, as well as health effects where the relationship to ETS exposure have been inconsistent. These include the risk of stroke or cerebrovascular disease, which may share a similar pathophysiological mechanism as coronary heart disease shown to be causally related to ETS exposure. Case-control studies have shown increased risks of stroke among non-smokers living with a smoker compared to non-smokers living with a non-smoking partner[52, 53]. The results from prospective cohort studies are less consistent with the

d Rate ratios for cohort studies and odds ratios for case control studies

Whincup et al[51] study suggesting no association, while a recent study by Iribarren et al[54] found increased risks of first ischemic stroke among men and women. They found that ETS exposure at home of 20 hours or more a week compared to less than 1 hour per week was associated with a 1.29 fold (95% CI=0.75- 2.20) and a 1.50 fold (95% CI=1.07 – 2.09) increased risk of first ischemic stroke among men and women respectively. However, this same study found no significant association between ETS exposure outside the home and ischemic stroke, or between ETS exposure at home or outside the home and the risk of transient ischemic attack.

3.4 Summary

The International Agency for Research on Cancer (IARC) (2004) concluded that there is sufficient evidence to categorize secondhand smoke as carcinogenic to the human lung. It also concluded that the evidence linking ETS exposure to breast cancer is inconsistent. Data relating to the following cancers among adults were also considered sparse, inconsistent and bias could not be ruled out: cancers of the naso-pharynx, nasal cavity, paranasal sinuses, cervix, gastrointestinal tract, and cancers of all sites combined. For childhood cancers the data suggest a modest association between exposure to maternal smoking during pregnancy and childhood cancer for all sites combined, but the data failed to show association for individual cancer sites.

The non-cancer health effects of ETS exposure from IARC's review are as follows:

- **ETS exposure increases the risk of a coronary heart disease event by 25 -30% for non smokers living a smoking spouse compared to non-smokers living with a non-smoking spouse.**
- **Involuntary smoking has an adverse effect on the respiratory system. The evidence suggests that exposure to ETS in-utero has a greater effect on the lung function of young children than post-natal exposure. In adults, the strongest evidence for a causal relation exists for chronic respiratory symptoms.**
- **Full-term infants born to women who smoke weigh about 200 g less than those born to non-smokers. A smaller adverse effect is attributed to babies born to mothers who are exposed to secondhand smoke.**
- **In contrast to the findings for active smokers, women exposed to secondhand smoke do not appear to weigh less than women not exposed to secondhand smoke.**
- **No consistent association of maternal exposure to ETS with fertility, fecundity or age at menopause has been identified.**

Six independent government scientific panels in the US, UK, Australia and internationally, concluded that exposure to ETS is a cause, or a possible cause, of fifteen health outcomes. These include four developmental diseases or conditions, seven respiratory diseases or conditions, three cancers and coronary heart disease.

4.0 ETS in Indoor Public Places and Health Effects

As the evidence of the harm of passive smoking accumulates, smoking restrictions have been progressively introduced in enclosed public spaces in the industrialized world. In Canada, and more specifically in British Columbia, there are smoking restrictions in cinemas, theatres, elevators, shopping malls, public transport and airlines, and 85% of our indoor workplaces. Workplaces, more specifically enclosed indoor spaces, are becoming smoke free by virtue of legislation or explicit management policy.[e]

Pubs, bars, restaurants and gaming venues are often not covered by workplace legislation relating to ETS exposure. They are instead classified under public places, defined as locations that are accessible to the public. ETS exposure levels and potential health effects of secondhand smoke among workers and patrons in these premises have been the subjects of extensive research.

4.1 ETS and Health Effects on Workers in Indoor Public Places

The impetus for adopting increased smoking restrictions has been largely aimed at protecting non-smokers from the health risks associated with secondhand smoke exposure. Eisner et al [55] argue that because of increasing morbidity and mortality from adult asthma, identifying modifiable environmental factors that exacerbate asthma is a priority. To measure ETS exposure in different microenvironments (indoors and outdoors) these authors recruited fifty subjects showing a positive screening for ETS exposure from an ongoing longitudinal cohort study in northern California. Following an initial screening interview these participants donned a personal nicotine badge monitor during regular activities for 7 days. This was then followed by a structured telephone interview that included questions relating to demographic characteristics, asthma history, asthma-specific severity, quality of life, environmental exposures, health status and health care utilization for asthma. In this way self-reported ETS exposure via interviews and questionnaire was validated with levels measured by the nicotine badge monitor. The data correlated moderately and ETS exposure in six micro-environments was monitored for 7 days: the respondent's home, another person's home, traveling in a car or another vehicle, workplace, bars and nightclubs and other locations. The results showed that nicotine concentrations were highest among those participants who reported home exposures (median 0.61 $\mu g/m^3$) versus exposure at work (0.03 $\mu g/m^3$), other (outdoor) exposure (0.025 $\mu g/m^3$), and no exposure (0 $\mu g/m^3$). It was noted that all subjects with workplace ETS exposure (38%) indicated recent exposure in an outdoor smoking area at work. California state law banning smoking

e WCB regulations, in effect on 1 May 2002 requires employers in the hospitality industry (restaurants, bars, pubs, lounges, nightclubs, bingo halls, bowling alleys, and gambling casinos) to control workers' exposure to ETS either by prohibiting smoking in the workplace or restricting smoking to separately structured and ventilated designated rooms. These regulations do not override municipal bylaws that may prohibit smoking even in designated smoking rooms (source: www.tobaccofacts.org/secondhand/working.html)

in nearly all workplaces since 1 January 1995 may have shifted ETS exposure from indoor to outdoor locations.

Participants were also assigned to three exposure levels to examine whether there was a relationship between these exposure levels and sensory and respiratory symptoms: no exposure, 0 – 0.05 µg/m^3 and > 0.05 µg/m^3 nicotine. Compared with the group with no ETS exposure, both exposed groups (0 – 0.05 µg/m^3 and > 0.05 µg/m^3 nicotine) were associated with increased risks of sensory symptoms including eye, nose and throat irritation (OR= 2.2, 95% CI: 0.3-15 and OR=5.9, 95% CI: 0.95-37). Similarly both exposed groups were associated with increased risks of respiratory symptoms (OR=1.9; 95% CI CI: 0.4-8.8 and OR=6.8; 95% CI CI: 1.4-32.3 respectively) and extra bronchodilator use.

10 adult subjects with mild asthma were exposed to carbon monoxide as a marker of ETS, (22.4 ± 1.2 ppm carbon monoxide) or ambient air (sham) for three hours (7.00 pm to 10.00 pm) in a 24 m^3 exposure chamber ventilated to ambient air. The intent was to examine the effect of ETS exposure in the evening on inflammatory changes to bronchoalveolar (BAL) and nasal lavage (NAL) fluid. The rationale for the approach was that: (i) patients with bronchial asthma are expected to be more susceptible to short term effects of ETS due to their pre-existing hyperresponsiveness, and (ii) potential effects of ETS are enhanced if they are introduced in the early evening when airway inflammation is known to be more pronounced in subjects with asthma. Each subject participated in this experiment on two different occasions at least 7 days apart[56]. Participants assessed the severity of respiratory and sensory symptoms with a questionnaire prior to entering the exposure chamber and immediately prior to the end of exposure. Spirometric measurements and bronchoscopy were also performed according to protocol. The results showed that a single ETS exposure in subjects with mild asthma caused a significant rise in symptoms of the eyes, nose, mouth, throat and chest compared with the sham. However, there was no accompanying detected change in lung function or airway inflammatory response.

A population based study in Helena, Montana, USA examined whether enactment of legislation banning smoking in all workplaces and public places (indoor as well as outdoor) was associated with a decline in hospital admissions for acute myocardial infarction (MI)[57]. This ban was in effect 5 June, 2002 to 3 December, 2002 when opponents won a court order suspending enforcement. The number of hospital admissions for MI fell significantly from an average of 40 admissions during the same months in the years before and after the law was in effect, to a total of 24 MI admissions during the six months that smoking was banned from all indoor workplaces and outdoor public places (-16; 95% CI = -31.7 to -0.3). There was a non-significant increase of 5.6 (95% CI = -5.2 to 16.4) in the number of MI admissions in jurisdictions outside Helena during the same period (12.4 in the years before and after the law, to 18 while the law was in effect). These authors point out that they were able to detect the effect of the smoke free policy because Helena is an isolated place with a single hospital that dealt with all admissions for MI. In most jurisdictions the effects of smoke free policies would be difficult to detect because there are several hospitals and the resident population tends to move across boundaries for work, housing and health care. Weaknesses in their study include: the absence of data on actual exposures to secondhand smoke; the small sample size; and

the unexpectedly large effect, lack of smoking prevalence data and other methodological deficits. However, a commentary by the editor of the British Medical Journal points out that " the main point ... is to draw readers' attention to the increasing evidence that small exposures to tobacco can cause large increases in the risk of cardiovascular disease"[58].

