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**REPORT ON THE HEALTH EFFECTS OF ENVIRONMENTAL  
TOBACCO SMOKE (ETS) IN THE WORKPLACE**

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## **PREFACE**

This report has been prepared by an independent scientific working group. The working group was commissioned by the Health and Safety Authority and the Office of Tobacco Control, Ireland, to 'identify and report on the degree of consensus that exists among leading international scientific authorities on the question of the hazard and risk posed by environmental tobacco smoke to human health in the workplace'.

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## **ACKNOWLEDGEMENTS**

The scientific working group thank

- \* Brenda Guihen for her efficient administrative assistance in compiling this report.
- \* Dennis Van Heeswijk for setting up the reference database.
- \* Colleagues, friends and family for editorial comment.

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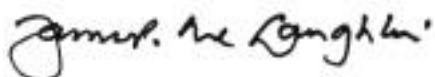
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## CONFLICT OF INTEREST STATEMENT

We the undersigned reviewers do not have any vested interest in the tobacco industry, nor with smoking cessation products, nor have we been involved to date with any anti-tobacco lobby groups.



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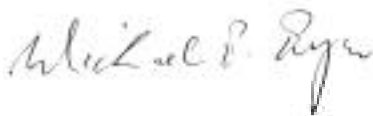
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## EXECUTIVE SUMMARY

### Introduction

The recent declaration (2002) by the World Health Organization's International Agency for Research on Cancer that exposure to environmental tobacco smoke (ETS)<sup>1</sup> is carcinogenic<sup>2</sup> to humans reflects the position of the scientific community as a whole. The increasing awareness that ETS is harmful to health places an onus on governments to safeguard public health by providing legislation to protect the general public from passive (involuntary) smoking<sup>3</sup>. The focus of occupational legislation is to provide safe work environments. Recent court cases have demonstrated that the protection of workers from ETS at their place of work is becoming an important occupational health issue.

Smoking rates vary by occupation within and between countries. In Ireland, smoking rates are highest in lower income groups. Although data are limited, the socio-economic differences appear to be reflected in the workplace with, for example, low smoking rates in teachers and high rates among construction workers. Current Irish legislation prohibits smoking in certain categories of workplace but there are many exceptions such as pubs, nightclubs and bookmakers. Workers in these and other leisure industries are exposed to high levels of ETS due to smoking by their customers.

Given the increasing concern about the health effects of ETS, the Health and Safety Authority and the Office of Tobacco Control commissioned an independent scientific working group to 'identify and report on the degree of consensus that exists among leading international scientific authorities on the question of the hazard and risk posed by environmental tobacco smoke to human health in the workplace'. The findings are presented in this report.

### International position statements on health effects of workplace Environmental Tobacco Smoke (ETS)

Numerous position statements on the harmful health effects of ETS have accumulated from government and scientific bodies worldwide.

On the basis of the evidence linking ETS with lung cancer, two independent assessments of carcinogenic risks to humans have declared ETS to be carcinogenic<sup>4</sup>. In relation to workplace exposure, many of the position statements specify that there is compelling evidence that working with smoking co-workers per se increases the risk of lung cancer. Statements on the relationship with other (i.e. non lung) cancers are less uniform.

Most of these bodies state that ETS causes heart disease although some describe it, less emphatically, as increasing the risk of heart disease. Heart disease is the single most important cause of death in Ireland and a major cause of premature death. Because heart disease is so common, much more common than lung cancer for example, from a public health perspective the association of ETS with heart disease is of the utmost importance.

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<sup>1</sup>ETS: when non-smokers share a space with someone who is smoking they are being exposed to ambient tobacco smoke. This ambient tobacco smoke is called environmental tobacco smoke (ETS), second-hand smoke or passive smoke.

<sup>2</sup>*Carcinogen*: Any substance capable of producing cancer or a chemical that causes or induces cancer.

<sup>3</sup>*Passive smoking*: the act of breathing environmental tobacco smoke is called passive smoking.

<sup>4</sup>WHO International Agency for Research on Cancer Monograph (Summary 2002) and US NIH National Toxicology Program 9th Report on Carcinogens (2000)

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Most agencies consider that exposure of pregnant women to ETS causes lower birth weight in their babies, and that children exposed to ETS are at increased risk of respiratory disease and sudden infant death syndrome (cot death). This has obvious implications for pregnant working mothers and for children spending time in an adult's workplace.

There is also general agreement that ETS causes respiratory disease in adults.

### **ETS constituents**

ETS is made up of exhaled mainstream smoke<sup>5</sup> and sidestream smoke<sup>6</sup>. Emissions contain both particulate and vapour contaminants. Sidestream smoke is the major component of ETS, contributing over half of the particulate matter and nearly all of the vapour phase. ETS, sidestream smoke and mainstream smoke are complex mixtures of over 4000 compounds. These include more than 50 known or suspected human carcinogens. A number of irritants and cardiovascular toxicants, including carbon monoxide and nicotine, are also present.

There are differences as well as similarities between the mainstream smoke and sidestream smoke components of ETS. The main differences are due to differences between the temperature of combustion of the tobacco, pH (level of acidity), and degree of dilution with air. These differences are consistent with animal and genotoxicity studies which suggest that sidestream smoke is more potent than mainstream smoke per unit of tobacco smoked.

### **Measurement of ETS**

Exposure can be assessed through measurement of indoor air concentrations of ETS constituents, through use of personal monitors, by measurement of biological markers<sup>7</sup> in saliva, urine and blood, including DNA adducts<sup>8</sup>, and by self-assessment using questionnaires. There are advantages and disadvantages associated with the various techniques.

The most widely used marker compounds for assessing the presence and concentration of ETS in indoor air are vapour-phase nicotine and respirable suspended particle mass. Although there are problems with measuring nicotine in the air, it has the advantage of being specific to tobacco smoke and of being present in large quantities in ETS. Respirable suspended particles are also present in large quantities in ETS but are not unique to ETS. When respirable suspended particles are used as a marker for ETS, background levels from other sources must be accounted for.

Carbon monoxide may also be used as a marker for ETS. There are many reliable and sensitive instruments commercially available to monitor carbon monoxide in indoor air. They can provide a continuous record of the carbon monoxide concentration, and many of them can be hand-held for personal monitoring. However, there are many sources of carbon monoxide in addition to tobacco smoke.

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<sup>5</sup>*Mainstream smoke*: The smoke inhaled and exhaled by smokers directly from tobacco products.

<sup>6</sup>*Sidestream smoke*: A mixture of the smoke emitted from the smouldering tobacco, contaminants emitted during puffs and contaminants that diffuse through the cigarette paper and the mouth end of the cigarette between puffs.

<sup>7</sup>*Biological marker*: any parameter that can be used to measure an interaction between a biological system and an environmental agent, which may be chemical, physical or biological (WHO 1993).

<sup>8</sup>*DNA adducts*: combination of other molecules with DNA.

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The most direct measurement of exposure to ETS is by using biological markers (biomarkers), that is, analysis of physiological fluids of potentially exposed people for tobacco smoke constituents or their breakdown products (metabolites). Cotinine, which is a breakdown product of nicotine, is the most widely used and probably the most reliable estimate of recent exposure to ETS. Other approaches include measurement of a tobacco-specific carcinogen and measurements of both protein and DNA adducts.

ETS exposure may also be estimated by means of questionnaires, usually through ascertainment of the smoking status of the spouse or by estimation of the number of hours a person is exposed at home, work, or elsewhere. Exposure levels may be under or over-estimated, with under-estimation generally considered to be more of a problem as individuals are often unaware of their ETS exposure, particularly outside the home.

### **Toxicological and pharmacological effects of ETS constituents**

ETS has many toxicological and pharmacological properties that can potentially result in adverse health effects. Chemicals known to be present in ETS can (a) be cancer-causing, (b) be irritating to the eyes and to the lungs, resulting in respiratory problems, (c) cause reproductive problems or harm the unborn child, and (d) have effects on the heart and blood vessels (cardiovascular system). Over 50 chemicals that are known or are suspected to cause cancer in humans have been identified in ETS. The presence of these chemicals in the air of a workplace means that if ETS is listed as an occupational carcinogen, the requirements of the Safety, Health and Welfare at Work (Carcinogens) Regulations should apply.

### **Epidemiological assessment of disease risks**

The epidemiological assessments of the health effects of ETS, on which the international position statements described above have been based, are outlined below.

Epidemiological studies have shown that ETS has effects on health similar to those seen from active smoking albeit at lower levels, namely (a) an increased risk of lung cancer (possibly increased by 20-30%), (b) an increased risk of heart disease (estimated at 25-30%), (c) an increased risk of stroke (possibly as high as 82%), (d) a reduction in birth weight of infants born to mothers exposed to ETS and (e) an increased frequency of chronic respiratory symptoms such as cough, phlegm production, shortness of breath and chest colds.

### **Health effects of occupational exposure to ETS**

The evidence is highly persuasive of a causal relationship between ETS in the workplace and lung cancer. There is a positive exposure-response relationship and the findings are biologically plausible and consistent across studies of varying designs. It has been estimated that working with smoking co-workers increases the risk of lung cancer by between 20-30% in non-smokers.

Most of the evidence linking heart disease with ETS comes from studies of spousal smoking whereas studies of the relationship between ETS exposure in the workplace and cardiovascular disease are relatively sparse. However, there is no biologically plausible reason to believe that the hazards of ETS exposure that have been demonstrated in the home should not also apply to the workplace. The consistency of results across countries with different lifestyles and diets and the positive exposure-response relationships are suggestive of a causal relationship. There is every reason to suspect that the adverse effects could be higher, given that ETS exposure as indicated by cotinine levels is considerably higher in the workplace.

Bar staff and other hospitality workers are a unique risk group in that their workplaces constitute extreme ETS exposure settings. It has been shown that premises that restricted customer smoking to

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certain areas reduced average exposure of staff to ETS but exposure remained far higher than in smoke-free premises.

The review of the scientific evidence indicates that ETS exposure in the workplace is associated with increased risk of disease, including lung cancer and cardiovascular disease.

### **Control mechanisms for ETS**

There are two standard approaches to reduce exposure to ETS: ventilation and legislation. Research suggests that presently available ventilation technology (well-mixed dilution ventilation) is unsatisfactory for controlling worker exposure to ETS. Air cleaning is similarly problematic. Of proposed new technology, displacement ventilation is viewed as having the potential for a 90% reduction in ETS levels but even this would still leave exposure levels 1500 to 2500 times the acceptable risk level for hazardous air pollutants.

The EU Commission has called on member states to 'provide adequate protection from exposure to passive smoking at workplaces, in enclosed public places and in public transport and to strengthen smoking prevention programs'.

In Ireland ETS exposure in the workplace can be controlled using both public health legislation and health and safety legislation. The Public Health (Tobacco) Act 2002 will give the Minister for Health and Children powers to make regulations including the banning of smoking in any 'place of work'. It is implicit in all health and safety legislation that workers should be protected from risks to their health, and there is potential in general health and safety legislation, such as section 6 of the Safety, Health and Welfare at Work Act, 1989, and the Safety, Health and Welfare at Work (Carcinogens) Regulations, S.I. No. 078 of 2001, to identify ETS as a hazard and controllable risk from which workers must be protected. The Finnish and United States experience has clearly shown the advantages of using a legislative approach as opposed to voluntary codes on workplace smoking, which have thus far been the approach in Ireland and the UK.

### **Conclusions**

- ETS is carcinogenic and causes lung cancer and probably other cancers.
- ETS causes heart disease.
- ETS causes respiratory problems in adults and children.
- ETS has adverse effects on reproduction, including low birth weight.
- Where workplace smoking is permitted, employee exposure to ETS is likely to be higher and more sustained than in the home environment.
- Employees need to be protected from exposure to ETS at work.
- Current ventilation technology is ineffective at removing the risk of ETS to health.
- Legislative measures are required to protect workers from the adverse health effects of ETS exposure.
- Research is required to assess occupational exposures to ETS in Ireland and the resultant adverse health outcomes, especially in high-risk groups such as pregnant workers and workers in the hospitality industry.

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## 1. INTRODUCTION

Overall there has been a decrease in smoking prevalence in European countries over the last 20 years (World Health Organization 1997), although in some countries the consumption of cigarettes per person is increasing (European Network for Smoking Prevention 2001). There is an almost universal social class gradient of higher smoking rates in lower income groups. Previously men smoked more than women but this trend has now been reversed in some countries among the young.

The social class gradient is reflected in differences in smoking prevalence by occupation. For example, in Canada in 1998 there was a decline in smoking intensity among all workers except 'those in outdoor blue-collar occupations'. (Gaudette, Richardson et al. 1998). Between 1987 and 1990 in the US, occupational prevalence was 57.8% for roofers and 57.6% for crane and tower operators, and between 1985 and 1992, 40% of men employed as handlers/labourers or transportation/material movers were smokers (Metropolitan Insurance Company 1992). In Finland, where only 27% of men and 19% of women are active smokers, there are also differences between occupations, e.g. in restaurants 41% of working men are smokers as are 31% of working women, and 53% of bartenders are active smokers. (Personal communication Dr. Asko Aalto, Senior Ministerial Occupational Medical Adviser, Finland)

In Ireland, smoking rates among men vary from 25% in the highest social class groups to 38% in the lowest social class groups and from 26% to 38% respectively in women (National Health and Lifestyles Surveys, Friel, Nic Gabhainn, et al. 1999). In the early nineties, teachers and farmers had low smoking rates: between 17.6% and 20.5% for teachers (Wynne, Clarkin, et al. 1991) and 19% among farmers (World Health Organization 1997). The rate among nurses was 24.5% (Wynne, Clarkin, et al. 1992). The level of current smoking among Irish construction workers is of the order of 44% compared to the national average of 29% (Construction Workers Health Trust 2002).

Working in an environment where smoking is permitted, as well as encouraging active smoking, can lead to high levels of exposure to environmental tobacco smoke (ETS) deriving from smoking by both employees and by the general public visiting such workplaces. This is a particular problem in the entertainment industry e.g. pubs and nightclubs, where large numbers of patrons smoke.