Research from different jurisdictions show that the introduction of smoke free policies in restaurants and bars reduce cotinine concentrations measured in biologic fluid, reduce exposure to secondhand smoke, and lead to improved respiratory and sensory symptoms among workers in these premises. A smoking ban covering all indoor workplaces, including bars and restaurants, was introduced in the Republic of Ireland in March 2004, but not in neighbouring Northern Ireland. To assess the health effects of this ban Allwright et al[59] followed 158 non-smokers working in three pubs in the Republic and 1 (control) in Northern Ireland six months prior to the ban and 12 months later. These workers were assessed at baseline and at 12 month follow up for the following: salivary cotinine concentration, self reported ETS exposure and respiratory and sensory irritation symptoms. In the Republic of Ireland salivary cotinine concentrations dropped by 80% after the smoking ban (from median 29.0 nmol/l (95%CI=18.2-43.2) to 5.1 nmol/l (95% CI=2.8-13.1) in contrast with a 20% decline in Northern Ireland over the same period, from a median of 25.3 nmol/l (95% CI=10.4-59.2) to 20.4 nmol/l (95% CI=13.2-33.8). The unexpected improvement in Northern Ireland was attributed to an economic downturn in the region and stricter enforcement of regulations on underage drinking during that period. After adjusting for confounders, the decline in cotinine concentration was twice as great in the Republic compared with Northern Ireland. At follow-up self reported work related secondhand smoke exposure also dropped significantly in the Republic of Ireland (median of 40 vs. 0 hours in the past week; $p<0.001$) compared with Northern Ireland (median of 42 vs. 40 hours; $p=0.02$). ETS exposure outside of work also declined in the Republic (median 4 vs. 0 hours; $p<0.001$) but increased in Northern Ireland (0 vs. 2.5 hours; $p=0.41$). Reporting of any respiratory symptoms declined significantly in the Republic (16% decline, 95% CI = -26.1 to – 7.3) but not in Northern Ireland (0% difference, 95% CI= -32.7 to 32.7).

A pre- and post-smoking ban longitudinal follow-up of hospitality workers in New York showed similar results[60]. The analysis was limited to those subjects who completed both an interview and provided a saliva specimen at baseline (pre-smoking ban) and at three, six and twelve month follow-up (post-smoking ban). Hours of exposure to secondhand smoke at work declined from 12.1 hours to 0.2 hours. Comparing baseline to the 12 month follow-up results showed the following: saliva cotinine concentrations decreased from 3.6 ng/ml (95% CI: 2.6 – 4.7 ng/ml) to 0.8 ng/ml (95% CI: 0.4 – 1.2 ng/ml), the prevalence of workers reporting sensory symptoms declined from 88% at baseline to 38%, and the total number of sensory symptoms declined by 69% from 1.6 to 0.5. Before the law went into effect, approximately 46% of respondents experienced any one of the following symptoms: wheezing/whistling in chest, shortness of breath, coughing in the morning, coughing during the day or at night, or bringing up any phlegm. At 12 month follow-up, this dropped to 29% but the decline was not statistically significant.

Bates et al[61] compared salivary cotinine concentrations among non-smoking workers in hospitality premises with government employees: hospitality premises that permitted smoking by customers

(either in designated areas or no smoking restrictions), hospitality premises that were smoke free (did not permit patrons to smoke) and government employees in smoke free workplaces (control). Participants also met the following eligibility criteria: they did not smoke in the previous six months or engage in nicotine replacement therapy, and on the day of participation they needed to be working at least four hours as salivary cotinine concentrations have been shown to plateau about four hours into a period of constant exposure[62]. Participants met with an interviewer before and after their work shift during which they answered a questionnaire and provided a saliva sample. Given the similarities in the data, the results for non-smoking workers in smoke free hospitality premises were combined with that for government workers in smoke free workplaces. Post-shift, non-smoking workers in smoke free work environments showed the smallest pre- and post-shift overall mean cotinine increase (0.2 ng/ml) compared with non-smoking workers in smoking only in designated areas (0.7 ng/ml) and non-smoking workers in hospitality premises with no smoking restrictions (1.8 ng/ml). These results indicate that working in hospitality premises where any smoking is permitted is associated with higher exposure to ETS over the work shift ($p = 0.002$ when pre- and post-shift mean differences in cotinine for all workers in smoke free places were compared with that for all workers in premises where smoking was permitted). This is consistent with results from other studies, which have shown that designated smoking areas only provide partial protection from exposure to ETS [63, 64, 65]. Prevalence ratios in the Bates et al study also showed a tendency for workers in premises permitting customer smoking to report a higher prevalence of respiratory and irritation symptoms compared to workers in smoke free workplaces. Physician diagnosed asthma and use of asthma medication were less common among workers in premises that permitted patron smoking, likely demonstrating the "healthy worker effect". To reduce their chances of an asthmatic attack persons sensitized to environmental tobacco smoke are likely to avoid working in smoky workplaces.

An Australian study compared pre- and post-shift saliva cotinine levels among non-smoking employees from three workplaces with varying smoking policies (non-smoking workers from clubs and casinos with either partial or no smoking restrictions and office workers from smoke free workplaces)[66]. Mean before and after-shift cotinine concentrations per hour worked was significantly higher among club (0.42 ng/ml/hr worked) and casino (0.18 ng/ml/hr worked) employees compared to office workers (0.03 ng/ml/hr worked) after adjusting for age, gender, educational attainment, secondhand smoke exposure at home and other sources, and shift length. Casino and club workers (either partial or no restrictions on smoking) reported similar levels of respiratory morbidity and were more likely to have sore eyes (OR=5.5; 95% CI=1.4-21.4) and a sore throat (OR=4.3; 95% CI= 1.1-17.4) compared with office employees. Despite claims that segregation of workers and separate ventilation systems protect workers from ETS exposure, this study showed that the prevalence of respiratory and irritative symptoms among casino workers were similar to club workers. This study, together with those reviewed above indicate that higher exposures to ETS (measured by salivary cotinine levels), is related to a poorer respiratory and sensory symptom profile.

In one US study[67] 53 bartenders from 25 freestanding bars and taverns were interviewed before and 1 month after a 1998 California smoking ban was extended to all bars and taverns. Forty (76%) of 53 bartenders who were ever smokers reported no change in smoking habits subsequent to the smoking ban. All 53 bartenders reported a decline in workplace ETS exposure after the smoke ban went into

effect, from a median of 28 to 2 hours per week. Thirty-nine of the 53 (74%) bartenders reported respiratory symptoms before the ban, twenty-three of the 39 (59%) no longer had symptoms at follow up. Forty-one of 53 bartenders (77%) reported sensory irritation before the ban, after the ban 32 of these 41 bartenders (78%) had symptom resolution. In this same study pulmonary function tests showed a 5% to 7% improvement for both smokers and non-smokers after working for 1 month in a smoke free environment.

A cross-sectional study of 382 non-smoking indoor workers in Victoria, Australia[68] found that 56% of hospitality workers reported ETS exposure during a typical day at work, compared with only 11% of workers in community, property and manufacturing industries. After controlling for confounders, exposure to ETS at work for part of the day compared to no exposure was significantly associated with an increased risk of wheeze (OR=4.26; 95% CI=1.78-10.21), frequent cough (OR=2.26; 95% CI=1.03-4.94), sore eyes (OR=3.77; 95% CI=2.03-7.01) and sore throat (OR=2.70; 95% CI=1.40-5.22). There was a dose-response relationship between increasing levels of ETS exposure at work and morning cough, frequent cough, sore eyes and sore throat, and a positive relationship for wheeze.

A cross-sectional study of hospitality workers in British Columbia[69], Canada, showed that the prevalence of irritant and sensory symptoms was higher among workers from premises that permitted smoking compared to workers from workplaces where smoking was prohibited. Those working where smoking was permitted had over five times the risk of chronic phlegm, over two times the risk for wheeze, chronic cough and burning or itchy eyes. The adjusted odds ratio for adult-onset asthma was elevated but not statistically significant (OR=3.3, 95% CI= 0.97-11.4). Hair nicotine levels were also lowest for the workers from workplaces where smoking was prohibited. Tests showed no significant difference in pulmonary function between the groups. An earlier BC study showed that nonsmoking workers in bars with no smoking restrictions showed the highest hair nicotine levels, similar or close to levels found in smokers[70].

The health significance of some of the acute effects of short-term ETS exposure is not clear. A recent study[71] examined the effects of an acute 6-hour exposure to ETS on lipid and lipoprotein levels. Twelve subjects were exposed to ETS for 6 hours in a smoking chamber with nicotine and carbon monoxide concentrations of 16.0 $\mu g/m^3$ and 12.0 ppm respectively, mimicking levels measured in local bars. Pre and post ETS exposure results showed that HDL-C and HDL_2-C levels were significantly reduced by 18% and 37% respectively while total cholesterol (TC) levels remained unchanged. The decreases in TC/HDL-C and HDL_2-C/HDL_3-C were sustained for at least 24 hours. Another study using before and after comparisons showed that saliva cotinine concentrations following 3 hours exposure in a pub were consistently higher than baseline among 11 non-smoking medical students [72]. The mean difference was 1.03 ng/ml (95% CI= 0.76 – 1.30); and adjustments to post-visit levels for metabolism and clearance of cotinine, assuming a first-order elimination half-life of 18 hours, made very little difference to these results. The smokiest pubs generated the greatest increase in cotinine. Males showed higher baseline cotinine levels than females and smaller increases post-exposure.

4.3 Summary

With bans of smoking in indoor workplaces and indoor public places, outdoor locations are likely relatively more important as a source of non-residential ETS exposure.

Mild asthmatics demonstrate an increased risk of respiratory and sensory irritant sensory symptoms, and extra bronchodilator use even at low ETS exposure levels of 0 – 0.05 µg/m^3 (nicotine).

Smoke-free laws may have an effect on morbidity from heart disease.

Higher exposures to ETS (measured by salivary cotinine concentrations) are related to a poorer respiratory and sensory symptom profile.

The introduction of smoke free policies in indoor hospitality workplaces is accompanied by:

- **a significant reduction in the number of hours exposed to ETS during work,**
- **a significant reduction in salivary cotinine concentrations,**
- **a significant reduction in the prevalence of respiratory and sensory irritative symptoms**
- **a significant reduction in the number of respiratory and sensory irritative symptoms reported**
- **improvements in lung function among both smoking and non-smoking workers**

ETS marker (nicotine, cadmium, PM_{10}, $PM_{2.5}$) concentrations, salivary cotinine concentrations, the number of hours exposed to ETS at work, the prevalence of respiratory and sensory irritant symptoms and the number of different symptoms reported were lowest in hospitality premises that banned smoking, increasing in premises that restricted smoking to designated spaces, and were highest in unrestricted smoking establishments.

5.0 ETS Concentrations in Indoor Public Places and Risk Assessment

Many studies examine the concentration of nicotine, particulate matter (PM_{10}, $PM_{2.5}$) and other indicators or markers (ultra-violet particulate matter, fluorescent particulate matter, solanesol particulate matter, carbon monoxide and 3-ethenylpyridine) of ETS in public places such as restaurants, bars, cafeteria, transportation facilities such as airplanes and private vehicles. For a given environment, the harm from passive smoking depends on the time spent in that environment and the concentration of ETS in that air space. The concentration of ETS, in turn, is affected by the size of the space, the number of people smoking there, and the ventilation rate.

Siegel and Skeer[73] completed a meta-analysis that examined the mean nicotine concentrations in the "5 B's": bars (limited to 13 US studies), bowling alleys, billiard halls, betting establishments and bingo parlours. They used a method that enabled them to compare the results of this meta-analysis to an earlier study[74] that examined ETS exposure in restaurants compared to office workplaces and homes with at least one smoker. Controlling for personal smoking habits and other confounders, Seigel[74] in this earlier study concluded that exposure levels in restaurants meant that there was likely a 50% (10-90% range) increase in lung cancer risk among restaurant workers compared to the general population. A review of California's mortality data for 1971-1981[75] found that female waitresses had high risks for lung cancer which was the third leading cause of premature death for women both in and out of the labour force (standardized mortality rate 368).

Table 3 shows that the mean nicotine concentrations in the 5 B's were 2.4 to 18.5 times higher than in offices or residences and 1.5 to 11.7 times higher than in restaurants. The highest concentrations were found in bars and bingo parlours. The higher mean nicotine concentrations found in bars in the US are consistent with, but not as high as some of the nicotine concentrations found in pubs and discos in seven European cities (median nicotine levels for Vienna: 122; Paris: 59; Athens (range): <5 – 200; Florence: 19; Barcelona: 19 and Orebro: < 5 $\mu g/m^3$)[76].

Siegel and Skeer[73] acknowledge the limitation posed by the small number of studies that looked at nicotine exposures in betting establishments, bowling alleys, billiard halls and bingo parlours. Even accounting for this limitation, these authors concluded that at these exposure levels, estimated working lifetime excess lung cancer mortality risk to workers in the 5 Bs is between 1.0 – 4.1/1000. If correct, these numbers indicate that we can expect 1.0 to 4.1 of every 1000 workers in these establishments (assuming they work for 40 years) will die of lung cancer that is attributable to their workplace ETS exposure. The lower limits of these risk estimates were approximately three or four times higher than the de manifestis risk level, discussed in the following section, at which the regulatory agencies usually take action. As the number of heart disease deaths far exceeds the number of lung cancer deaths, Siegel and Skeer go on to say that estimating lung cancer mortality risk in fact underestimates the burden of disease by a factor of 10.

Table 3: Indoor Air Concentrations of Nicotine (µg/m³) in a Variety of Public Places

Type of workplace	Number of studies	Number of establishments sampled	Weighted mean*	Range	Ratio**
Offices	22	940	4.1	0.8 – 22.1	1.0
Residences	7	91	4.3	1.6 – 21.0	1.0
Restaurants	17	402	6.5	3.4 – 34.0	1.6
Betting establishments	3	4	9.8	8.0 – 10.7	2.4
Bowling alleys	2	6	10.5	10.1 – 10.7	2.6
Billiard halls	2	3	13.0	9.8 – 19.4	3.2
Bars	10	27	31.0	7.4 – 105.4	7.6
Bingo Parlours	2	3	76.0	65.5 – 81.2	18.5

* Mean of average nicotine values reported in individual studies weighted by number of establishments sampled in each study

** Ratio of weighted mean nicotine concentration in each workplace type to weighted mean nicotine concentration in offices

Source: Siegel M & Skeer M (2003)

Siegel and Skeer adopted lifetime excess cancer mortality risk estimates from the model previously developed by Repace and Lowrey[77]. This model assumes a 40 year working lifetime and exposure data is based on the weighted mean nicotine concentrations in each workplace as well as on the low and high end of the range of mean nicotine concentrations reported. Exposure of workers in the "5 Bs" was assessed based on eight hours per work day, five days per week, except for bingo parlour workers whose exposure was based on a two hour workshift twice a week. Although most workers likely work less than 40 years in these establishments, Siegel and Skeer argue that the assessment of risk should be based on the safety of the working conditions over a long period, and the lowered risk among short-term workers should not change the assessment of these risks (i.e. that transient exposures no matter how high would not pose a cancer risk to workers who tend to work in these premises only for several years).

Repace and Lowrey's model also used a forty year working lifetime and atmospheric nicotine measurements to estimate nonsmokers' ETS lung cancer risk in individual workplaces. They derived a health-based standard for ETS in the workplace based on concepts of de minimis (the legal principle of "the law does not concern itself with trifles") and de manifestis risk (a risk of obvious concern). These concepts are discussed by Travis et al[78] in their review of 132 US federal regulatory decisions to determine the level of cancer risk that triggers regulation, and whether there is consistency between and within regulatory agencies in their decision making. Their retrospective review of these regulatory decisions showed that, except for one Federal Drug Administration case, no action was taken to reduce individual lifetime risk levels that were below 1×10^{-6} (1 chance in 1 million). Every chemical with an individual risk above 4×10^{-3} (4 chances in 1000 that chronic exposure will lead to cancer) was regulated. These authors mention that in a population the size of the United States, de manifestis risk drops to about 3×10^{-4} (3/ 10,000) which represents a population risk approaching 250 cancer deaths. Repace and Lowrey, in their model, show that the equivalent air nicotine (8-hour TWA) concentration of 2.3 µg/m³ (for 40 years working lifetime)

represents a risk level of 3/10,000, as does a steady state urine cotinine and steady state plasma cotinine concentrations of 0.08 ng/ml and 0.12 ng/ml respectively.

The US Occupational Safety and Health Administration (OSHA) often consider action based on a "significant risk" level (called the "significant risk of material impairment of health") of 1 in 1000 and 45 years of working life. Based on this OSHA definition of significant risk, Repace et al have estimated significant health risk for ETS exposure would occur at or above 6.8 µg/m^3 for lung cancer, and at 0.68 µg/m^3 for heart disease for a working lifetime[79].

Authors of the 7-City study[76] concluded that mean nicotine levels in smoking-permitted bars and discos in Vienna and Barcelona translate into an estimated lifetime excess lung cancer risk for bar workers of 16 per 1000 and 12 per 1000 respectively, assuming an average 40 year working lifetime.