As far back as 1986, the US Surgeon General proclaimed: "Passive smoking is a cause of disease, including lung cancer, in healthy non-smokers", (US Department of Health and Human Services 1986). Agreement with this view has been growing steadily since then (see section 2). Awareness of the harmful effects of ETS places an onus on governments to protect public health by providing legislation to protect the general public from passive involuntary smoking.

The focus of occupational health and safety legislation is to provide a safe work environment. ETS is now considered carcinogenic to humans (US National Toxicology Program 2000; International Agency for Research on Cancer 2002). Finland and Germany have listed ETS as a workplace carcinogen. Some countries or regions, such as Finland and California, have introduced stringent legislation to protect the general public and those in the workplace from ETS. Legislation in other countries is less comprehensive and involves voluntary codes of practice rather than legislation (Great Britain Department of Health 1998). In Ireland regulations have been introduced over the last 16 years to prohibit or restrict smoking in most public places (see section 6.2). Although these regulations provide protection for most workers, there are significant exemptions with restricted smoking allowed in restaurants, trains and psychiatric hospitals, and unlimited smoking allowed in bars, betting bookmakers, prisons and some other workplaces.

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As evidenced by recent court cases, protection of workers from ETS at their place of work is becoming an important occupational health issue. For example, in 1997 US flight attendants won a \$300 million settlement in a class action lawsuit on behalf of flight attendants harmed by ETS (Josefson 1997). In 2000 in The Netherlands, a court upheld a postal worker's complaint that her exposure to tobacco smoke in the workplace infringed her right to work in a smoke-free environment. In 2001 in Australia, a non-smoking barmaid was awarded US\$235,000 for cancer caused by working for 11 years in a smoky bar.

Given the increasing concern about the health effects of ETS, the Health and Safety Authority and the Office of Tobacco Control commissioned an independent scientific working group to **'identify and report on the degree of consensus that exists among leading international scientific authorities on the question of the hazard and risk posed by environmental tobacco smoke to human health in the workplace'**.

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## 2. SUMMARY OF INTERNATIONAL POSITION STATEMENTS ON THE HEALTH EFFECTS OF WORKPLACE ENVIRONMENTAL TOBACCO SMOKE (ETS)

Numerous position statements on the harmful health effects of ETS have now accumulated from government and scientific bodies worldwide. For example:

- 2002 **World Health Organization**, International Agency for Research on Cancer *Tobacco smoke and involuntary smoking (Summary)* IARC Monograph Vol. 83 (International Agency for Research on Cancer 2002)
- 2001 **European Network for Smoking Prevention** (ENSP) European Status Report, *Smoke free workplaces* (European Network for Smoking Prevention 2001)
- 2000 **US National Institute of Health (NIH)** National Toxicology Program *9th Report on Carcinogens* (US National Toxicology Program 2000)
- 2000 **American College of Occupational and Environmental Medicine** (ACOEM) position statement (American College of Occupational and Environmental Medicine (ACOEM) 2000)
- 2000 **World Health Organization** (WHO), *Air Quality Guidelines for Europe 2000* (World Health Organization 2000a)
- 1999 **Chartered Institute of Environmental Health** (CIEH) (UK Chartered Institute of Environmental Health (CIEH) Policy Unit 1999)
- 1999 **World Bank** *Curbing the Epidemic: Governments and the Economics of Tobacco Control* (World Bank 1999)
- 1999 **Institute of Global Tobacco Control**, Johns Hopkins School of Public Health Environmental tobacco smoke (Institute for Global Tobacco Control 1999)
- 1998 Report of the Scientific Committee on Tobacco and Health (SCOTH), UK (UK Scientific Committee on Tobacco and Health 1998)
- 1998 UK White Paper on Tobacco, *Smoking kills* (**Great Britain Department of Health** 1998)
- 1997 **California Environmental Protection Agency** (California EPA), *Smoking and Tobacco Control, Monograph 10 Health effects of exposure to environmental tobacco smoke* (California Environmental Protection Agency 1997)
- 1997 **Australian National Health and Medical Research Council**, Health effects of passive smoking (Australian National Health and Medical Research Council (NHMRC) 1997)
- 1994 **US Occupational Safety and Health Administration** (OSHA) *Indoor Air Quality* (Proposed rules) (US Occupational Safety and Health Administration (OSHA) 1994)
- 1992 **US Environmental Protection Agency** (EPA) (US Environmental Protection Agency (EPA) 1992)
- 1987 IARC Monograph Supplement 7 (**International Agency for Research on Cancer** 1987)
- 1986 **US Surgeon General** *The Health Consequences of Involuntary Smoking* (US Department of Health and Human Services 1986)

Summaries of the positions taken on ETS by each of these agencies are provided in the Appendix. The main collective findings are summarised below and in Table 1. Position statements of particular importance, in terms of depth, scientific quality and relevance to the workplace are: summary of the new monograph from IARC (2002), US NIH National Toxicology Program 9th Report on Carcinogens (2000), ENSP European Status Report (2001), UK SCOTH Report (1998), California EPA Monograph 10 (1997), and Australian National Health and Medical Research Council Report (1997).

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Most agencies have taken the position that ETS is likely to have the same health risk profile as that established for smoking, even where evidence is not yet conclusive for ETS. Over the past 16 years various authoritative bodies have declared that ETS is carcinogenic to humans. Considerable weight has been added to this judgement by recent definitive statements from two independent assessments of carcinogenic risks to humans:

- 9th Report on Carcinogens published in 2000 by the US National Institute of Health National Toxicology Program, and
- Summary of the new monograph on second-hand smoke (2002) from IARC (a branch of the World Health Organization).

On the basis of the evidence linking ETS with lung cancer, both these reports have declared ETS to be carcinogenic to humans. For some of the constituents of ETS, there is no evidence of a safe level of exposure. The IARC summary monograph confirming that ETS causes lung cancer is particularly important as IARC is a global public health agency.

All the statements listed above agree that ETS can cause lung cancer in non-smokers. Many also specify that there is compelling evidence that working with smoking co-workers increases the risk of lung cancer by between 20% and 30%.

Statements on the relationship with other cancers are less uniform. The California EPA monograph states that ETS causes nasal sinus cancer, while other groups maintain that ETS increases the risk of sinus cancer. The California EPA report finds that there is suggestive evidence of a causal association with cervical cancer.

Most groups state that ETS causes heart disease (e.g. WHO Air Quality Guidelines, California EPA, UK SCOTH), although some describe it less emphatically, as increasing the risk of heart disease. The IARC summary monograph reports that the available meta-analyses estimate that involuntary smoking increases the risk of an acute coronary heart disease event by 25-30%. Heart disease is the most common cause of death in Ireland and a major cause of premature death. Because heart disease is so common, much more common than lung cancer for example, the association of ETS with heart disease is of the utmost importance, from a public health perspective.

Most agencies agree that exposure of pregnant women to ETS causes lower birth weight and that children exposed to ETS are at increased risk of respiratory disease (lower respiratory tract infection, asthma, and chronic respiratory symptoms), and sudden infant death syndrome (SIDS). As this report is primarily concerned with ETS in the workplace, the primary focus is adults but it should be recognised that ETS impacts on children who spend time in adult workplaces e.g. crèche, school. The ENSP report states that ETS is especially harmful to pregnant women.

Many of the statements, including the California EPA and ENSP reports, cite ETS as causing various forms of respiratory disease in adults.

Not all the evidence underpinning the conclusions reached in these statements relates to workplace exposure per se. However most of the reports point out that exposure to ETS outside the home, for example in the workplace and in public places, would cause a similar burden of illness amongst adults, qualitatively and quantitatively, as exposure in the home. Most statements conclude that there is sufficient evidence for an adverse effect of ETS on health to recommend that policies and practices be introduced to reduce exposure to ETS generally and that, wherever possible, smoking should not be allowed in the workplace.

**TABLE 1**  
**Summary of position statements (+ + denotes causal link and + denotes suggestive of causal link)**

	Adults										Children					
	Carcinogenic	Lung cancer	Sinus cancer	Cervical cancer	Coronary heart disease	Respiratory disease	Lung function	Eye/nasal irritation	LBW /SGA	SIDS	LRTI/ asthma/ chronic RS					
IARC 2002	++	++			++	++	++		++							
ENSP 2001		++, also workplace			++, also workplace, also CBVD	pneumonia	dose response	++								
NIH 9th RoC 1999	++	++, also workplace			+											
ACOEEM 2000		++				++										
WHO 2000	++, no threshold signif health risk	++	++	Studies contradictory	++	asthma		++	++							
CIEH 1999																
World Bank 1999		++			increased risk											
Inst Global Tobacco Cntrl 1999		++			++	asthma										
SCOTH 1998		++			++					++						++
UK White Paper 1998		++				asthma exacerbation										
California EPA 1997		++	++	+	mortality & morbidity	++, & decr pulm functn		++	++	++	++					++
Aust NHMRC 1997		++			++			++		++						LRTI & asthma
US OSHA 1994		++														
US EPA 1992	++	++														++
IARC 1987	++	increased risk														
US Surgeon General 1986		++														+(Children of smoking parents)

SIDS sudden infant death syndrome/ LBW low birth weight/ SGA small for gestational age/ LRTI lower respiratory tract infection/CBVD cerebrovascular disease



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### **3. CHARACTERISTICS OF ETS**

#### **3.1 ETS Constituents**

ETS is a complex mixture of several thousand compounds. It contains many known or suspected human carcinogens and toxic agents (World Health Organization 2000a).

ETS is made up of exhaled mainstream smoke, sidestream smoke emitted from smouldering tobacco, contaminants emitted during the puffs and contaminants that diffuse through the cigarette paper and the mouth end of cigarettes between puffs. Emissions contain both particle phase and vapour phase contaminants. Sidestream smoke is the major component of ETS, contributing over half of the particulate matter and nearly all of the vapour phase. ETS, sidestream smoke and mainstream smoke are complex mixtures of over 4000 compounds. These include more than 50 known or suspected human carcinogens, such as 4-aminobiphenyl, 2-naphthylamine, benzene, nickel, and a variety of polycyclic aromatic hydrocarbons and *N*-nitrosamines. A number of irritants, such as ammonia, nitrogen oxides, sulphur dioxide and various aldehydes, and cardiovascular toxicants, such as carbon monoxide and nicotine are also present.

There are substantial similarities as well as differences between the mainstream smoke and sidestream smoke components of ETS (Guerin, Jenkins, et al. 1992; US Environmental Protection Agency (EPA) 1992). The main differences arise due to the differences between the temperature of combustion of the tobacco, pH, and degree of dilution with air. This dilution is accompanied by a corresponding rapid decrease in temperature. Mainstream smoke is generated at a higher temperature than sidestream smoke (mainstream smoke at approximately 800-900°C as against 600°C between puffs for sidestream smoke). Mainstream smoke has a lower pH (6.0-6.7) than sidestream smoke (6.7-7.5). Sidestream smoke being slightly alkaline contains more ammonia, is depleted of acids, contains greater quantities of organic bases, and contains less hydrogen cyanide than mainstream smoke. Differences in mainstream smoke and sidestream smoke are also ascribable to differences in the oxygen content (16% in mainstream smoke as against 2% in sidestream smoke). Because sidestream smoke is produced at lower temperatures and under more reducing conditions than mainstream smoke, many carcinogens and other toxicants are generated in greater amounts in sidestream smoke than in mainstream smoke. These quantitative differences are consistent with animal and genotoxicity studies, suggesting that sidestream smoke is more potent than mainstream smoke per unit of tobacco smoked (Wynder and Hoffman 1967; Claxton, Morin, et al. 1989).

After its production, sidestream smoke is rapidly diluted in air. This results in the sidestream smoke particle size distribution being smaller than for mainstream smoke. For example nicotine is predominantly in the particle phase in mainstream smoke but is found mainly in the gas phase in sidestream smoke. This shift to the gas phase is due to the rapid dilution in sidestream smoke. The particle size range for sidestream smoke is typically 0.01-1.0 µm while the mainstream smoke particle size range is typically 0.1-1.0 µm (Pritchard et al. 1988; Guerin, Jenkins, et al. 1992). These differences in size distributions for sidestream smoke and mainstream smoke particles, as well as the different breathing patterns of smokers and non-smokers, have implications for the deposition patterns of the particles in the various regions of the human respiratory tract. It has been estimated that about 47% to 90% of mainstream smoke particles are deposited in the respiratory tract while for sidestream smoke 10% deposition has been reported. In general submicron size particles (mainly found in sidestream smoke) are predominantly deposited by diffusion in the lower respiratory tract while above micron size particles may deposit preferentially in the upper respiratory tracts, both thoracic and extra thoracic (International Commission on Radiological Protection 1994). It should be noted that the risks arising from deposited ETS material in the respiratory tract are not simply proportional to the amount of material deposited. The site of deposition, presence of sensitive cells, solubility, clearance mechanisms and other physiological factors all have a major influence on the potential for risks to health.

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In terms of human exposure to ETS in a given enclosed or indoor space, there may be persons receiving individual exposures considerably greater than the average. For example in the drinks industry, behind the bar staff may receive a greater exposure to mainstream smoke and sidestream smoke due to their close proximity to smokers sitting at the bar than is being received by floor bar staff. An extreme example of such high exposure to ETS may arise in the case of an infant in the arms of a smoking parent.

In addition to the production of vapours and particulates, tobacco smoking causes significant emissions of carbon monoxide. Environmental tobacco smoke in dwellings, offices, vehicles and restaurants can raise the 8-hour average carbon monoxide concentration by up to 23-46 mg/m<sup>3</sup> (20-40 ppm) (US Environmental Protection Agency (EPA) 1991).

### **3.2 Measurement of ETS**

Assessment of exposure to ETS is essential in epidemiological investigations of the health impacts of ETS and also in evaluating the effectiveness of strategies to reduce exposure. Exposure can be assessed in a number of ways including indirect and direct methods such as:

- measurements of indoor air concentrations of ETS constituents
- personal monitors to detect ETS constituents
- biological markers of exposure in samples from exposed subjects
- surveys and questionnaires

There are advantages and disadvantages with each technique. These must be considered in interpreting results from studies.