Hammond et al[80] found worksite (other than in the hospitality industry) smoking policy to have a major effect on air nicotine concentrations, increasing from a median of 0.3 µg/m^3 at worksites that banned smoking to 1.3 µg/m^3 at worksites that restricted smoking to designated areas, and to 8.6 µg/m^3 at worksites that permitted smoking. Comparing nicotine concentrations across a range of public places by smoking policy in 7 European cities[55] also showed that these were likely lower in those public places where smoking was banned compared to where smoking was permitted in designated areas.

However, smoking policy change in the hospitality industry has not been accompanied by the same magnitude of decrease shown by Hammond et al[80]. A recent study investigated whether a smoking ban in Ireland had an impact on secondhand smoke exposure for hospitality workers using a pre- and post-smoking ban design[81]. Unlike earlier studies it monitored air nicotine concentrations as well as salivary cotinine levels of non-smoking workers before and after the smoking ban. Salivary cotinine concentrations were reduced by 69%, from 1.6 ng/ml to 0.5 ng/ml. Self reported secondhand smoke exposure showed a significant reduction from a median of 30 hours a week to zero ($p<0.001$). Median air nicotine concentrations were reduced from 35 µg/m^3 to 5.95 µg/m^3 after the ban, a decline of 83%. At baseline, three bars (16%) were below the 6.8 µg/m^3 air nicotine risk level for lung cancer; at follow up this increased to 10 (53%). Eight of the 19 bars included in this study had instituted designated outside smoking areas adjacent to the main entrance doorway. These consisted of areas cordoned off by waist high demountable screens. At follow up these eight pubs showed higher median nicotine concentrations indoors (mean = 13 µg/m^3) than their counterparts (mean= 5.6 µg/m^3) that did not institute these outside smoking areas. This study suggests that smoking outdoors in the vicinity of building entrances and other openings may well influence ETS concentrations indoors. Smoke may drift indoors from these informal outdoor gathering places that permit smoking.

Designated smoking areas, either in separate rooms or by isolating smoking patrons to a certain proportion of the seating area, is another policy option adopted by the hospitality industry to protect the non-smoking public. One study in British Columbia[82] showed that mean PM_{10}, $PM_{2.5}$ and cadmium (a more specific marker for ETS) were significantly higher in unrestricted smoking establishments (190 µg/m^3, 190 µg/m^3, 6.5 ng/m^3 respectively) than in restricted (54 µg/m^3,

57 µg/m³, 1.3 ng/m³ respectively) and non-smoking establishments (44 µg/m³, 38 µg/m³, 0.97 ng/m³ respectively). Mean cigarette counts accounted for 85% of the variability in airborne cadmium concentrations, indicating that ETS is the major source of particulate matter in restaurants that allowed smoking. The authors of the study concluded that smoking restrictions reduce, but do not eliminate, ETS exposures of restaurant patrons in non-smoking areas. Exposure to ETS is further reduced in non-smoking establishments, but the particulate exposure may also be influenced by other factors, especially differences in ventilation and cooking emissions.

Another study examined $PM_{2.5}$ and nicotine concentrations in restaurants in the US that restricted smoking in restaurants to one third of the seating area[83]. Median concentration of $PM_{2.5}$ and nicotine were 40% (20.7 µg/m³ and 53.2 µg/m³) and 60% (1.0 µg/m³ and 3.2 µg/m³) lower respectively in the no smoking than in the smoking sections. The 7 city European[76] study showed mixed results: in Vienna and Florence there was no difference in the median nicotine concentrations between smoking and non smoking sections in restaurants, in Paris this was shown to reduce exposure although not completely, and in Orebro (Sweden) the nicotine concentration in the non-smoking section was at the level of detection (0.01 µg/m³). These differences could have been related to different smoking policy regulations, compliance by patrons and workers, ventilation system and weather, as well as differences in the placement of the sampler. None of these were described or discussed in the paper.

Recent studies also suggest that the location of the non-smoking area, whether in a separate room or located within or adjacent to smoking permitted areas and ventilation systems, influences ETS exposure levels[84]. An Australian study compared nicotine concentrations in hospitality premises[85]. The mean nicotine concentrations in the separate non-smoking room was 10.7 µg/m³ compared to 24.2 µg/m³ in the smoking areas. Where the designated no-smoking area was a subsection of a room, where smoking otherwise occurred, the mean nicotine concentration in the no-smoking section was 19.4 µg/m³ compared to 54.3 µg/m³. These separate non-smoking rooms were initially differentiated according to whether or not they had separate ventilation systems. Preliminary assessment showed that data from these premises were not notably different from venues that did not provide separate ventilation for these rooms. All such venues were treated as one group for the analysis. Thus, designated no smoking areas, whether in a separate room or within one space, provides about a 50% reduction in exposure, a level that may be less than patrons might have understood.

In addition, studies have shown that the use of ventilation systems (sophisticated HVAC systems and extractor fans in either the on or off mode) did not have a significant effect on ETS marker concentrations in either smoking or non-smoking areas. In 1999 the UK Government introduced a Public Places Charter. Its intent was to permit customers or patrons to make an informed choice about their potential ETS exposure. Five categories of pub establishments were created: non-smoking at all times, clearly defined designated smoking areas, ventilated premises with separate areas for smoking and non-smoking, ventilated premises with smoking permitted throughout premises, and smoking-permitted premises throughout with no segregation or special ventilation equipment. Carrington et al[86] conducted a study of sixty representative pubs and bars in the Greater Manchester area between October 2000 and July 2001 to examine the effects of smoking area status and

ventilation on ETS concentrations. At each pub surveyed, one of three types of ventilation systems were identified: mechanical ventilation applied to sophisticated systems that included electrostatic filters, heating, ventilation and air conditioning units, extractor fans (wall or ceiling mounted and that expelled internal air through the unit) in the on mode and extractor fans in the off mode (representing natural ventilation conditions). A formal assessment of ventilation efficiency was not carried out. ETS markers RSP, UVPM, FPM and SolPM were sampled and analyzed using standard methods. Data from pubs with both smoking and non-smoking areas were used for comparison.

Similar to findings from other studies, results showed that the provision of non-smoking areas significantly reduces ETS marker pollution in non-smoking areas compared to smoking areas. However, ETS marker concentrations were reduced even in the smoking areas within these premises. Median concentrations of RSP and nicotine were reduced by 18% and 68% respectively in the non-smoking sections, with the smaller reduction in RSP likely explained by the contributions of cooking emissions and ambient air. The greater reduction associated with nicotine (68%) compared to UVPM (27%), FPM (34%) and SolPM (39%) suggests that a more appropriate methodology in measuring ETS marker concentrations in hospitality premises might include nicotine as a vapour phase marker and SolPM as a particulate marker.

ETS concentrations in the smoking and non-smoking sections of these pubs were compared to examine the effects of different ventilation systems (mechanical ventilation, extractor fan and natural ventilation). Concentrations for nicotine, UVPM, FPM and SolPM were lower in the no-smoking areas compared to the smoking areas for all pubs. In addition, for each ETS marker, concentrations were lowest in mechanically ventilated pubs followed by pubs ventilated by extractor fans and then those by natural ventilation. Pubs ventilated with extractor fans in the on position showed nicotine concentrations of 72.7 $\mu g/m^3$ in the smoking sections compared to 19.3 $\mu g/m^3$ in the non-smoking section. Pubs using natural ventilation showed nicotine levels of 49.6 $\mu g/m^3$ and 39.7 $\mu g/m^3$ in the smoking and non-smoking sections and those equipped with mechanical ventilation 32.6 $\mu g/m^3$ and 17.3 $\mu g/m^3$ respectively. The highest median concentrations of nicotine and ETS particle markers corresponded to areas ventilated by active extractor fans. These results demonstrate that ventilation systems are not as effective as they are believed to be. It is possible that non-smoking areas ventilated by mechanical systems are also subject to some smoke drift and re-circulation of ETS pollutants through air being mixed and recycled, as indicated by the slight increase in particulate ETS markers in mechanically ventilated non-smoking areas compared to non-smoking areas that were naturally ventilated. Another possible reason could be that patrons may not be complying with the rules in these no-smoking areas. However, the smoking behaviour of patrons was not observed during the sampling protocols.

A more recent publication,[87] using the data from Carrington et al[86] study, showed statistically significant differences for all ETS markers except RSP when comparing smoking and non-smoking areas of these pubs. ETS concentrations were significantly lower (27% – 69%) in non-smoking areas in comparison to smoking areas, the range in the data reflecting the different ETS markers. Using a formula derived from chamber studies, these researchers showed that smoking was the major source

of particulate matter, even in non-smoking areas. The proportion of particle (RSP) that could be attributed to ETS was considerable, even in non-smoking areas (43 – 55%).

To investigate the effects of ventilation method on ETS levels, pubs allowing smoking throughout their premises were categorized by ventilation system (mechanical, active extractor fan and natural ventilation) and compared. Results showed that median concentrations of ETS (nicotine, UVPM, FPM, SolPM) markers were highest in those pubs ventilated by extractor fans and were lowest in pubs ventilated by mechanical ventilation. However, these differences were not statistically significant.

The provision of non-smoking areas clearly reduces the concentration of ETS in the non-smoking compared to the smoking areas. Non-smoking areas showed a mean nicotine concentration of 28 µg/m^3 (median: 26 µg/m^3) compared to 89 µg/m^3 (median 63 µg/m^3) in the smoking sections, a 57% reduction. ETS particulate markers showed smaller reductions. The greater reduction in nicotine levels, compared to particulate markers, may be associated with its tendency to be absorbed by room surfaces and furnishings. As in the above study, these authors point out the need to sample for nicotine as well as ETS particle phase markers when monitoring ETS exposure levels. Both studies conclude that the effectiveness of ventilation methods in controlling ETS levels in pubs and bars appears to be limited. Both studies also raise questions about the capacity of mechanical ventilation systems to adequately control particulate phase ETS pollutants, and to prevent its re-circulation within buildings

An Australian study monitored ETS in both smoking and non-smoking hospitality premises in metropolitan Adelaide (South Australia) and assessed the effectiveness of control measures such as ventilation, separation distance and partial barriers to prevent the flow of ETS from smoking permitted areas to non-smoking areas[88]. Seven hospitality premises: hotels (4), clubs (1 sporting club and 1 nightclub) and cafes (1 unlicensed) were monitored for airborne nicotine and particulate matter (PM_{10}) as markers of ETS exposure. Monitoring in the smoking and non-smoking sections took place simultaneously over a period of 2-4 hours during a normal to busy dinner or lunch session. Five of the seven venues were ventilated with refrigerative air conditioning systems, one used an evaporative air conditioning system and one used both systems. On week prior to monitoring, a field officer visited each site to obtain information on the following: physical dimensions, ventilation type, location of air conditioning supply, return and exhaust openings, and the size, physical separation and potential airborne particulate sources in each venue. During the monitoring, airflow rates through supply vents and exhaust fans were measured using a Shortridge Instrument Model CFM Flow Meter with electronic micrometer. Ventilation settings were also monitored before, during and following the sampling period. Also noted were the number of patrons and the location of the smokers in each venue.

The results showed that average concentrations of nicotine and PM_{10} were higher in smoking-permitted areas (nicotine = 15 µg/m^3, PM10 = 255 µg/m^3) compared with non-smoking dining areas (nicotine = 7 µg/m^3, PM_{10} = 192 µg/m^3). Unenclosed non-smoking dining areas showed higher mean nicotine concentration (7.5 µg/m^3) and median PM_{10} concentrations (200 µg/m^3) compared to enclosed non-smoking areas (nicotine = 4.4 µg/m^3 and PM10 = 17 µg/m^3). These results suggest that

non-smoking areas within hospitality premises have approximately 2-fold lower ETS concentrations than smoking-permitted areas. These results indicate that mechanical ventilation is only partially effective in preventing the propagation of ETS throughout these premises. Enclosing non-smoking areas and a separation distance of approximately 10 m from a smoking area may further reduce nicotine concentration.

Hospitality premises ventilated with refrigerative air conditioning systems showed the highest concentrations of nicotine. Refrigerative air-conditioning systems are associated with low turnover of outside air compared to evaporative systems. It was noted that in Australia there was an increasing trend toward the installation of refrigerative HVAC or air conditioning systems due to their superior thermal control through recycling of air within the building. Differences in filtration of particulates and vapours through ventilation and air handling systems determine the efficiency with which they are re-circulated in buildings. Unlike refrigerative systems, evaporative systems do not recycle air in the building. It supplies outside air to buildings and air within buildings is flushed out through windows, doors and other openings, potentially removing ETS pollutants from indoor environments. In their discussion these authors also mention that exhaust fans are often considered for smoking-permitted areas but their performance can be compromised by inappropriate location and competing air currents.

5.1 Summary

Smoking policy changes in the hospitality industry have not been accompanied by the same decrease in exposure levels as shown in workplaces outside the hospitality industry.

Meta-analysis of the mean nicotine concentrations in the 5 Bs (betting establishments, bowling alleys, billiard halls, bars and bingo parlours) showed that these were 2.4 to 18.5 times higher than in offices or residences, and 1.5 to 11.7 times higher than in restaurants.

Overall, smoking restrictions reduce, but do not eliminate ETS exposure to restaurant and hospitality industry workers and patrons. When no- smoking areas are located in a separate room, and there is a physical barrier separating it from a smoking area, there is a 50% to 66% reduction in ETS marker concentrations in the designated no-smoking area. The reduction of ETS marker concentrations in these separate no-smoking rooms is greater than in the no-smoking areas that are subsections of rooms where smoking otherwise occurs.

Ventilation systems reduce ETS marker concentrations. ETS marker concentrations were usually lowest in both the smoking and non-smoking sections of hospitality premises equipped with mechanical ventilation, compared with those using extractor fans and natural ventilation. These marker concentrations were usually highest in both the smoking and non-smoking areas in premises that relied on extractor fans. Premises that relied on natural ventilation showed lower ETS marker concentrations compared with those using extractor fans. However, ventilation systems are not as effective as they are believed to be. Residual mean and median air nicotine concentrations in both the smoking and non-smoking sections of hospitality premises, regardless of the type of ventilation system in place, may still present a significant risk to health.

An Australian study suggests that enclosing non-smoking areas and placing a separation distance of approximately 10 m from a smoking area may reduce nicotine concentrations. Refrigerative air-conditioning systems (which recycle air within a building) compared to evaporative air conditioning systems (which replaces air in a building) are only partially effective in preventing the propagation of ETS throughout a building.

In response to a law banning smoking in all workplaces in Ireland some pubs set up outdoor patios where smoking was permitted. Comparison of pre and post- smoking ban results showed that indoor air nicotine concentrations declined as a result of the ban. Pubs with outdoor patios had higher indoor air nicotine concentrations than pubs that did not have these patios. Smoke may drift indoors from these informal outdoor gathering places located next to building entrances and other openings.

6.0 ETS in Outdoor Public Places

With bans on smoking in indoor public places and workplaces, remaining ETS exposures may occur largely in outdoor settings. Mulcahy et al[81] mention that the policy prohibiting smoking in indoor public workplaces in Ireland has spawned the growth of outdoor patios, pavement cafes, and other informal outdoor gathering places for smokers. There is no legal requirements as to their minimum proximity to buildings or entrances/exists and other openings to buildings. Additionally, Mulcahy et al[81] suggest that the distance at which these outdoor smoking places are located from building entrances and openings may have an impact on indoor exposure levels through smoke drifting indoors.

There are a limited number of studies that have measured ETS exposures in outdoor public places[55, 89]. To fill in the gaps existing in outdoor measurements of ETS the California Air Resources Board (CARB) conducted ambient air monitoring at outdoor smoking areas during 2003[6]. Eight-hour time-weighted average (TWA) nicotine concentrations were measured outside an airport, junior college campus, public building, office complex and amusement park.

Sampling for nicotine was conducted during business hours (8am-5pm) over a three-day time period. Two of the sampling days were spent collecting 8-hours samples and 1 day was spent collecting six 1-hour samples. For each sampling period 2 samplers were placed adjacent to the outdoor smoking area, with a third sampler located away from the smoking area as a background sampler (to monitor background levels) in the expected upwind direction. Staff also collected the following data: wind/speed direction and ambient temperatures, the number of cigarettes smoked during each sampling period and quality assurance samples. The results are presented in Table 4. This study also demonstrated that outdoor concentrations were dependent on the number of cigarettes smoked (source strength), the position of the smokers relative to the monitoring equipment (or receptor), and atmospheric conditions.

The CARB study uses a scenario-based approach to estimate exposure of various subgroups of the California population to ETS. The scenario-based exposure method used the results of CARB's air monitoring study, available indoor ETS concentration data, and scenario-based activity patterns to estimate exposure under different conditions. For non-smokers, not living with or working with smokers, 100% of their exposure may be attributable to the time they spend either in, or in proximity to, these formal and informal outdoor public places that permit smoking.