#### **Measurements of indoor air concentrations of ETS constituents**

Exposure to ETS can be measured by various tracers: acrolein, aromatic hydrocarbons, CO, nicotine, oxides of nitrogen, nitrosamines, and inhalable particles (Maroni, Seifert, et al. 1995). Usually just one compound in ETS is monitored because it is neither practical nor possible to monitor the full range of compounds. According to the US National Academy of Sciences optimum tracers for ETS should have the following characteristics:

- Unique (or nearly so) to ETS. This is to ensure there is minimal contribution from other sources
- Detectable at low concentrations
- Similar emission rates among various tobacco products
- There should be a consistent ratio between the individual contaminant of interest and the composite pollutant, ETS, under a range of environmental conditions

While various ETS-related compounds can be measured above background levels in indoor environments (e.g. polycyclic aromatic hydrocarbons and carbon monoxide), most are not practical markers of ETS either because they have many sources in addition to tobacco smoke and/or because they are difficult or expensive to measure. The most widely used marker compounds for assessing the presence and concentration of ETS in indoor air are vapour-phase nicotine and respirable suspended particles mass. Respirable suspended particles are present in large quantities in ETS, and levels in indoor air are a useful marker for the particulate phase of ETS. Even under conditions of low smoking rates, easily measurable increases in respirable suspended particles have been recorded above background levels (Repace and Lowrey 1980). However, respirable suspended particles in indoor air are not unique to ETS, and background levels from other sources must be accounted for when using respirable suspended particles as a marker for ETS.

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While there is no single measure of ETS that meets all these criteria, and it is unlikely that any one measure can be representative of all the constituents of ETS, nicotine is unique to ETS and techniques for its measurement have recently been improved. Nicotine has the advantage of being specific to tobacco smoke and of being present in large quantities in ETS. A potential drawback is that it has a high affinity for interior surfaces and, under certain circumstances, measurements could lead to an underestimate of the levels of other ETS constituents. Similarly, nicotine can be later re-emitted from surfaces, after other ETS constituents have been removed. None the less, many studies have demonstrated that nicotine is a reliable marker of ETS levels and that it correlates well with other exposure indices, such as respirable suspended particles and the number of cigarettes smoked as reported in questionnaires (Leaderer and Hammond 1991).

(i) *Nicotine measurement*

There are several well-known problems with measuring nicotine in the air: (a) sampling and analysis are both difficult as it is highly reactive; (b) nicotine can be present in both the vapour and particulate phases; and (c) vapour or particulate phase nicotine can be re-emitted from surfaces on which it has deposited. While nicotine is a good marker for ETS exposure, the relationship between exposure to nicotine and to other components of ETS, which may have health consequences, has not been established. This has implications for the estimation of health effects of ETS based on nicotine exposure.

Two approaches are generally used for the determination of nicotine in indoor air, one for total nicotine and one for the vapour phase. Total nicotine is measured using a treated filter media, and methods which only measure vapour phase nicotine use a solid sorbent trap. There appears, however, to be little information available on the inter-comparison of the precision and accuracy among the various methods to measure nicotine.

Nicotine is generally specific to tobacco and therefore detectable levels can be attributed to tobacco smoke (with exceptions including areas where tobacco is processed). The highest concentrations of nicotine in indoor environments were found in bars and in smoking sections of airplanes (before smoking was banned in airplanes) with levels reaching as high as 50 to 75 microgram per cubic metre (US Environmental Protection Agency (EPA) 1992). In selected studies, using controlled and field conditions, the concentration of nicotine was found to increase as a function of the number of smokers present and the number of cigarettes consumed (US Environmental Protection Agency (EPA) 1992). Studies suggest that nicotine may disappear from the air faster than other ETS constituents, and therefore its use as a marker may underestimate the relative concentrations of other constituents.

(ii) *ETS-associated respirable suspended particles*

In contrast to nicotine, respirable suspended particles are not specific to ETS and therefore respirable suspended particle measurements in environments where smoking occurs must be compared to respirable suspended particle concentrations in comparable environments where smoking does not occur. Studies where smoking does occur consistently show higher respirable suspended particle concentrations compared to environments where smoking does not occur (US Environmental Protection Agency (EPA) 1992).

(iii) *Carbon monoxide measurement*

Usually carbon monoxide detection requires the use of a non-dispersive infra-red analyzer. The infra-red radiation passes through two parallel optical cells, one with the air sample, the second with carbon monoxide-free air as reference: the difference in absorbance relates to carbon monoxide concentration. The measured absorption is made at one or more of the characteristic wavelengths of the infra-red spectrum of carbon monoxide.

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Other carbon monoxide measuring instruments use gas filter correlation techniques and make use of optical filters to limit photometer sensitivity to the infra-red band of interest. In gas filter correlation instruments the infra-red radiation passes through a spinning filter wheel containing a sealed carbon monoxide reference cell and a reference cell filled with nitrogen. A more specific and sensitive carbon monoxide detector, the photoacoustic-infra-red detector, has been developed in recent years. Another type of carbon monoxide detector is based on electrochemical oxidation: an air sample is passed through an electrochemical cell where the oxidation of carbon monoxide to carbon dioxide produces a signal proportional to carbon monoxide concentration.

There are many instruments commercially available, based on the reported principles: they are reliable and sensitive, can provide a continuous record of the carbon monoxide concentrations, and many of them can be hand-held for personal monitoring. Miniaturized carbon monoxide monitoring devices are also available which usually operate on the principle of the electrochemical or catalytic oxidation of carbon monoxide (Yocom and McCarthy 1991).

(iv) *Other constituents*

Other studies have investigated specific constituents of ETS including benzene, benzo[a]pyrene, polycyclic aromatic hydrocarbons, formaldehyde, toluene and N-nitrosamines (Guerin, Jenkins, et al. 1992; US Environmental Protection Agency (EPA) 1992). Because sources other than ETS exist for many of these constituents, studies have to be very carefully controlled with comparable environments where no smoking occurs.

### **Personal monitors**

Personal monitors have also been used to assess exposure to ETS and should theoretically provide a more accurate assessment of an individual's exposure to ETS. Studies with personal monitors for nicotine have shown that the average personal exposure associated with specific environments in the US ranged from 4.7 to 20.4 microgram per cubic metre (Guerin, Jenkins, et al. 1992; US Environmental Protection Agency (EPA) 1992). Jenkins, Palausky, et al. (1996) demonstrated that the mean 24-hour weighted average nicotine concentration for those exposed to ETS at work and away from work (3.27 microgram per cubic metre) was higher than for those only exposed to ETS away from work (1.41 microgram per cubic metre). The mean nicotine concentration measured by personal monitoring for those indicating no exposure to ETS was 0.05 microgram per cubic metre.

### **Biological markers of exposure**

Exposure to ETS can be measured directly by analysis of physiological fluids (blood including plasma, urine or saliva) for tobacco smoke constituents or their metabolites known as biomarkers. Ideally the biomarker should be specific to tobacco. Some of the biomarkers give an indication of exposure only while others may also provide an indication of toxicity.

Biological markers of ETS include (a) nicotine and cotinine (b) thiocyanate and carboxyhaemoglobin (c) metabolites of a tobacco-specific carcinogen, namely 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) and (d) biomarkers of genotoxicity namely protein and DNA adducts.

The relationship between these biomarkers and exposure to ETS is complex and varies as a function of both environmental and physiological factors. The degree of exposure is a function of the time an individual spends in each setting and the air concentration of the tobacco-related constituents in that environment. For a given air concentration, several factors will affect an individual's intake including gender, age, weight, and activity level (inhalation rate) at the time of exposure. In addition, individual differences in uptake, distribution, metabolism and excretion will affect the biomarker concentration in physiological fluids. These differences may be genetically determined and the genomic revolution

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will lead to further developments in this area.

(i) *Nicotine and cotinine*

Nicotine concentrations in blood, saliva and urine have been measured as indices of exposure to ETS. While low levels of nicotine are found in tea and some other plants, in general the levels of nicotine present in food have not been found to significantly impact the levels resulting from exposure to nicotine from tobacco (California Environmental Protection Agency 1997). However, nicotine has a short biological half life of approximately 2 hours. Therefore plasma, saliva or urinary nicotine is only a good indicator of exposure occurring within the previous few hours. Hair nicotine may be a more sensitive biomarker of longer-term exposure (Al-Delaimy, Crane, et al. 2002).

The most widely used biomarker of ETS is cotinine. Cotinine is a major metabolite of nicotine. Cotinine is specific to tobacco and can be measured in saliva, blood or urine either by gas chromatography, mass spectrometry or radioimmunoassay. Saliva levels correlate well with blood levels. The plasma half life in adult humans is approximately 15 hours (Zevin, Jacob, et al. 2000). This makes it a good indicator of exposure over the previous two to three days. The half life of cotinine in infants and children is much longer, in the order of 40 to 60 hours.

A comparison of mean concentrations of nicotine and cotinine in the saliva of plasma and urine of ETS-exposed volunteers is shown in Appendix Table A.1. This table shows that nicotine has a short half life in plasma and saliva and therefore cotinine is a more reliable marker of exposure for a number of hours after exposure to ETS. A comparison of biomarkers in unexposed and ETS-exposed non-smokers and also in active smokers is shown in Appendix Table A.2. This table shows that plasma and saliva cotinine levels are a very good index of exposure to ETS. Thiocyanate was not a good indicator.

Many population studies have demonstrated the association of cotinine levels in saliva, serum and urine with reported exposure to ETS, including a number of European studies (Wald, Boreham, et al. 1984; Jarvis, McNeill, et al. 1991; Tunstall-Pedoe, Woodward, et al. 1991). A ten country study conducted by IARC collected data for non-smoking women from thirteen cities including five in Europe (Riboli, Preston-Martin, et al. 1990). For all thirteen centres combined, regression analysis generated the following comparisons:

**TABLE 2**  
**Mean urinary cotinine levels (ng/mg creatinine) from ETS**

Exposure Setting	Cotinine/creatinine levels (ng/mg)
Exposure from husband's smoking	6.2
Exposure from workplace smoking	2.4
Exposure from husband and workplace smoking	9.0
Exposure from smoking in public places	3.1

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A study (Trout, Decker, et al. 1998) of casino workers in the US demonstrated that serum cotinine levels were sensitive enough to pick up differences between pre-shift (1.34 ng/ml) and post-shift (1.85 ng/ml).

A recent study in New Zealand (Bates, Fawcett, et al. 2002) of exposure of hospitality workers to ETS demonstrated that non-smoking hospitality workers in premises allowing smoking by customers had significantly greater increases in salivary cotinine over the course of their work shift than workers in smoke-free premises.

(ii) *Thiocyanate and carboxyhaemoglobin*

While thiocyanate has been used to distinguish between smokers and non-smokers it is not very useful as a biomarker of ETS and has not been widely used as such. Although carbon monoxide and carboxyhaemoglobin have been used to distinguish between smokers and non-smokers, they are generally not good indicators of ETS exposure because of their lack of sensitivity and specificity.

(iii) *Tobacco-specific carcinogen*

The use of a tobacco specific carcinogen 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) and its metabolite 4-(methylnitrosoamino)-1-(3-pyridyl)-1-butanol (NNAL) may prove very useful in both monitoring exposure and providing a possible index of potential toxicity.

NNK is formed by the oxidation and nitrosation of nicotine during the curing and smoking of tobacco (International Agency for Research on Cancer 1985). NNK and its metabolite NNAL are potent lung carcinogens in rodents (Castonguay, Lin, et al. 1983; Rivenson, Hoffmann, et al. 1998).

A recent study (Hecht, Ye, et al. 2001) demonstrated that levels of NNAL and cotinine were significantly higher when exposure to ETS was reported compared to when no exposure to ETS was reported.

(iv) *Protein and DNA adducts*

Protein and DNA adducts are both markers of exposure and of a biochemical and possibly toxic effect. Protein adducts can serve as surrogates for DNA adducts. One of the most common protein adducts measured is the haemoglobin adduct of 4-aminobiphenyl. Tobacco smoke is the primary source of environmental 4-aminobiphenyl. Because of the relatively long half life of these adducts, their levels reflect exposure over the previous four months (California Environmental Protection Agency 1997).

Another group of protein adducts which have been measured are albumin adducts of polycyclic aromatic hydrocarbons. Multiple polycyclic aromatic hydrocarbons are present in tobacco smoke. Crawford, Mayer, et al. (1994) demonstrated a significant correlation between polycyclic aromatic hydrocarbon-albumin and cotinine against total ETS exposure in non-smoking mothers.

DNA adducts of tobacco specific constituents can also be measured. A recent study (Georgiadis, Topinka, et al. 2001) demonstrated correlations between declared times of exposure to ETS, plasma cotinine levels and bulky DNA adducts.

Individual variability and enhanced susceptibility is an issue not only for bio-monitoring but also for possible toxic and health effects following exposure to ETS. Individual variability and enhanced susceptibility to ETS will come more into focus with the impact and availability of genomic technology. The impact of the information from the human genome project coupled with emerging technologies to detect individual variations in DNA sequences and genes known as polymorphisms has the potential to be able to detect individual sensitivities to ETS. Nicotine is metabolised to cotinine by CYP2A6 enzyme which shows polymorphisms. These polymorphisms have implications for the plasma half lives of nicotine and of cotinine, and for smoking addiction and possible carcinogenesis (Tyndale and Sellers 2002). Likewise, glucuronidation of nicotine and cotinine also shows polymorphisms and will have

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implications for interpretation of detection levels and consequences of exposure to ETS (Benowitz, Perez-Stable, et al. 1999).

### **Questionnaires**

ETS exposure is usually estimated by ascertainment of whether a spouse smokes, or estimation of the number of hours a person is exposed at home, work, or elsewhere. Qualitative information obtained from questionnaires is generally reliable but quantitative information is less so (California Environmental Protection Agency 1997).

Exposure levels may be under or over-estimated, with under-estimation generally considered to be more of a problem. Individuals are often unaware of their ETS exposure, particularly outside the home. In studies using both self-reporting and biological markers, the exposure prevalence was higher when determined using biological markers (California Environmental Protection Agency 1997). On the other hand, smokers may not declare their smoking. However, studies have shown that few smokers do this (Lam, Ho, et al. 2000; Jarvis 2001) and that sensitivity and specificity for self-report are close to 90% (Lam, Ho, et al. 2000).