Table 4: Results of CARB Nicotine Air Monitoring Adjacent to Outdoor Smoking Areas

Site Tested	8-hr data	Concentration ($\mu g/m^3$)	Cigarettes smoked (8 hours)	1-hr data	Concentration ($\mu g/m^3$)[b]	Cigs smoked (1 hour)
Airport	Mean Day 1[a]	0.61	261	Maximum	1.5	61
	Mean Day 2[a]	0.74	326	Mean	0.72	75
	2-day mean	0.68	294	Range	0.36-1.5	
	Mean bkgd	0.021		Mean bkgd	0.46	
Junior College[c]	Mean Day 1	0.035	30	Maximum	0.15	5
	Mean Day 2	0.018	34	Mean	0.051	4
	2-day mean	0.027	32	Range	0.017 – 0.15	
	Range	0.013-0.044		Mean	< EQL[d]	
	Mean bkgd	0.012				
Local Government Centre[c]	Mean Day 1	0.066	59	Maximum	0.18	15
	Mean Day 2	0.055	60	Mean	0.097	11
	2-day mean	0.061	60	Range	0.039-0.18	
	Range	0.042-0.073		Mean bkgd	< EQL	
	Mean bkgd	0.009				
Office Complex[c]	Mean Day 1	0.12	261	Maximum	0.28	31
	Mean Day 2	0.14	251	Mean	0.19	29
	2-Day Mean	0.13	256	Range	0.10-0.28	
	Range	0.11-0.15		Mean	0.06	
	Mean bkgd	0.09		Mean bkgd		
Amusement Park	Mean Day 1	2.6	653	Maximum	4.6	148
	Mean Day 2	2.8	719	Mean	2.4	91
	2-Day mean	2.7	636	Range	0.66-4.6	
	Range	2.4-3.1		Mean	0.17	
	Mean bkgd	0.12				

a Mean concentration of samples adjacent to outdoor smoking area

b Maximim, mean, range and mean background concentration of six 1-hour sampling periods (Means include all samples, with trace values below the EQL assigned 0.017, the midpoint between the EQL and the limit of detection)

cLight to moderate winds occurred on all three days of monitoring at this location

d EQL for 1-hour samples = 0.029 $\mu g/m^3$; EQL for 8-hour samples = 0.0038 $\mu g/m^3$ (1 $\mu g/m^3$ nicotine = 0.15 ppbv)

Source: State of California Air Resources Board (CARB) (2005) Proposed identification of environmental tobacco smoke as a toxic air contaminant. Part A: Exposure Assessment. California Environmental Protection Agency Air Resources Board. Page: v-9

An unpublished report by Repace[90] examines how smoke behaves outdoors. Repace lists 4 key features of a model in discussing the dispersal of ETS emissions from a point source at ground level: (a) the downwind concentration at any location is directly proportional to the mass emission rate at the source, (b) the more turbulent the atmosphere the more rapid the spread of the plume transverse to the direction of plume, (c) the maximum concentration is directly downwind on the plume line and inversely proportional to the downwind distance from the source, and (d) the maximum concentration decreases for higher wind speed because the diffusion constants are inversely proportional to wind speed.

Repace measured secondhand smoke concentrations outdoors at a university campus. Real-time battery operated monitors were deployed in various locations indoors as well as outdoors where smokers were encountered. The same measurements were made using smoldering Camel cigarettes for comparison purposes. Respirable suspended particulate (RSP) with aerodynamic diameter < 3.5 µm; and carcinogenic particulate polycyclic aromatic hydrocarbons (PPAHs) were monitored using MIE personalDataRAM (pDR-1200) and the EcoChem PAS 2000CE respectively. The intent was to quantify the concentration of secondhand smoke levels as a function of distance from the source. Measurements outdoors were taken at different distances downwind from the point source. With breezes blowing from a West-Southwest to North-Northwest at 3 to 7 mph, RSP peaks reached 90 $\mu g/m^3$ (averaging time 5 minutes) within 4 – 6 feet of 4 smokers outside the cafeteria entrance, and 28 $\mu g/m^3$ (average time 2.5 minutes) within 10 – 30 feet from 1- 3 smokers outside the buildings. Peaks for RSP and PPAH reached 60 $\mu g/m^3$ and 60 ng/m^3 (averaging time 1 minute) within 6 feet of the 5 smoldering cigarettes.

To eliminate variation in secondhand smoke concentration due to changes in wind direction during the time it takes to smoke a cigarette Repace devised two sets of outdoor experiments. In one experiment he arranged smoldering cigarettes on chairs in a ring around the RSP and PPAH monitors, at 1.5 meters, 3 and 5 meters. In the second experiment up to 10 smokers were recruited and arranged in a ring around the RSP and PPAH monitors. They sat on chairs and smoked at three different distances in the ring radius around the monitors - 1.5 meters, 3 and 5 meters. The arrangement of the smoldering cigarettes and smokers in a ring allowed the monitors to pick up the smoke plume no matter which way the wind blew.

Figure 1 shows the concentration of secondhand smoke emitted from the smoldering cigarettes as a function of distance. PPAH concentration declines as the inverse square of the source-receptor distance whereas RSP declines inversely with distance. Both reach concentrations close to background levels at approximately 7 meters.

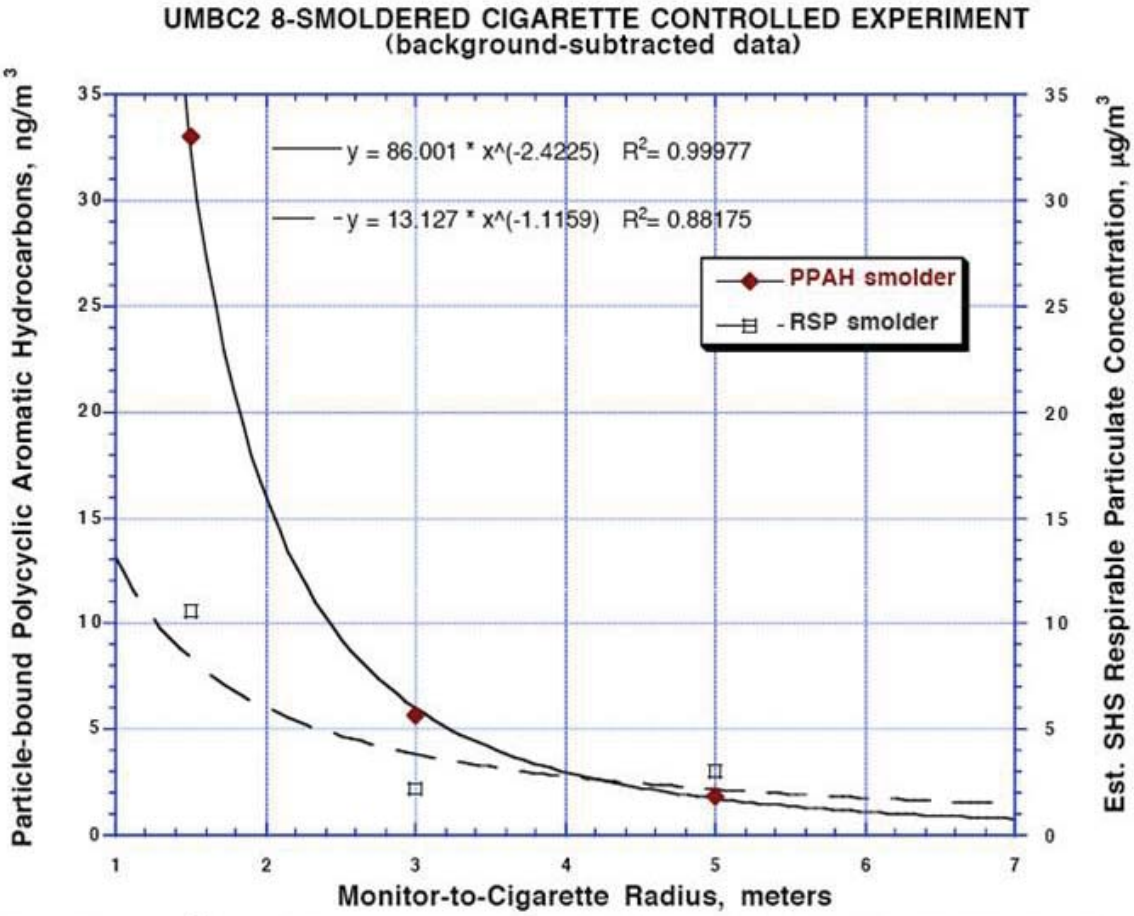

Figure 1. April 15th Experiment. Smoldered cigarettes (Marlboro Medium 100s, filtered) located at 8 equally spaced compass positions at ring radii 1.5, 3, and 5 meters. Curve fits to the PPAH and RSP curves are shown, and extrapolated to 7 meters (23 feet). PPAH declines as the inverse square of the source-receptor distance *x*, whereas RSP declines inversely as the distance, as expected.

source: Repace J (2005) Measurements of outdoor air pollution from secondhand smoke on the UMBC campus. Repace Associates Inc. MD: Bowie

Figure 2 shows the results for the smoldering cigarettes and the recruited smokers added together. There is more variability or scatter when the results are combined (R^2 of 0.88 and 0.10 respectively for RSP). The same dependence with distance is demonstrated in Figure 2. The distance-decay curve for RSP suggests that it may decline more slowly than PPAH concentrations and reach background levels at distances greater than 7 meters (10 meters approximately).