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## 4. HEALTH EFFECTS OF ETS

### 4.1 Toxicological and Pharmacological Effects of ETS Constituents

The many chemicals contained in ETS, both sidestream smoke and mainstream smoke, can be divided into four main groups with respect to their toxicological and pharmacological properties:

- carcinogenic and/or mutagenic chemicals,
- irritant chemicals,
- chemicals having a possible effect on human reproduction (reproductive toxicants),
- chemicals with other toxicological or pharmacological effects, including agents having an effect on normal body (physiological) functions such as lung function and the cardiovascular system.

#### Carcinogenic and/or mutagenic chemicals

Over 50 known or suspected human carcinogens have been identified in mainstream tobacco smoke, including for example benzene, nickel, 2-naphthylamine, 4-aminobiphenyl, formaldehyde, various N-nitrosamines and polycyclic aromatic hydrocarbons. The presence of nine of the 44 chemical agents classified by IARC as known human (Group I) carcinogens has been reported, namely benzene, cadmium, arsenic, nickel, chromium, 2-naphthylamine, vinyl chloride, 4-aminobiphenyl and beryllium (Smith, Livingston, et al. 1999). As indicated in section 3.1, because sidestream smoke is produced at lower temperatures and under more reducing conditions than mainstream smoke, many carcinogens are generated in greater amounts in sidestream smoke than in mainstream smoke, and hence are present in ETS. ETS is the only indoor source of some of these carcinogens, and particularly of the 'tobacco-specific nitrosamines' discussed below. Appendix Table A.3 provides a non-exclusive listing of the carcinogens reported in tobacco smoke, with their carcinogenic status in the EU system, their IARC classification and their US EPA status. IARC (International Agency for Research on Cancer 1986) has declared that there is *sufficient evidence* to categorise tobacco smoke as carcinogenic to humans, and tobacco smoke is listed as a carcinogen under California's Proposition 65.

Tobacco-specific nitrosamines are formed during the curing (drying) of the tobacco leaf and during combustion while smoking. N-Nitrosamines identified in tobacco smoke include N-nitrosodimethylamine, N-nitrosodiethanolamine and the tobacco-specific N-nitrosornicotine and N-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), formed by N-nitrosation of nicotine and other pyridine alkaloids. Most of the identified nitrosamines are carcinogens in experimental animals and some (e.g. N-nitrosodimethylamine) are present in sidestream smoke in amounts 10 to 200 times greater than in mainstream smoke (US Department of Health and Human Services 1986; Lofroth 1989). By weight, the tobacco-specific nitrosamines are the most prominent of the suspected carcinogens identified thus far in cigarette smoke (International Agency for Research on Cancer 1986). In addition to the nitrosamines, over 35 polycyclic aromatic hydrocarbons have been identified in tobacco smoke (International Agency for Research on Cancer 1986), several of which are proven carcinogens (e.g. benz[a]anthracene, benzo[a]pyrene, and dibenz[a,h]anthracene), while others have not been tested. Tobacco also contains a number of carcinogenic metals, derived from soil, fertilizers, agricultural sprays, and polluted rainfall, and also naturally occurring radionuclides, e.g. polonium-210 (Cohen, Eisenbud, et al. 1980).

Although many toxicological studies have confirmed the carcinogenicity of mainstream tobacco smoke (International Agency for Research on Cancer 1986), studies on ETS are fewer in number. A long-term chronic bioassay on ETS has not been carried out. The recent work of Witschi and co-workers (Witschi, Espiritu, et al. 1997; Bogen and Witschi 2002) has however shown induction of benign and malignant lung tumours in mice exposed to high levels of ETS, and short term inhalation studies of sidestream smoke in rats have shown pathological changes in the nose and larynx (Coggins, Ayres, et al. 1993; Haussmann, Anskeit, et al. 1998). The carcinogenic potential of ETS is also suggested



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by the finding that the semi-volatile and particle-bound organic fractions of sidestream smoke are mutagenic in bacterial systems (World Health Organization 2000a). These findings parallel the results for mainstream smoke and tobacco smoke condensate in a variety of short-term tests for genetic endpoints as reviewed by IARC (International Agency for Research on Cancer 1986). In addition, many of the individual constituents of ETS are positive in short-term tests (Claxton, Morin, et al. 1989). Finally, it has been reported that the urine of individuals exposed to high levels of ETS has mutagenic activity in short-term tests, in parallel to findings in smokers (Bos, Theuws, et al. 1983; Husgafvel-Pursiainen, Sorsa, et al. 1987; Mohtashamipur, Muller, et al. 1987).

Many of the known or suspected carcinogens listed in Appendix Table A.3 are so-called genotoxic or non-threshold carcinogens, and this is supported by the results of mutagenicity tests, as discussed above. It is generally accepted that there is no safe level of exposure for genotoxic carcinogens, which have also been described as 'one-shot carcinogens'. A relationship between the presence of such carcinogens in ETS and the epidemiological evidence suggesting an association between development of lung cancer in non-smokers and exposure to ETS in the workplace and/or at home is therefore plausible. ETS is included in the National Toxicology Program 9th list of known human carcinogens (US National Toxicology Program 2000) and IARC has recently decided that there is *sufficient evidence* that passive smoking causes lung cancer in humans (International Agency for Research on Cancer 2002).

The presence of any category 1 or category 2 mutagen or carcinogen in workplace air makes that workplace subject to the requirements of S.I. No. 078 of 2001, the Safety, Health and Welfare at Work (Carcinogens) Regulations, with their core requirements of elimination or substitution, or if not possible, introduction of engineering controls to reduce levels of the carcinogen to as low as technically feasible, together with other measures to protect workers' health. Given that ETS contains such carcinogens and mutagens, the presence of ETS in the air of a workplace should make that workplace subject to the Regulations if ETS is specifically listed as an occupational carcinogen, as it is in the legislation of Finland and Germany.

### **Irritant chemicals**

Irritation of the eyes, nose, and respiratory tract is the most common and best-established adverse health effect associated with exposure to ETS, and it has been reported that approximately 30% of individuals experience eye irritation at levels of ETS-derived carbon monoxide of 2.5 ppm over background (Muramatsu, Weber, et al. 1983). Known irritant constituents of tobacco smoke include acrolein, acrylonitrile, ammonia, crotonaldehyde, formaldehyde, nitrogen oxides, propionic and acetic acids, phenol and sulphur dioxide. Sidestream smoke and several of its components including acrolein, crotonaldehyde, formaldehyde and hydrogen cyanide are reported to affect mucociliary function (Bascom, Kesavanathan, et al. 1995; World Health Organization 2000a) and at a sufficiently high concentration can inhibit clearance of smoke particles from the lung (Battista 1976). Decreased tidal volume has been demonstrated in human volunteers exposed to propionic acid (Kendal-Reed, Walker, et al. 1996), while Walker, Kendal-Reed, et al. (1996) have showed marked irritant effects of nicotine on the nose and on olfactory sensation. There is therefore evidence for a causal relationship between exposure to constituents of ETS and the reported increase in respiratory disorders including emphysema in non-smoking adults and in children.

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## **Reproductive toxicants**

ETS has been shown to contain a number of known developmental toxicants, such as carbon monoxide, carbon disulphide, nicotine, cadmium, lead and toluene (California Environmental Protection Agency 1997). Five of these six are classified as reproductive toxicants in the EU (see Appendix Table A.3), the exception being nicotine. Lead and carbon disulphide also have effects on male and female fertility. It should be noted that many of the over 50 chemicals identified in ETS as having carcinogenic effects have not been tested for reproductive toxicity. The polycyclic aromatic hydrocarbons have however been found to cause developmental and reproductive effects in experimental animals. Exposure to tobacco smoke due to active smoking is regarded as a developmental toxicant, and also as having effects on female and male fertility, by a number of international bodies including the California EPA (California Environmental Protection Agency 1997). In relation to ETS, the conclusion of the California EPA was that epidemiological evidence supports a link between ETS exposure and adverse effects on foetal growth, but that it was not possible to show an association between ETS exposure and birth defects, changes in female fertility or with male reproductive dysfunction.

The Safety, Health and Welfare at Work (Pregnant Employees etc.) Regulations, S.I. No. 218 of 2000, may be considered to apply in any workplace where pregnant workers may be exposed to ETS, reflecting the developmentally toxic constituents present. These Regulations require that an assessment of the risk is carried out in relation to exposure of any pregnant worker to ETS, in order to protect the health of both the mother and her unborn child. The developmental toxicants present in ETS fall within the definition of agents listed in Part A of Schedule 2 of those Regulations.

## **Chemicals with other toxicological or pharmacological effects, including agents having an effect on normal functions such as lung function and blood pressure.**

Many toxic agents such as carbon monoxide, nitrogen oxides, ammonia, and hydrogen cyanide are found in tobacco smoke. Carbon monoxide, in addition to its reprotoxic effects, binds directly to haemoglobin, forming carboxyhaemoglobin and thus decreasing the oxygen-carrying capacity of the blood, which in turn can result in adverse health effects. Nicotine, which is one of the principal active chemicals in tobacco, has diverse pharmacologic and toxicological actions, ranging from acute poisoning to chronic effects, and also has direct effects on the cardiovascular system. Many toxicological and pharmacological studies of ETS and its components have been carried out in experimental animals. This report focuses, however, on the evidence for adverse effects in humans and is not intended to provide an extensive review of the animal studies. Extensive research has also been carried out into the relationship between ETS exposure and adverse effects in children including asthma, but this aspect is outside the scope of this report, which primarily focuses on effects of ETS on adult humans.

In relation to findings of physiological, toxicological or pharmacological effects on the respiratory system in humans (other than irritation, which is discussed above) which would correlate with the apparent increased risk of respiratory disease, a number of inhalation chamber studies with ETS (Menon, Rando, et al. 1992; Jindal, Gupta, et al. 1994) have shown decreases in lung function indices and increased airway responsiveness in non-smoking asthmatics exposed to ETS, compared with non-exposed asthmatics. In relation to healthy adults, a range of studies (White and Froeb 1980; Brunekreef, Fischer, et al. 1985; Kauffman, Dockery, et al. 1989; Masjedi, Kazemi, et al. 1990; Carey, Cook, et al. 1999; Chen, Tunstall-Pedoe, et al. 2001; Smith, Bombick, et al. 2001) have shown adverse effects on lung function in non-smoking individuals exposed to ETS at home or at work. However, other investigators (Kentner, Triebig, et al. 1984; Jaakkola, Jaakkola, et al. 1995; Kunzli, Schwartz, et al. 2000) have shown no such effects.

In addition to the other recognised or postulated health effects of carcinogenicity and irritancy,

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discussed above, particular attention has been paid to the presence of chemicals such as carbon monoxide and nitric oxide in ETS that could have an effect on the cardiovascular system. Subchronic and acute exposures to tobacco smoke and various tobacco smoke constituents have been shown to elicit a wide variety of cardiovascular effects in several animal species, including promotion of atherosclerosis, activation of platelets and white blood cells, and exacerbation of ischaemia/reperfusion injury (California Environmental Protection Agency 1997; World Health Organization 2000a).

A significant body of research has indicated that the known effects of tobacco smoke on the cardiovascular system of smokers may in part be mediated by its effects on the vascular endothelium. A similar effect of ETS on endothelial function of the coronary circulation in healthy non-smokers was suggested by the work of Celermajer, Adams, et al. (1996). Similarly, Raitakari, Adams, et al. (1999) showed arterial endothelial dysfunction in 20 non-smoking passive smokers, and Sumida, Watanabe, et al. (1998) showed an impairment of acetylcholine-induced coronary artery dilation in 19 non-smoking passive smokers compared with non-exposed non-smokers. Otsuka, Watanabe, et al. (2001) found that basal coronary flow velocity in healthy non-smokers was unaffected by ETS, but acute exposure to ETS significantly reduced coronary flow velocity reserve, a finding which the authors suggested could be due to an effect of ETS on the endothelium of the coronary circulation. A significant relationship has been demonstrated between ETS exposure and carotid wall thickening (Howard, Burke, et al. 1994; Diez-Roux, Nieto, et al. 1995), which is also indicative of an effect of ETS on the vascular system. A causal link between ETS and heart disease is therefore biologically plausible.

## **4.2 Epidemiological Assessment of Disease Risk**

In biological and biomedical scientific research it is difficult to prove absolutely that a particular agent causes a disease. Scientific advances are made in these areas through the gradual accumulation of evidence pointing towards a causal role for the agent.

In experimental animal research and in some situations in clinical medicine, for example testing the efficacy of a new drug, it is possible to carry out clinical 'experiments' comparing groups receiving different treatments. However, in epidemiological research requiring large populations for the evaluation of potentially harmful exposures, alternative approaches are needed. For example, to 'prove' that ETS causes cancer or heart disease would require the conduct of long-term experiments (randomised controlled trials) involving hundreds of thousands of individuals half of whom would be randomly assigned to long term ETS exposure and the other half assigned to non-exposure. But because it is not ethical to expose human subjects to a potentially harmful substance (in this case ETS), the only research approaches possible are those based on observational studies of non-smokers. Either disease rates in individuals exposed to ETS at home or at work are compared with rates in individuals not so exposed (cohort study); or past ETS exposures are compared in cases (those with the diseases in question e.g. heart disease or lung cancer), and in those without these conditions (controls) (case control study). There is no certainty in either type of study that the two groups being compared are similar with respect to other relevant variables. Thus there is the possibility that any differences observed between the groups could be due to factors other than the ETS exposure. If such factors also affect the risk of disease, they are referred to as confounding variables. The consequence is that part or all of the observed association between ETS and disease may be spurious.

Although there are methodological and statistical techniques to minimise confounding and other biases, the judgement as to whether the links observed are causal or not remains difficult. The criteria for causality proposed by Bradford-Hill are usually used to guide the evaluation of a body of evidence as to whether or not an association between an outcome (e.g. lung cancer) and a (putative) risk factor (in this instance, ETS) is causal (see Table 3). The more criteria met, the more likely that the link is causal. But the judgement remains subjective and 'proof' is elusive.

For example, using these criteria, most people eventually accepted that smoking causes lung cancer. Yet because there was no experimental 'proof', in spite of the ever-stronger evidence, some individuals

continued to claim that the association was indirect rather than causal. Another example is the evaluation of the link between infection with *Helicobacter pylori* and peptic ulcers. Initially, many considered this link to be an artefact because there was, at that time, no biological rationale for an infectious aetiology for peptic ulcers. Large-scale trials involving random assignment of the infectious agent were obviously not possible for ethical reasons, but over time, epidemiologic and laboratory evidence accumulated until the point was reached where it became accepted that *H. pylori* infections cause the majority of peptic ulcers.

This section of the report (4.2) summarises the evidence relating to the longer term effects of exposure to ETS in general, and section 5 focuses on the workplace in particular. Specific reference to the criteria for causality is made for the various outcomes considered. Because there cannot be experimental proof, debate is likely to continue around the long-term effects of ETS even when most of the criteria are satisfied, as shown in Table 3 for lung cancer. Public health decisions often need to be based on this type of circumstantial evidence because the balance of probabilities is that failing to act on the evidence will cause unnecessary harm. Under the precautionary principle, where there is risk of harmful exposure, the presence of uncertainty should not be used as a reason for postponing cost-effective measures to prevent such exposure (World Health Organization 2000b).

**TABLE 3**  
**Summary of evidence regarding ETS and lung cancer using the Bradford-Hill criteria**  
(adapted from COC-UK (UK Committee on the Carcinogenicity of Chemicals 1998))

Criterion	Evidence regarding passive smoking	Comments
Experiment	Limited	Possible only in animals. Limited evidence that sidestream smoke is carcinogenic in animals. Sufficient evidence that mainstream smoke is carcinogenic in animals. No adequate study with ETS.
Strength	No	Only a weak effect would be predicted from the nature of exposure. Most studies and meta-analyses yield overall relative risks between 1.1 and 1.3.
Dose response	Yes	
Temporality	Yes	Where exposure was assessed prior to diagnosis of lung cancer, the results of retrospective studies are consistent with prospective studies in showing that ETS exposure preceded onset of lung cancer.
Consistency	Yes	Most of the published studies and meta-analyses show consistent evidence of a small increase in relative risk.
Specificity	No	Active smoking is also non-specific in its effects.
Plausibility	Yes	Evidence of exposure of the lung to genotoxic carcinogens present in ETS, data from published reports of increased carcinogen-protein adducts in passive smokers and the linear nature of the dose response for lung cancer in active smokers, indicate it is plausible that ETS induces lung cancer.
Coherence	Yes	Epidemiological findings, biological monitoring studies for exposure using cotinine, and evidence from protein adduct investigations are coherent.
Analogy	Yes	An association between passive smoking and lung cancer is consistent with the known causal association for active smoking and lung cancer.

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Epidemiological studies have shown that ETS has effects on health similar to those seen from active smoking; that it is a human carcinogen; and that passive smoking increases the risk of illnesses such as lung cancer, heart disease and respiratory disease. There is now widespread agreement about these effects among the various national and international scientific and regulatory bodies (see section 2). Relevant epidemiological studies on which these international position statements are based are outlined in this section and in section 5.

### **Lung cancer**

The principal epidemiological evidence that ETS increases the risk of lung cancer in non-smokers comes from studies of non-smoking women married to smokers where the number of cigarettes smoked per day by the husband is a common surrogate for ETS exposure. The risk of lung cancer from passive smoking rises with the number of years of exposure and with the strength of the exposure (Hackshaw, Law, et al. 1997). It has been estimated from meta-analyses that, after controlling for potential sources of bias and confounding, the excess risk is of the order of 20% for women and 30% for men (International Agency for Research on Cancer 2002).

The US Environmental Protection Agency (US Environmental Protection Agency (EPA) 1992) has declared ETS to be a known human lung carcinogen based on the total weight of evidence namely:

- Similarities in chemical composition between ETS and exhaled mainstream smoke, including over 50 known or suspected human carcinogens
- The known lung carcinogenicity of mainstream smoke, with clear exposure-response relationships down to low levels and no evidence of a threshold
- Supporting evidence from animal bioassays and genotoxicity tests
- Measured exposure to, and bodily uptake of ETS constituent
- The consistent exposure-related increases in risk seen in many epidemiological studies from different countries using different designs.

See also Table 3.

### **Cardiovascular disease**

There are several plausible mechanisms by which ETS exposure can increase the risk of heart disease. The major biological and physiological effects include decreased oxygen-carrying capacity resulting in decreased exercise ability and, potentially, ischaemia, as well as increased platelet activation, endothelial damage, altered lipoprotein levels and increased arterial wall thickness, which can promote atherosclerosis and, in the case of platelet activation, thrombosis. Ischaemia, atherosclerosis and thrombosis increase the risk of myocardial infarction and other serious cardiovascular effects.

Unlike lung cancer the primary cause of which is smoking, there are many risk factors for heart disease other than smoking. For example, the diet of smokers and of non-smokers who live with them is 'worse' (lower consumption of fruit and vegetables) than that of non-smokers who live with non-smokers (Law, Morris, et al. 1997). This complicates the assessment of causality. Epidemiological studies of ETS and heart disease morbidity and mortality in non-smokers have varied with respect to the disease outcome (e.g. fatal and/or non fatal events), gender and surrogate measures for ETS exposure (e.g. spousal smoking, cohabitant smoking, work exposure). Nevertheless over the last ten years the evidence linking ETS exposure from spousal smoking with increased risk of heart disease has become increasingly indicative of a causal relationship (Law, Morris, et al. 1997; He, Vupputuri, et al. 1999). It has also been shown that non-smokers with high serum cotinine levels have a higher risk of coronary heart disease (Tunstall-Pedoe, Brown, et al. 1995). Passive smoking has been estimated to increase the risk of an acute coronary heart disease event by 25-30% (International Agency for Research on Cancer 2002).

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### **Cerebrovascular disease**

A recent New Zealand population based study investigated the relationship between passive smoking and the risk of stroke (Bonita, Duncan, et al. 1999). After adjusting for known risk factors for stroke, such as high blood pressure, diabetes and heart disease the study provided compelling evidence that passive smoking could, in a similar way to active smoking, increase a non-smoker's risk of cerebrovascular disease by a factor of almost two.

### **Low birth weight**

It is well established that maternal smoking during pregnancy is causally associated with low birth weight, in an exposure-related manner (US Department of Health and Human Services 1990). With respect to exposure to ETS in non-smoking mothers, the weight of evidence provided by the consistent results of numerous studies of ETS and birth weight from a variety of countries (Haddow, Knight, et al. 1988; Eskenazi, Prehn, et al. 1995; Rebagliato, du V. Florey, et al. 1995), the finding of ETS constituents in the urine of newborn infants of non-smoking mothers (Jordanov 1990), and the established causal association between active maternal smoking and reduced birth weight (US Department of Health and Human Services 1990) has led to the conclusion that ETS exposure in non-smoking women during pregnancy causes a small reduction in birth weight. Reductions in birth weight are causally related to adverse health outcomes.

### **Respiratory health effects**

There is strong epidemiological evidence from a number of studies that adult non-smokers exposed to ETS may experience reductions in lung function and an increased frequency of chronic respiratory symptoms, (White, Froeb, et al. 1991; Lam, Ho, et al. 2000). Passive smokers have reported significantly more cough, greater phlegm production, more shortness of breath, greater eye irritation and more chest colds than non-exposed non-smokers.

### **Attributable risk: lung cancer and cardiovascular disease**

Measures such as population attributable risk provide a guide to the size of a particular environmental problem. While attributable risk estimates are not precise predictions, they do provide an indication for policy makers of the magnitude of the health problem that faces them. In the US passive smoking has been estimated to cause between 30-60,000 deaths per year and between 90-180,000 non-fatal cardiovascular events (Glantz and Parmley 1991; Glantz and Parmley 1995). Extrapolating these figures to the European Union (EU), a total population larger than the US, exposure to ETS may be estimated to cause 50-100,000 deaths and 200-400,000 cases of non-fatal cardiovascular events a year in the EU (European Network for Smoking Prevention 2001). The public health importance of the potential link between ETS and cardiovascular disease lies with the fact that heart disease is much more common than lung cancer.

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## **5. HEALTH EFFECTS OF OCCUPATIONAL EXPOSURE TO ETS**

### **Lung cancer**

While the number of studies with direct observation of occupational exposure to ETS is limited, given the causal association between spousal smoking and lung cancer incidence in non-smokers, it is reasonable to expect the same result from exposure to ETS at work, especially since workplace ETS levels are often higher than those encountered in the home. An increasing body of scientific evidence supports this. Wells (1998) undertook a review of 14 studies that examined the risk of passive smoking in never smokers and concluded that passive smoking in the workplace increased the risk of lung cancer by 39%. Work conducted by IARC has provided evidence of a dose response relationship between exposure to ETS in the workplace and the risk of lung cancer. This large study found an excess risk of the order of 17% with evidence of increasing risk for increasing duration of exposure (Boffetta, Agudo, et al. 1998). Brown (1999) has estimated that the increase in lung cancer risk for non-smokers from exposure to ETS at work is of the order of 25%.

The biological plausibility, the consistency of the reported excess risk of the association, the dose response and the consistency of findings across studies of varying designs are highly persuasive of a causal relationship between ETS in the workplace and lung cancer.

### **Cardiovascular disease**

In contrast to accumulating evidence on the increased risk of heart disease from exposure to spousal ETS, studies of ETS exposure in the workplace are relatively sparse and somewhat inconclusive. Kawachi and Colditz (1999) after examining the six available (published) studies concluded that, although most of the results did not individually achieve statistical significance, the results were consistent across countries with different lifestyles and diets, and there was evidence of a dose-response relationship between intensity of ETS exposure in the workplace and risk of cardiovascular disease. Further studies are needed to conclusively demonstrate an effect of ETS exposure in the workplace on heart disease risk, and in particular studies are needed to relate the intensity and duration of ETS exposure at work to heart disease risk. However the current paucity of evidence on ETS exposure in the workplace and heart disease should not take away from the fact that there is no biologically plausible reason to believe that the hazards of ETS exposure that have been demonstrated in the home should not also apply to the workplace. Dietary and other lifestyle confounding is likely to be less of a problem in occupational studies as non-smokers who work with smokers do not necessarily share their diet and lifestyle. There is every reason to anticipate that the adverse effects could be higher in workplaces where ETS exposures, as indicated by cotinine levels, are considerably higher than that seen in the home.

### **Low birth weight**

ETS exposure in non-smoking women during pregnancy causes a reduction in birth weight. Reductions in birth weight are causally related to adverse health outcomes. Special provisions are required to protect pregnant women in the workplace. (See section 4.1).

### **Respiratory health effects**

Lam, Ho, et al. (2000) showed a clear association and dose response relationship between ETS exposure at work and respiratory symptoms such as cough, phlegm production, shortness of breath and chest colds in Hong Kong policemen. Similar findings in the workplace were also reported by Leuenberger, Schwartz, et al. (1994). It could be argued that there may be other lifestyle factors predisposing to such respiratory symptoms that are more prevalent in people willing to work in smoky workplaces than in those who work in non-smoking workplaces, thereby confounding the association. What is more likely however is that there is a 'healthy worker' effect whereby employees with predisposing illnesses such as

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asthma, do not stay in these types of employment.

A study of workers in California bars found that the reported prevalence of these respiratory symptoms decreased in workers when the law was changed to make all bars and restaurants smokefree (Eisner, Smith, et al. 1998).

There are additional consequences for employers and employees. Passive smoking at work is strongly associated with absence from work, doctor consultations and use of medications thereby resulting in costs to employers, non-smoking employees and the health services (McGhee, Adab, et al. 2000).

### **Exposure To ETS among bar staff and hospitality workers**

Bar staff and other hospitality workers are of particular importance with respect to occupational ETS exposures as they are likely to have higher and more sustained exposure than other occupational groups (see Table 4). The exposure of bar staff and other hospitality workers to ETS is involuntary, whereas the exposure of patrons socialising in bars and restaurants is voluntary.

Table 4 shows the amounts of nicotine that have been measured in various workplaces.

**TABLE 4**  
**Measurement of ETS in the Workplace**

Area	Nicotine in the air (Ng/m <sup>3</sup> )
Nightclubs	37.1
Services	3.0
Industry Workplaces	2.7
Offices	0.6
Workplaces with:	
Ban on smoking	0-0.39
Restrictions on smoking	1.3-5.9
Smoking allowed	8.6-10

(European Network for Smoking Prevention 2001)

Waiters, bartenders and counter workers etc. who are exposed to high levels of exposure to ETS have been shown to have an increased risk of death from lung cancer (Dimich-Ward, Gallagher, et al. 1988; Lindsay, Stavrak, et al. 1993). These and earlier studies though have often not taken into account the smoking habits of the subjects and have also relied on death certificates, a method which has been criticised because of the questionable quality of their occupational information.

Bars and nightclubs can constitute extreme ETS exposure settings (Jarvis, Foulds, et al. 1992; Siegel 1993). Nightclub musicians have been shown to be an occupational group exposed to high concentrations of ETS (Bergman, Johnson, et al. 1996). Siegel (1993) undertook a review of epidemiological studies examining ETS and the risk of lung cancer in food-service workers. He reported that the increase in risk of lung cancer in these professions could be as high as 50%, which in part was attributable to tobacco smoke exposure in the workplace.

There is an increasing body of research that has provided objective data on the actual extent of exposure of hospitality workers to ETS in the workplace by measuring cotinine levels. In Hong Kong, a survey of non-smoking workers in the catering industry assessed passive smoking exposures in different work settings and their risks for heart disease and cancer (Hedley, McGhee, et al. 2001). The majority of



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catering workers (waiters and other staff) were shown to have high levels of exposure to ETS in their workplace. Among a catering workforce of 200,000 it was estimated that over a working lifetime 6,000 would die from passive smoking due to heart disease and lung cancer and of these deaths, 64% would be workers who had never smoked.