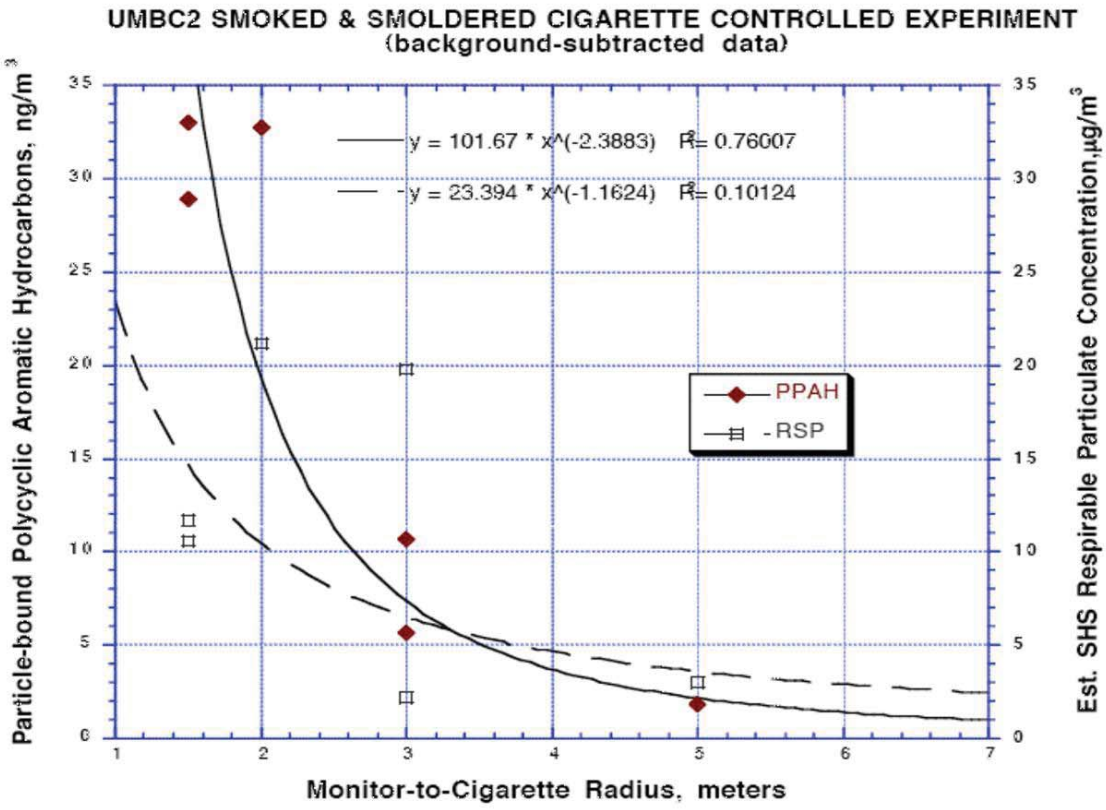

Figure 2. Smoked cigarettes at 1.5, 2, and 3 meters overlayed on the smoldered cigarette plot of figure 5 with curve fits to the combined PPAH and RSP data, extrapolated to 7 meters (23 feet). Although there is more scatter in the data when the smokers are added, approximately the same dependence of PPAH and RSP with distance is seen.

Source: Repace & Associates (2005)

Repace argues that smoking outdoors cannot be ignored, given that the variables that determine its concentration are source strength (i.e. the number of smokers), distance of the receptor from the source, and atmospheric conditions. His experiments demonstrate that if a receptor such as a doorway, open window, or air intake vent is surrounded by an area source (i.e. group of smokers nearby) then regardless of the wind direction, the receptor is always downwind from the source. Greater number of smokers standing in an area leads to higher concentrations locally which will then dissipate with distance, and weather conditions. As mentioned above, some of the monitors deployed outside the UMBC cafeteria entrance recorded RSP peak levels as high as 100 µg/m³ with 4 smokers standing within 4-6 feet outside the cafeteria entrance (averaging time 5 minutes), and 150 µg/m³ with 1 to 8 smokers at closer distances

Secondhand smoke causes a number of acute respiratory and sensory irritant symptoms which can be attributed to RSP. The studies reviewed above demonstrate that besides chronic diseases (lung cancer and coronary heart disease), ETS exposure can cause eye, nose and throat irritation, headaches, dizziness and nausea, and is known to trigger asthma among asthmatics. Junker et al[91] examined ETS odor detection thresholds and eye, nasal and throat irritation thresholds for twenty-

four females (age range 18-35) nonsmokers (healthy, with no record of allergy to pollen or dust, and not to have smoked in the last 5 years). They participated in an olfactory and full-body exposure experiment where they were subjected to repeated exposures of ETS in ascending concentrations over the course of two hours in an exposure chamber. They provided baseline measures via questionnaire to indicate their annoyance to ETS, automobile exhaust fumes, solvents and perfumes. Participants provided baseline information before the experiments. An odor threshold value was given when a subject stated perceiving an odor during two consecutively ascending concentrations. Subjects also scaled their perception of the intensity of their sensory symptoms. The results for the sensory symptoms showed that levels of irritation began at ETS-$PM_{2.5}$ concentrations as low as 4 $\mu g/m^3$ and odor was detected at ETS-$PM_{2.5}$ concentrations as low as 1 $\mu g/m^3$. Extrapolating from Junker et al's[89] findings to his distance decay curves (Figure 2), Repace estimates that odour would be detectable as far as 7 meters from the source, and levels of irritation would begin at 4 meters from the source.

6.1 Summary

Two studies examine ETS exposure in outdoor public places. Outdoor ETS exposures are not well characterized or available in the published scientific literature. Accurate characterization of outdoor ETS exposures will require additional research.

Repace demonstrated that if a receptor such as a doorway, open window, or air intake vent is surrounded by an area source (i.e. group of smokers nearby), then regardless of the wind direction, the receptor is always downwind from the source. ETS concentrations will be affected by the number of smokers, distance of the receptor from the area source, and atmospheric condition.

Cigarette smoke respirable suspended particulate (RSP) concentrations decline more slowly than cigarette smoke particulate polycyclic aromatic hydrocarbons (PPAH) concentrations.

A study of 24 young healthy females in an exposure chamber reported eye, nose and throat irritation at ETS-$PM_{2.5}$ concentrations as low as 4 $\mu g/m^3$ and odor was detected at ETS-$PM_{2.5}$ concentrations as low as 1 $\mu g/m^3$. Extrapolating from Junker et al's[89] findings to his distance decay curves (Figure 2), Repace (2005) estimates that odour thresholds could be encountered as far as 7 meters (23 feet) from the source, and sensory irritation would begin at approximately 4 meters (13 feet) from the source.

7.0 Conclusions

The objectives of this report were to (i) review the epidemiologic studies of ETS exposures in the workplace and home settings to evaluate the extent to which these findings could be generalizable to outdoor and other settings where ETS exposure occurs, (ii) summarize the peer-reviewed scientific evidence on the health effects of ETS exposure in indoor and outdoor public spaces, and (iii) review and summarize the exposure levels and associated levels of risk.

The summary of epidemiologic studies of the health effects of ETS exposures in the workplace and home settings is based on reviews of reviews. Six independent government and national scientific reviews were completed in the 1990s in the US (USEPA[1], CalEPA[3], NTP[44]), Australia (NHMRC[42]), United Kingdom (SCOTH[43]) and internationally (WHO[41]). Each of these reviews focused on a different number and types of health endpoints that are listed in Appendix Table A. They concluded that exposure to ETS is causally related, or possibly causally related, to fifteen health outcomes. These include four developmental diseases or conditions, seven respiratory diseases or conditions, three cancers and coronary heart disease.

In 2004 the International Agency for Research on Cancer[26] (IARC) completed its review of evidence relating to involuntary smoking. It concluded that there is a statistically significant and consistent association between lung cancer risk in spouses of smokers and exposure to secondhand smoke at home and in the workplace. The evidence linking ETS exposure to breast cancer is inconsistent, as is the evidence linking childhood cancer and ETS exposure from parental smoking. Data relating to the following cancers among adults were considered sparse, inconsistent and bias could not be ruled out: cancers of the naso-pharynx, nasal cavity, paranasal sinuses, cervix, gastrointestinal tract and cancers of all sites combined.