In New Zealand it was found that hospitality workers who worked in premises that allowed smoking by customers had substantially increased exposures to ETS (based on changes to salivary cotinine concentrations pre and post-shift) compared with those working in smoke-free premises (Bates, Fawcett, et al. 2002). It was also noted that those premises which operated clean air policies by restricting customer smoking to certain areas did reduce average exposure of staff to ETS but such exposure remained far greater (approximately 60-fold) than in smoke-free premises.

In the only published European study of bar workers, Jarvis, Foulds, et al. (1992) studied salivary cotinine in 42 non-smoking bar staff in 27 pubs in London and Birmingham. They found that non-smokers who work in pubs were a group particularly exposed to tobacco smoke with resultant likely harm to health. Ten years later a similar study was undertaken. The position remains that non-smokers working behind the bar in pubs are an occupational group with extremely high exposure to other people's tobacco smoke (Jarvis, In Press). The only Irish study to examine ETS exposure found that carbon monoxide levels were considerably higher than the outdoor background levels in 14 bars in Galway (Mulcahy and Repace 2002).

### **Workplace attributable risk: lung cancer and heart disease**

Research from New Zealand (population 3.8 million) revealed that in 1989, over 270 deaths from heart disease and lung cancer were attributable to ETS with over 150 ischaemic heart disease deaths due to passive smoking in the workplace (Kawachi, Pearce, et al. 1989). More recently it was estimated that if ETS exposure was eliminated there would be over 300 fewer deaths per year (Woodward and Laugesen 2001). A similar picture has emerged from Finland (population 5.1 million). In a comprehensive study of the burden of work-related fatalities in 1996, researchers estimated the mortality from exposure to passive smoking at work (Nurminen and Jaakkola 2001): 2.8% (52 deaths) of all lung cancer deaths, 3.4% (106) of all ischaemic heart disease deaths and 9.4% (78) of all strokes. Overall just under 1% of all deaths (about 250) in the relevant age categories was estimated to be due to passive smoking in the workplace.

In conclusion, ETS exposure in the workplace can cause lung cancer and heart disease. This position would appear to be supported from a legal perspective. It has become increasingly evident that passive smoking is considered an occupational health risk.

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## 6. CONTROL MECHANISMS FOR ETS

Two approaches to controlling exposure to ETS are ventilation and legislation.

### 6.1 ETS and Ventilation

The World Health Organization has stated that under the principle of the human right to health, everyone has the right to breathe healthy indoor air (World Health Organization 2000b). Following the basic laws of physics, ETS rapidly diffuses throughout a room. At a ventilation rate of one air change per hour it can take more than three hours for 95% of the smoke in a typical room to dissipate once smoking has ended. This indicates that using ventilation to eliminate ETS in indoor spaces presents a considerable if not impossible task to ventilation engineering professionals (Repace 1994). Air quality may not be the same throughout a ventilated space. What really counts for the occupants of an indoor space is the air quality in the breathing zone (European Concerted Action 1992).

At a workshop sponsored by the Federal Occupational Safety and Health Administration (OSHA) and the American Conference of Governmental Industrial Hygienists (ACGIH) in June 2000, a panel of ventilation experts concluded that *dilution ventilation*, used in virtually all mechanically ventilated buildings, will not control second-hand smoke in the hospitality industry (e.g., restaurants, bars, etc). In *displacement ventilation* there is an air supply zone, occupied by people, and an exhaust air zone above this (European Concerted Action 1992). The best conditions are achieved when there is minimal mixing between the exhaust and supply zone. *Air cleaning* was judged to be somewhere between dilution and displacement ventilation in efficacy, depending on the level of maintenance. The panel failed to quantify the ETS exposure or risk for workers or patrons either before or after the application of the new technology. Panelists observed that building ventilation codes are not routinely enforced. They also noted the lack of recognized standards for acceptable ETS exposure as well as the lack of information on typical exposure levels.

However, indoor air quality standards for ETS have been proposed in the scientific literature, and reliable mathematical models exist for predicting pollutant concentrations from indoor smoking. These proposed standards and models permit application of an indoor air quality procedure for determining ventilation rates as set forth in American Society for Heating, Refrigeration and Ventilation Engineers Standard 62 (American Society of Heating Refrigeration and Ventilation Engineers (ASHRAE) 1989). Using this procedure, it is clear that dilution ventilation, air cleaning, or displacement ventilation technology even under moderate smoking conditions cannot control ETS risk to *de minimis* levels for workers or patrons in hospitality venues without massively impractical increases in ventilation.

Panelists concluded that well-mixed dilution ventilation, used in the overwhelming majority of current installations, was unsatisfactory for controlling worker exposure to ETS in hospitality venues. Local area exhaust ventilation, smokeless ashtrays, air cleaning, and displacement ventilation were identified as potentially more effective. Of these, displacement ventilation was viewed as the most promising, with estimated 90% reductions under the most favorable conditions. However these estimates were based on professional judgment rather than on measured data. Moreover, the panel raised several concerns about displacement technology, including lack of familiarity by many ventilation engineers, difficulty with retrofitting existing installations, and potential aesthetic problems.

Although air filters are capable of high capture efficiencies, they also require high airflow to be effective, and need regular effective maintenance to remain effective. Costs are a major consideration in the restaurant industry, which limits the implementation of high technology solutions such as 100% outside air 1-pass systems.

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As stated above, the OSHA/ACGIH panel estimated that ETS risk levels for lung cancer and heart disease combined would be reduced by 90% under ideal displacement ventilation. This places estimated ETS risks between 1.5 to 2.5 per 1000 workers, which is 1.5 to 2.5 times OSHA's Significant Risk level, and 1,500 to 2,500 times the *de minimis* or 'acceptable risk' level for federally regulated hazardous air pollutants. Therefore even a 90% reduction in ETS exposure yields massively unacceptable risk. The conclusion of the OSHA/ACGIH ventilation panel is that dilution ventilation is not a viable control option for ETS.

Ventilation systems are therefore unlikely to provide the basic human right of good quality indoor air. Smoking bans remain the only viable control measure to ensure that workers and patrons of the hospitality industry are protected from exposure to the toxic byproducts from tobacco combustion.

## **6.2 Workplace ETS Legislation, Ireland and elsewhere**

### **European Union legislation controlling ETS in the workplace**

To date the only EU instrument found which attempts to address ETS as a hazard in itself is an Opinion of 14 June 1999 by the Consumer Committee on Socially Responsible Community Tobacco Policy calling, on health and safety grounds, for a ban on smoking in public places, such as restaurants, workplaces, schools, hospitals, cinemas and public transport.

On 17 June 2002 the EU Commission (through Commissioner David Byrne) adopted a proposal for a Council Recommendation on the 'Prevention of Smoking and on Initiatives to Improve Tobacco Control'. It called on Member States to, among other tobacco control actions, 'provide adequate protection from exposure to passive smoking at the workplaces, in enclosed public places and in public transport, and to strengthen smoking prevention programs'.

### **Legislative approaches to workplace ETS in Ireland**

ETS exposure in the workplace can be controlled using both public health legislation and health and safety legislation.

Public health legislation, although primarily aimed at the protection of the general public, will also protect some workers such as those who work in public areas of offices and public transport (e.g. the Tobacco (Health Promotion and Protection) Act, 1988 and subsequent Regulations in 1990 and 1995). A voluntary code on workplace smoking 'Working together for cleaner air' was agreed between the government and social partners and published in 1994. There is not, to our knowledge, any baseline or follow up data on its effectiveness. In Ireland the Public Health (Tobacco) Act, 2002 will give the Minister for Health and Children further powers to make Regulations including the banning of smoking in any 'place of work' (using the definition of 'place of work' in the Safety, Health and Welfare at Work Act, 1989).

Health and safety legislation has the potential to protect workers in several ways. It can protect workers from ETS indirectly by forbidding smoking because it might cause an explosion in a particular work process or because it might cause the inhalation of toxic material which has contaminated workers' hands (Safety, Health and Welfare at Work (Carcinogens) Regulations, 2001). These same Carcinogens' Regulations could also be used to define ETS as a carcinogen in its own right (see section 4.1). In the area of safety and health legislation there is also the need to protect 'vulnerable groups'. An example is the Irish Safety, Health and Welfare at Work (Pregnant Employees etc.) Regulations, 2000. Regulation 4 (a) requires an employer 'to assess any risk to the safety or health of employees, and any possible effect on the pregnancy of, or breastfeeding by employees, resulting from any activity at that employer's place of work likely to involve a risk of exposure to any agent, process or working condition and, for that purpose, to determine the nature, degree and duration of any employee's exposure to any agent, process

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or working condition and to take the preventive and protective measures necessary to ensure the safety and health of such employees and to avoid any possible effect on such pregnancy or breastfeeding'. The known effects of ETS on the foetus may well require pregnant employees to be protected under these regulations (see also section 4.1).

It is implicit in all health and safety legislation that workers should be protected from risks to their health and the potential is there in general health and safety legislation, such as Section 6 of the Safety, Health and Welfare at Work Act, 1989, to identify ETS as a hazard and controllable risk from which workers must be protected.

## **Legislative approaches to ETS in other countries**

### **Finland**

Finland provides a good model of the use of health and safety legislation to control ETS. Smoking restrictions at workplaces in Finland were voluntary until March 1995 when reform of the Tobacco Control Act prohibited smoking in all common and public premises. The new legislation gave employers two options when implementing the Act: either impose a total ban on smoking or allow smoking in designated smoking rooms with separate ventilation systems and lower air pressure than non-smoking facilities.

Heloma, Jaakkola, et al. (2001) found that in terms of reducing smoking and nicotine concentrations in indoor air, legislation has clearly achieved better results than voluntary programmes. This study was conducted at twelve medium-sized and large workplaces by investigators from the Finnish Institute of Occupational Health. The prevalence of smoking decreased from 29.6% to 25% and the decline was significant for both men and women, and varied by education level. The average number of cigarettes consumed daily by smokers fell from 19 to 16. At industrial workplaces the median nicotine concentration in air declined from 1.2 to 0.05 micrograms per cubic meter, while the corresponding fall in the service sector was from 1.5 to 0.2 and in offices from 0.4 to 0.1 micrograms per cubic meter. Legislation on smoking in restaurants and cafeterias has also been tightened recently, resulting in a remarkable fall in exposure to ETS at work for tens of thousands of employees in the catering industry (extract from Finnish trade union website <http://www.kaapeli.fi/unions/2001/20011120.htm>). ETS is now defined as a workplace carcinogen and accordingly workers exposed to it must be registered and notified to the labour inspectors on an annual basis. Similarly, specific maternity leave legislation was extended to include ETS-exposed workers (personal communication, Dr. Aalto Asko, Senior Occupational Medical Adviser, Finland).

### **United Kingdom**

The United Kingdom has followed a voluntarist approach to the control of ETS. The government published a white paper on tobacco on 10 December 1998 (Great Britain Department of Health 1998). This stated, "We are not going to ban smoking at work but the Health and Safety Commission is going to consult on a new Approved Code of Practice on smoking in the workplace. This will considerably toughen existing measures. The Code will be designed to improve protection of the welfare of all employees by defining the kind of smoking policies employers need to operate to comply with existing health and safety legislation. Consultation will begin in Spring 1999. The Health and Safety Executive proposed a Code of Practice in September 2000. This includes options which employers might use including banning, ventilation, segregation etc." It appears that consultations on this Code of Practice are still ongoing.

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## **United States**

Governmental efforts to regulate exposure to ETS have occurred at the federal, state, and local levels. To date, most of the activity to restrict public smoking has occurred at the state and local levels.

The State of California has eliminated smoking in enclosed workplaces including bars and restaurants through a section of its health and safety legislation since 1995. The California State Labour Code Section 6400-6413.5 covers occupational health and safety in the workplace. Section 6404.5 of the code covers workplace ETS in great detail. It 'prohibits the smoking of tobacco in all (100%) enclosed places of employment'. Where employers provide smoking break rooms for employees, there are strict requirements governing ventilation. Establishment of smoke-free bars and taverns in California was associated with a rapid improvement in the respiratory health of bartenders (Eisner, Smith, et al. 1998).

It is of interest that New York City may soon follow California's example: a ban on smoking in bars is currently being considered.

It would appear that the only effective way to control ETS in workplaces is to have legislation banning the exposure of workers to ETS.

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## 7. CONCLUSIONS

ETS has many adverse health effects.

- ETS constituents are individually known to have harmful physiological effects and ETS *per se* has also been shown to have harmful physiological effects.
- ETS is carcinogenic and causes lung cancer and probably other cancers.
- ETS causes heart disease.
- ETS causes respiratory problems in adults and children.
- ETS has adverse effects on reproduction, including low birth weight.

ETS exposure infringes the basic human right to good quality air.

- Where workplace smoking is permitted, employee exposure to ETS is likely to be higher and more sustained than in the home environment.
- Employees need to be protected from exposure to ETS at work.
- Current ventilation technology is ineffective at removing the risk of ETS to health.
- Legislative measures are therefore required to protect workers from the adverse health effects of ETS exposure.

Research is required into the levels and effects of ETS in the Irish workplace in order to:

- Measure and monitor occupational exposures.
- Assess resultant adverse health outcomes.

High risk groups require special consideration. High risk groups include:

- Workers who are exposed to high levels of occupational ETS exposure such as those employed in the hospitality industry.
- Pregnant workers.
- Those with enhanced susceptibility to ETS due to genetic variations, including polymorphisms.

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## **APPENDIX**

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## **SUMMARY OF INTERNATIONAL POSITION STATEMENTS ON HEALTH EFFECTS OF WORKPLACE ETS** (Most recent listed first)

### ***International Agency for Research on Cancer (IARC) 2002***

*Tobacco Smoke and Involuntary Smoking (Summary), vol. 83*

<http://monographs.iarc.fr/htdocs/indexes/vol83index.html>

(International Agency for Research on Cancer 2002)

(Monograph in press)

A scientific working group of 29 experts from 12 countries convened by the monographs programme<sup>1</sup> of the International Agency for Research on Cancer (IARC) of the World Health Organization reviewed all significant published evidence related to tobacco smoking and cancer, both active and involuntary. Because of the public health importance of this evaluation, the summary has been posted in advance of the full monograph (due end of 2002 or early 2003). The group confirmed the conclusions of the 1986 group and re-stated that tobacco smoking and tobacco smoke are *carcinogenic to humans (Group 1)*. The group also concluded that involuntary smoking (exposure to second-hand tobacco smoke / ETS) is *carcinogenic to humans (Group 1)*:

- There is *sufficient evidence* that involuntary smoking (exposure to ETS) causes lung cancer in humans.
- There is *limited evidence* in experimental animals for the carcinogenicity of mixtures of mainstream and sidestream tobacco smoke.
- There is *sufficient evidence* in experimental animals for the carcinogenicity of sidestream smoke condensates.

The group stated that exposure to ETS is causally associated with

- Lung cancer. (Non-smokers exposed to ETS are exposed to the same carcinogens as active smokers. Even the typical levels of passive exposure have been shown to cause lung cancer among never smokers. Published meta-analyses of lung cancer in never smokers exposed to second-hand tobacco smoke at the workplace have found a statistically significant increased risk of 16 -19%).
- An increase in risk of coronary heart disease of 25-35 %.
- Chronic respiratory symptoms in adults.

The group reported the concern that breast cancer or any other cancer not caused by active smoking might be caused by involuntary smoking is unjustified by the evidence, and that the evidence for increased cancer risks among children exposed to parental and other passive exposures is uncertain at this time.

### ***World Health Organization and European Commission:***

Globally binding Framework Convention on Tobacco Control. (press release WHO/45, 6/6/02)

### ***ENSP (Smoking Prevention European Network)***

*Smoke Free Workplaces: Improving the health and well-being of people at work*

(European Network for Smoking Prevention 2001)

Finland, Germany and the US Toxicology Program have listed ETS as a workplace carcinogen.

*Conference recommendations:*

Smoke free workplaces are a necessity for preventing premature death and disability.

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<sup>1</sup>The IARC Monographs series publishes authoritative independent assessments by international experts of the carcinogenic risks posed to humans by a variety of agents, mixtures and exposures.

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### **NIH, NIEHS National Toxicology Program**

*Report on Carcinogens - 9th edition*

<http://ehp.niehs.nih.gov/roc/toc9.html>

<http://ntp-server.niehs.nih.gov/NewHomeRoc/9RoCFacts.html>

(US National Toxicology Program 2000)

ETS has been added to the 9th edition of the list of known human carcinogens. This listing is based on the observed causal relationship between passive exposure to tobacco smoke and human lung cancer. The listing states that there are conclusive published studies that indicate increased risk of lung cancer in non-smoking women living with smoking husbands or working with smoking co-workers.

### **American College of Occupational and Environmental Medicine (ACOEM)**

[http://www.acoem.org/position/statements.asp?CATA\\_ID=8](http://www.acoem.org/position/statements.asp?CATA_ID=8)

(American College of Occupational and Environmental Medicine (ACOEM) 2000)

In 1993, ACOEM issued a position statement saying that there was 'sufficient evidence to support minimizing occupational exposure to ETS'. The recent position statement (2000) states that 'Since then, additional scientific evidence has accumulated that compels ACOEM to update and strengthen this statement'. The goal of the 2000 document is 'to address and reduce involuntary ETS exposure in public places, including worksites'. The document concludes:

- ETS contains numerous toxins.
- Robust epidemiological evidence implicates ETS as a cause of lung cancer and as a primary cause and a source of exacerbation of excess respiratory disease.
- There is increasing evidence that ETS may be associated with other outcomes, including heart disease.

There is little doubt that ETS is an important and avoidable health hazard. Unfortunately, ETS is frequently encountered in the workplace - where it is no safer than in other environments and where it presents hazards to exposed workers and others. A unique aspect of workplace ETS is that exposure is rarely an outcome of essential manufacturing, extraction, or service delivery process. Moreover, ETS exposure, with its growing list of known hazards, is preventable by engineering or policy means.

### **World Health Organization**

From: *Background material to WHO Air Quality Guidelines for Europe 2000*, Chapter 8.1, *Environmental tobacco smoke*.

<http://www.who.int/dsa/cat98/env8.htm>

(World Health Organization 2000a)

ETS health effects in adult non-smokers are consistent with effects attributed to active smoking:

- Chronic exposures to ETS increase lung cancer mortality.
- Combined evidence from epidemiology and studies of mechanisms leads to the conclusion that ETS increases the risk of morbidity and mortality from cardiovascular disease (CVD) in non-smokers.
- ETS irritates the eyes and respiratory tract.
- ETS increases the severity and frequency of asthma attacks.
- ETS reduces birth weight in the offspring of non-smoking mothers.
- Strong suggestive evidence that ETS increases mortality from sino-nasal cancer.

Populations at special risk are infants, young children, asthmatics and adults with other risk factors for cardiovascular disease.

'Because of the extensive prevalence of ETS exposure and the high incidence of some of the health effects associated with ETS exposure, eg CVD in adults, even small increases in relative risks can translate into substantial mortality and morbidity on a population basis.'

Quantitative population estimates for CVD mortality are less certain than those for lung cancer.

*Guidelines:* 'ETS has been found to be carcinogenic in humans and to produce a substantial amount of morbidity and mortality from other serious health effects at levels of 1-10g/m<sup>3</sup> nicotine... There is no evidence for a safe exposure level.'

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**UK Chartered Institute of Environmental Health (CIEH) Policy Unit Consultation Response to Health and Safety Commission Discussion Document** entitled 'Proposal for and Approved Code of Practice on Passive Smoking at Work'.

<http://www.cieh.org/about/policy/responses/smoking.htm>

(UK Chartered Institute of Environmental Health (CIEH) Policy Unit 1999)

CIEH believes that:

- There is a significant risk to health from exposure to ETS.
- Unless by their own choice, no-one should be exposed to ETS.
- All places where people are working should be free of ETS.

CIEH is of the opinion that the introduction of prescriptive legislation prohibiting smoking in the workplace is the only truly effective method of ensuring that all places where people are working are free of ETS.

### **World Bank**

*Curbing the Epidemic: Governments and the Economics of Tobacco Control*

Chapter 2, *The health consequences of smoking*

<http://www1.worldbank.org/tobacco/chapter2.asp>

(World Bank 1999)

Smokers affect not only their own health but also the health of those around them. 'Adults exposed chronically to others' tobacco smoke also face small but real risks of lung cancer and higher risks of cardiovascular disease.'

### **Institute for Global Tobacco Control, Johns Hopkins School of Public Health.**

*Environmental Toxicants*

*Environmental Tobacco Smoke*

<http://www.jhsph.edu/gtc/ets.html>

(Institute for Global Tobacco Control 1999)

ETS is now considered a cause of asthma and a factor exacerbating asthma and as a cause of heart disease as well as a cause of lung cancer. The adverse effects of involuntary exposure to ETS have provided a strong rationale for policies directed at reducing and eliminating exposure of non-smokers to ETS. Complete protection of non-smokers in public locations and the workplace may require the banning of smoking.

### **Report of the Scientific Committee on Tobacco and Health (SCOTH), UK**

<http://www.archive.official-documents.co.uk/document/doh/tobacco/report.htm>

(UK Scientific Committee on Tobacco and Health 1998)

*Environmental Tobacco Smoke (Part Two)*

Conclusions:

- Exposure to environmental tobacco smoke is a cause of lung cancer and, in those with long term exposure, the increased risk is of the order of 20-30%.
- Exposure to environmental tobacco smoke is a cause of ischaemic heart disease and, if current published estimates of the magnitude of relative risk are validated, such exposure represents a substantial public health hazard.
- Smoking in the presence of infants and children is a cause of serious respiratory illness and asthmatic attacks.
- Sudden infant death syndrome, the main cause of post-neonatal death in the first year of life, is associated with exposure to environmental tobacco smoke. The association is judged to be one of cause and effect.
- Middle ear disease in children is linked with parental smoking and this association is likely to be causal.



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*Recommendations:*

Smoking in public places should be restricted on the grounds of public health. The level of restriction should vary according to the different categories of public place but smoking should not be allowed in public service buildings or on public transport, other than in designated and isolated areas. Wherever possible, smoking should not be allowed in the work place.

Health education programmes should focus on the dangers of ETS in foetal development and, postnatally, in sudden infant death syndrome.

***Statement by the UK Committee on the Carcinogenicity of Chemicals (COC) in Food, Consumer Products and the Environment to SCOTH on Environmental Tobacco Smoke (ETS) and Lung Cancer***

(UK Committee on the Carcinogenicity of Chemicals 1998)

Exposure to ETS over a wide range of exposure levels, including those normally encountered in homes, at work and in public places, can lead to the inhalation and delivery of genotoxic carcinogens to all parts of the respiratory tract. The COC advice on genotoxic carcinogens is to make the prudent assumption that any exposure may be associated with some increased health detriment, in this case a risk of lung cancer. (Paragraph 18)

Taking all the supporting data into consideration we conclude that passive smoking in non-smokers exposed over a substantial part of their life is associated with a 10-30% increase in the risk of lung cancer which could account for several hundred lung cancer deaths per annum in the UK. (Paragraph 30)

***UK White Paper on Tobacco***

*Smoking kills*

<http://www.archive.official-documents.co.uk/document/cm41/4177/contents.htm>

(Great Britain Department of Health 1998)

‘Passive smoking also kills.’

- Passive smoking, even in low levels, can cause illness.
- Asthma sufferers are more prone to attacks in smoky atmospheres.
- A non-smoker, living or working in a very smoky environment over a prolonged period is 20-30% more likely to get cancer than a non-smoker who does not.
- Hundreds of people die every year in the UK as a result of high levels of exposure to passive smoke.

However the White Paper concludes: ‘We do not think a universal ban on smoking in all public places is justified while we can make fast and substantial progress in partnership with industry.’ Agreement has been reached with representatives of the hospitality trade to a Charter to ensure that consumers are better able to choose whether to eat, drink or socialise in smoky atmospheres.

***California Environmental Protection Agency (California EPA) 1997***

*Smoking and Tobacco Control Monograph 10 (1999)*

*Health effects of exposure to environmental tobacco smoke*

[http://rex.nci.nih.gov/NCI\\_MONOGRAPHS/MONO10/MONO10.HTM](http://rex.nci.nih.gov/NCI_MONOGRAPHS/MONO10/MONO10.HTM)

(California Environmental Protection Agency 1997)

The executive summary includes the following table.

***Table ES.1. Health Effects Associated with Exposure to Environmental Tobacco Smoke Effects Causally Associated with ETS Exposure***

*Developmental Effects*

Foetal Growth: Low birthweight or small for gestational age

Sudden Infant Death Syndrome (SIDS)

*Respiratory Effects*

Acute lower respiratory tract infections in children

(e.g., bronchitis and pneumonia)

Asthma induction and exacerbation in children

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Chronic respiratory symptoms in children  
Eye and nasal irritation in adults  
Middle ear infections in children  
*Carcinogenic Effects*  
Lung Cancer  
Nasal Sinus Cancer  
*Cardiovascular Effects*  
Heart disease mortality  
Acute and chronic coronary heart disease morbidity

***Effects with Suggestive Evidence of a Causal Association with ETS Exposure***

*Developmental Effects*  
Spontaneous abortion  
Adverse impact on cognition and behavior  
*Respiratory Effects*  
Exacerbation of cystic fibrosis  
Decreased pulmonary function  
*Carcinogenic Effects*  
Cervical cancer

***Australian National Health and Medical Research Council report***

*Health Effects of Passive Smoking*

<http://www.health.gov.au:80/nhmrc/advice/nhmrc/index.htm>

(Australian National Health and Medical Research Council (NHMRC) 1997)

*Introduction.* The health effects of active smoking are well documented. Given the seriousness of these effects, and the toxic nature of environmental tobacco smoke, researchers and health authorities have for some time been concerned about the possible health effects of 'passive' smoking (exposure to environmental tobacco smoke or ETS). In 1986, the NHMRC examined this issue and found sufficient evidence of an adverse effect on health to recommend that policies and practices be introduced to reduce exposure to ETS. Since 1986, the quantity and quality of scientific research into passive smoking and health has increased greatly. Hence, the NHMRC has conducted a further review of the scientific literature on the health effects of passive smoking.

The various estimates relate to home exposure. It seems likely that exposure to ETS outside the home, for example in the workplace and in public places, would cause a similar burden of illness amongst adults as does exposure in the home.

*Conclusions.* The scientific evidence shows that passive smoking causes lower respiratory tract illness in children and lung cancer in adults and contributes to the symptoms of asthma in children. Passive smoking may also cause coronary heart disease in adults. It is estimated that passive smoking contributes to the symptoms of asthma in 46,500 Australian children each year and causes lower respiratory illness in 16,300 Australian children. It also causes about 12 new cases of lung cancer each year in adult Australians. Passive smoking may also cause 77 deaths a year from coronary heart disease.

***US Occupational Safety and Health Administration (OSHA)***

*Indoor Air Quality (Proposed rules)*

[http://www.osha.gov/pls/oshaweb/owadisp.show\\_document?p\\_table=FEDERAL\\_REGISTER&p\\_id=13369&p\\_text\\_version=FALSE](http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=FEDERAL_REGISTER&p_id=13369&p_text_version=FALSE)

(US Occupational Safety and Health Administration (OSHA) 1994)

The epidemiological and clinical studies, taken in aggregate, indicate that exposure to environmental tobacco smoke may produce mucous membrane irritation, pulmonary, cardiovascular, reproductive, and carcinogenic effects in non-smokers. Exposure to ETS may aggravate existing pulmonary or cardiovascular disease in non-smokers. In addition, animal studies show that both mainstream and sidestream tobacco smoke produce similar adverse effects.

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**US Environmental Protection Agency (EPA)**

*Respiratory health effects of passive smoking: Lung cancers and other disorders*

<http://www.epa.gov/iaq/pubs/etsfs.html>

(US Environmental Protection Agency (EPA) 1992)

Review 1992: ETS classified as a class A (known human) carcinogen.