Important conclusions for non-cancer health effects of ETS exposure by IARC (2004) included:

- Exposure to ETS increases the risk of a coronary heart disease event by 25 -30% for non smokers living with a smoking spouse compared to non-smokers living with a non-smoking spouse.
- Involuntary smoking has an adverse effect on the respiratory system. In adults, the strongest evidence for a causal relation exists for chronic respiratory symptoms.
- Full-term infants born to women who smoke weigh about 200 g less than those born to non-smokers. A smaller adverse effect is attributed to babies born to mothers who are exposed to secondhand smoke.
- In contrast to the findings for active smokers, women exposed to secondhand smoke do not appear to weigh less than women not exposed to secondhand smoke.
- No consistent association of maternal exposure to ETS with fertility or fecundity has been identified.

- There is no clear association of ETS exposure with age at menopause.

A more recent review[45] of the relationship between ETS exposure and respiratory effects in children suggests that exposure to ETS in-utero has a greater effect on their lung function than postnatal exposure. Additionally, in-utero exposure to maternal smoking was independently associated with deficits in lung function that were larger for children with asthma. Boys and girls with a history of in-utero exposure to maternal smoking showed deficits in maximum midexpiratory flow (MMEF) and a decrease in FEV(1)/FVC ratio. Compared with children without asthma, boys with asthma had significantly larger deficits from in-utero exposure in FVC, MMEF, and FEV(1)/FVC, and girls with asthma had larger decreases in FEV(1)/FVC.

As noted above, the health effects of exposure to ETS in these epidemiologic studies are based on non-smoking spouses of smokers or non-smokers exposed in the workplace. Exposure to ETS in the home or in the workplace is generally of long duration. Commonly, for non-smoking spouses of smokers the husband's smoking status is the exposure surrogate and exposure has been assessed in the home. Similarly, where exposure is assessed in the workplace the smoking status of co-workers is the exposure surrogate. Cotinine concentration in biologic fluid, the number of years married to a smoking spouse, the number of cigarettes smoked per day by the smoking spouse or smoking co-workers are used to calculate the total dose and the risk to an exposed individual. The health outcomes examined in these studies are associated with long term or chronic ETS exposure, and there is a latency effect associated with these health endpoints. These studies also adopt a prospective cohort or a case-control design.

The health effects of ETS exposure from epidemiologic studies have led to many successful public health initiatives to protect non-smokers from ETS exposure. Smoking bans have been progressively introduced in workplaces and enclosed public places where it was inferred that workers and the public would be subject to the adverse health effects resulting from chronic ETS exposure. For a given environment, the harm from exposure to passive smoking depends on the time spent in that environment and the concentration of ETS in that air space. The concentration of ETS, in turn is affected by the size of the space, the number of people smoking there and the ventilation rate.

The potential health effects of short term exposure to secondhand smoke have been the subjects of extensive research. One population study showed that mild asthmatics demonstrate an increased risk of respiratory and sensory irritative symptoms, and extra bronchodilator use ever at low ETS exposure levels of 0.05 $\mu g/m^3$ (nicotine)[55]. This study is one of the first in the literature to demonstrate that with the banning of smoking in indoor workplaces and public places, outdoor locations are becoming relatively more important as a source of non-residential ETS exposure. Another population based study in Helena[57], Montana suggests that laws controlling ETS exposure may reduce morbidity from heart disease. Where studies focused ETS exposure of non-smoking workers in hospitality premises, researchers have adopted a pre and post study design to examine the impact of smoking policy. These studies demonstrate that higher exposures to ETS (measured by salivary cotinine concentrations) are related to a poorer respiratory and sensory symptom profile among non-smoking workers in these premises[59,60,61,62,63,64,65,66,67,68,69,70,71,72]. Studies have found the introduction of smoke-free policies in hospitality premises can be accompanied by:

- a significant reduction in the number of hours spent exposed to ETS during work,
- a significant decline in the prevalence and number of different respiratory and sensory irritative symptoms reported,
- a significant decline in salivary cotinine concentrations and
- improvements in lung function among both smoking and non-smoking workers.

Smoking policy change in the hospitality industry has not been accompanied by the same decrease in exposure levels shown in workplaces outside the hospitality industry[73,74,76]. Meta-analysis of the mean nicotine concentrations in the 5 Bs (betting establishments, bowling alleys, billiard halls, bars and bingo parlours) showed that these were 2.4 to 18.5 times higher than in offices or residences, and 1.5 to 11.7 times higher than in restaurants[73].

Overall, smoking restrictions reduce, but do not eliminate ETS exposure of workers in restaurant and other hospitality industry premises. There is a 50% to 66% reduction in ETS marker concentrations when the no-smoking area is located in a separate room. The reduction of ETS marker concentrations in the separate no-smoking room is greater than that in the no-smoking areas that are subsections of rooms where smoking otherwise occurs[80,81,82,83,84,85].

Ventilations systems reduce ETS marker concentrations, and mechanical ventilation systems are relatively more effective than extractor fans or natural ventilation[86,87]. However, residual mean and median air nicotine concentrations in both the smoking and non-smoking sections of hospitality premises, regardless of the type of ventilation system in place, may still present a significant risk to health.

An Australian study showed that enclosing non-smoking areas and locating it at a distance of approximately 10 m away from a smoking permitted area may reduce nicotine concentrations. This study also suggests that refrigerative air conditioning systems which recycle air, compared to evaporative air conditioning systems that supply fresh air, are only partially effective in preventing the propagation of ETS throughout a building[88].

In response to a law banning smoking in all workplaces in Ireland some pubs set up outdoor patios where smoking was permitted. Comparison of pre and post- smoking ban results showed that indoor air nicotine concentrations declined as a result of the ban. Pubs with outdoor patios had higher indoor air nicotine concentrations than pubs that did not have these patios[81]. Smoke may drift indoors from these informal outdoor gathering places located next to building entrances and other openings.

With laws banning smoking in workplaces and indoor public places, outdoor gathering places are likely relatively more important as sources of non-residential ETS exposure[55,81]. Only two studies were identified that attempted to characterize outdoor ETS exposure levels[6,90]. Unlike indoor ETS marker concentrations that remain in the room long after a cigarette has been extinguished,

secondhand smoke outdoors does not accumulate. Outdoor ETS concentrations are more sensitive to source-receptor distance and wind conditions. Therefore for each cigarette smoked total dose to an individual indoors will be greater than for a cigarette smoked outdoors.

Repace (2005)[90] demonstrated that respirable suspended particulate (RSP) concentrations decline more slowly than cigarette smoke polycyclic aromatic hydrocarbons (PPAH) concentrations. Ultrafine particulate matter can cause breathing difficulty for those with chronic respiratory diseases and trigger asthmatic attacks among asthmatics.

7.1 Future Research

Only two studies, one of which was published in the gray literature, were found that characterized outdoor ETS exposures. More studies are needed to characterize ETS exposure of workers and non-workers in outdoor patios, pavement cafes and other informal outdoor gathering places. There is a need to replicate Repace's study in different outdoor settings and weather conditions.

There is some suggestion that the distance separating these outdoor gathering places from building entrances and other openings may influence ETS concentrations indoors. Additional studies would be required to explore whether smoking outdoors at different distances from building entrances influence ETS levels indoors or whether different HVAC systems make a difference to indoor ETS levels when smoking is permitted at different distances from building openings. Additional work could also provide information on how smoke moves indoors from outdoor locations and the effect of barriers around outdoor smoking locations in influencing smoke drift.

Appendix Table A

Summary of conclusions of six major reviews concerning exposure to second-hand

Disease or condition	1992: USEPA	1997: Australian NHMRC	1997: Cal EPA	1998: UK SCOTH	1999: WHO	2000: US National Toxicology Program
Developmental Effects						
Fetal growth : low birth-weight or small for gestational age		√ ?	√	√	√	
Sudden infant death syndrome (SIDS)		√	√	√	*	
Spontaneous abortion			*			
Adverse impact on cognition and behaviour			*		*	
Respiratory Effects						
Acute lower respiratory tract infections in children (e.g. bronchitis and pneumonia	√	√	√	√	√	
Asthma exacerbation in children	√	√	√	√	√	
Asthma induction in children		√?	√		* √	
Respiratory symptoms	√	√	√	√	√	
Middle ear disease in children	√	√	√	√	√	
Decreased pulmonary function	√	√	*		*	
Exacerbation of cystic fibrosis			*			
Carcinogenic Effects						
Lung cancer	√	√	√	√		√
Nasal sinus cancer		*	√			√?
Cervical cancer			*			
Cardiovascular effects						
Coronary heart disease		*	√	√		

A check mark (√) indicates that the review concluded the relationship to the disease or condition was causal. An asterisk (*) indicates that the review concluded the relationship was possibly causal. In both cases protective public health action is warranted. A blank cell indicates that the relationship was reviewed only briefly or not at all. A question mark (?) indicates some inconsistency or ambiguity in the report's conclusions as to whether the relationship is causal or not.

Source: Ontario Tobacco Research Unit (2001). Protection from second-hand tobacco smoke in Ontario. A report of the Ontario Tobacco Research Unit, University of Toronto.

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