The report concludes that exposure to ETS - commonly known as second-hand smoke - is responsible for approximately 3,000 lung cancer deaths each year in non-smoking adults and impairs the respiratory health of hundreds of thousands of children.

**International Agency for Research on Cancer (IARC)**

*Tobacco smoke, Supplement 7*

<http://www.iarc.fr/Monographs/>

(International Agency for Research on Cancer 1987) (p.359)

**Evidence for carcinogenicity to humans (sufficient)**

*Cigarette smoking*: shown to cause lung cancer, bladder cancer, etc. etc.

*ETS*: Tobacco smoke affects not only people who smoke but also those who are exposed to the combustion products of other people's tobacco (passive smokers). The most numerous observations hitherto available concern lung cancer, and the results of most of the 13 main epidemiological studies carried out so far are compatible with either an increased risk from passive smoking or an absence of risk. However, the aggregate evidence from these studies, taken together with knowledge of the nature of sidestream and mainstream smoke, of the materials absorbed during passive smoking and of the quantitative relationships between dose and effect that are commonly observed after exposure to carcinogens, lead to the conclusion that passive smoking does carry some risk for lung cancer.

*Overall evaluation*:

Tobacco smoke is carcinogenic to humans (Group 1)

**International Agency for Research on Cancer (IARC)**

*Tobacco Smoking, Volume 38*

<http://www.iarc.fr/Monographs/>

(International Agency for Research on Cancer 1986)

There is sufficient evidence that tobacco smoke is carcinogenic to humans. The major cause of lung cancer is tobacco smoking, primarily of cigarettes. The occurrence of malignant tumours of the respiratory tract and of the upper digestive tract is causally related to the smoking of different forms of tobacco (cigarettes, cigars, pipes, bidis). The occurrence of malignant tumours of the bladder, renal pelvis and pancreas is causally related to the smoking of cigarettes.

**US Surgeon General**

*The Health Consequences of Involuntary Smoking*

<http://sgreports.nlm.nih.gov/NN/Views/AlphaChron/alpha/10006/>

(US Department of Health and Human Services 1986)

Involuntary smoking is a cause of disease, including lung cancer, in healthy non-smokers. The simple separation of smokers and non-smokers within the same airspace may reduce, but does not eliminate, the exposure of non-smokers to environmental tobacco smoke.

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**IRELAND**

***Health Promotion Unit, Department of Health and Children with IBEC and ICTU:***

*Working together for cleaner air. Developing smoke-free policies in the workplace.*

(Irish Health Promotion Unit, IBEC et al.)

Booklet written to assist employers and employees adopt the Voluntary Code of Practice on Smoking in the Workplace. Health Promotion Unit of Department of Health and Children actively promoting the development of a smoke-free atmosphere in the workplace.

***Department of Health and Children,***

Report of the Tobacco Free Policy Review Group: *Ireland - a smoke free zone. Towards a tobacco free society.*  
(Tobacco Free Policy Review Group 2000)

Action plan proposed which includes advocating improved compliance with existing laws and further protection against the harmful effects of passive smoking.

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**TABLE A.1**

**MEAN CONCENTRATIONS OF NICOTINE AND COTININE IN THE SALIVA, PLASMA, AND URINE OF ETS-EXPOSED VOLUNTEERS<sup>1</sup>**

<b>Time</b>	<b>Saliva (ng/ml)</b>		<b>Plasma (ng/ml)</b>		<b>Urine (ng/mg creatinine)</b>	
	<b>Nicotine</b>	<b>Cotinine</b>	<b>Nicotine</b>	<b>Cotinine</b>	<b>Nicotine</b>	<b>Cotinine</b>
Minutes of exposure						
0 (baseline)	3	1.0	0.2	0.9	17	14
40	830	1.1	0.3	0.9	-- <sup>2</sup>	--
60	880	2.1	0.3	1.2	--	--
80	730	1.4	0.5	1.3	84	28
Minutes post exposure						
30	148	1.7	0.4	1.8	--	--
150	17	3.1	0.7	2.9	100	46
240	3	2.0	1.1	3.3	--	--
300	7	3.5	0.6	3.4	48	55

Source: (California Environmental Protection Agency 1997) (from Hoffman *et al.* 1984)

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<sup>1</sup> Individuals were exposed to ETS generated from continuous smoking of 4 cigarettes by machine. The air concentration of nicotine stabilized at approximately 280 ug/m<sup>3</sup> within 10 to 15 minutes.

<sup>2</sup> Samples not taken for this exposure interval.

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TABLE A.2

COMPARISON OF BIOMARKERS IN UNEXPOSED AND ETS-EXPOSED NON-SMOKERS, AND ACTIVE SMOKERS<sup>3</sup>

Biochemical parameter	Unexposed non-smokers (n=46) Mean value	% of active smokers' value	ETS-exposed non-smokers (n=54) Mean value	% of active smokers' value	Active smokers (n=94) Mean value
CO in expired air (ppm [mg/m] <sup>3</sup> )	5.7[6.5]	27	5.5[6.3]	26	20.8[24]
COHb (%)	0.9	23	0.8	21	3.9
Nicotine (ng/ml)					
In plasma	1.0	7	0.8	5.4	14.8
In saliva	3.8	0.6	5.6	0.8	672.5
In urine	3.9	0.2	12.1*	0.7	1749.9
Cotinine (ng/ml)					
In plasma	0.8	0.3	2.0*	0.7	275.2
In saliva	0.7	0.2	2.5**	0.8	309.9
In urine	1.6	0.1	7.7**	0.6	1391.0
Thiocyanate (μmol/l)					
In plasma	48	39	53	43	123
In saliva	1270	52	1327	54	2450
In urine	73	47	77	50	155

Source: (California Environmental Protection Agency 1997)

<sup>3</sup> From IARC (1986) using data from Jarvis and russel (1984).

• indicates p<0.01 between exposed and unexposed non-smokers

•• indicates p<0.001 between exposed and unexposed non-smokers

**TABLE A.3**

**CHEMICAL CONSTITUENTS OF TOBACCO SMOKE THAT HAVE BEEN CLASSIFIED OR IDENTIFIED AS TO THEIR CARCINOGENICITY**

Modified from California EPA report (California Environmental Protection Agency 1997) with the classifications of the chemicals in the European Union also included (column 2)

<b>COMPOUND</b>	<b>EU CLASSIFICATION</b>	<b>IARC CLASSIF. (a)</b>	<b>US EPA CLASSIF. (b)</b>
<b>Organic Compounds</b>			
Acetaldehyde	Carc. Cat 3; R40, Xi, R36/37	<b>2B</b>	<b>B2</b>
Acetamide	Carc. Cat. 3; R40, Xn; R68	<b>2B</b>	<b>NC</b>
Acrolein	T+; R26, T; R24/25, C; R34	<b>3</b>	<b>C</b>
Acrylonitrile	Carc. Cat. 2; R45, T, R23/24/25, Xi; R37/38-41, R43	<b>2A</b>	<b>B1</b>
4-Aminobiphenyl	Carc. Cat. 1; R45, Xn; R22	<b>1</b>	
Aniline	Carc. Cat 3; R40, T; R48/23/24/25, Xn; R20/21/21	<b>3</b>	<b>B2</b>
o-Anisidine	Carc. Cat. 2; R45, Muta Cat. 3; R68, T; R23/24/25	<b>2B</b>	<b>P65-MC</b>
Benz[a]anthracene 1,3-	Carc. Cat. 2; R45	<b>2A</b>	<b>B2</b>
Benzene	Carc. Cat. 1; R45, T; R48/23/24/25	<b>1A</b>	
Benzo[b]fluoranthene	-	<b>2B</b>	<b>B2</b>
Benzo[j]fluoranthene	Carc. Cat. 2; R45	<b>2B</b>	
Benzo[k]fluoranthene	Carc. Cat. 2; R45	<b>2B</b>	<b>B2</b>
Benzo[a]pyrene	Carc. Cat. 2; R45	<b>2A</b>	<b>B2</b>
Butadiene	[Carc. Cat. 1]*	<b>B2</b>	<b>B2</b>
Captan	Carc. Cat. 3; R40, T; R23, Xi; R41, R43	<b>3</b>	<b>B2</b>
Carbon disulfide	Repr. Cat. 3; R62-63 T; R48/23, Xi; R36/38		<b>D</b>
Carbon monoxide	Repr. Cat. 1; R61, T; R23-48/23		<b>D</b>
Chrysene	Carc. Cat. 2; R45, Muta. Cat. 3; R68	<b>3</b>	<b>B2</b>
DDT	Carc. Cat. 3; R40, T; R25-48/25	<b>2B</b>	<b>P65-MC</b>
Dibenz[a,h]acridine	-	<b>2B</b>	<b>P65</b>
Dibenz[a,j]acridine	-	<b>2B</b>	<b>P65</b>
Dibenz[a,h]anthracene	Carc. Cat. 2; R45	<b>2A</b>	<b>B2</b>
7H-Dibenzo[c,g]carbazole	-	<b>2B</b>	<b>P65</b>
Dibenzo[a,e]pyrene	-	<b>2B</b>	<b>P65</b>
Dibenzo[a,h]pyrene	-	<b>2B</b>	<b>P65</b>
Dibenzo[a,i]pyrene	-	<b>2B</b>	<b>P65</b>
Dibenzo[a,l]pyrene	-	<b>2B</b>	<b>P65</b>
1,1-Dimethylhydrazine	[Carc. Cat. 2; R45]*	<b>2B</b>	<b>P65</b>
1-Naphthylamine	Xn; R22	<b>3</b>	<b>NC</b>
Formaldehyde	Carc. Cat. 3; R40, T; R23/24/25, C; R34	<b>2A</b>	<b>B1</b>
Hydrazine	Carc. Cat. 2; R45 T; R23/24/25, C; R34 R43	<b>2B</b>	<b>P65</b>
Indeno(1,2,3-cd)pyrene	-	-	
2-Naphthylamine	Carc. Cat. 1; R45 Xn; R22	<b>1</b>	<b>P65</b>

**CHEMICAL CONSTITUENTS OF TOBACCO SMOKE THAT HAVE BEEN CLASSIFIED OR IDENTIFIED AS TO THEIR CARCINOGENICITY (continued)**

<b>COMPOUND</b>	<b>EU CLASSIFICATION</b>	<b>IARC CLASSIF. (a)</b>	<b>US EPA CLASSIF. (b)</b>
<b>Organic Compounds</b>			
Nicotine 2-Nitropropane	T+; R27, T; R25 Carc. Cat. 2; R45 Xn; R20/22	<b>2B</b>	
N-(Methylnitrosamino) -1-(3-pyridyl)-1-butanone (NNK)	-	<b>NC</b>	<b>NC</b>
N-Nitrosodi-n-butylamine	-	<b>2B</b>	<b>B2</b>
N-Nitrosodiethanolamine	-	<b>2B</b>	<b>B2</b>
N-Nitrosodiethylamine	-	<b>2A</b>	<b>B2</b>
N-Nitroso-n-methylethylamine	-	<b>2B</b>	<b>B2</b>
N'-Nitrosornicotine	-	<b>2B</b>	
N-Nitrosopiperidine	-	<b>2B</b>	
N-nitrosdi-n-propylamine	-	<b>2B</b>	
N-Nitrosopyrrolidine	-	<b>2B</b>	
Styrene	Xn; R20, Xi; R36/38	<b>2B</b>	<b>C</b>
Toluene	Xn; R20, [Repro. Cat. 3; R63]*		<b>NC</b>
2-Toluidine	-	<b>2B</b>	<b>P-65</b>
Urethane	Carc. Cat. 2; R45	<b>2B</b>	<b>P65</b>
Vinyl chloride	Carc. Cat. 1; R45	<b>1</b>	<b>A</b>
<b>Inorganic Compounds</b>			
Chromium V1	Carc. Cat. 2; R49 R43	<b>1</b>	<b>A</b>
Arsenic	T; R23/25	<b>1</b>	<b>A</b>
Cadmium	Cadmium chloride & fluoride Carc. Cat. 2, R45 Repro. Cat. 2, R60-61, Muta. Cat. 3, R68, T+R26, T; R25, T;48/23/25  Cadmium oxide Carc. Cat. 2, R49 Repro. Cat. 3, R63, Muta. Cat. 3, R68, T; R23/25 T;48/23/25	<b>2A</b>	<b>B1</b>
Lead	Repro. Cat. 1; R61 Repro. Cat. 3; R62 Xn; R20/22, R33	<b>2B</b>	<b>B2</b>
Nickel	Carc. Cat. 1; R45	<b>1</b>	<b>A</b>

Sources: ARB(1993); IARC (1985, 1986, 1987, 1992); California Code of Regulations (1994); US EPA (1994).

(a) International Agency for Research on Cancer (IARC) Classification: 1, carcinogenic to humans; 2A, probably carcinogenic to humans; 2B, possibly carcinogenic to humans; 3, not classifiable as to its carcinogenicity to humans.

(b) US EPA Classification: A, human carcinogen; B1, probable human carcinogen (primarily on the basis of epidemiological data); B2, probable human carcinogen (primarily on the basis of animal data).

C, possible human carcinogen.

NC = Not classified as yet.

P65 = carcinogen under California's Proposition 65.

\* This classification is still under discussion in the European Union.

- = Not classified in the European Union.



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## **ABBREVIATIONS/ACRONYMS**

ASHRAE	American Society for Heating, Refrigerating and Ventilating Engineers.
EPA	Environmental Protection Agency.
ETS	Environmental tobacco smoke.
HSA	Health and Safety Authority
IARC	International Agency for Research on Cancer.
NAOSH	National Authority for Occupational Safety and Health aka Health and Safety Authority (HSA).
OSHA	US Occupational Safety and Health Administration.
OTC	Office of Tobacco Control.
UICC	International Union against Cancer.
WHO	World Health Organization.

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## GLOSSARY

**Atherosclerosis:** Arterial disease in which raised areas of degeneration and cholesterol deposits (plaques) form on the inner surfaces of the arteries. Hardening of the arteries.

**Attributable risk** (also called **attributable fraction**): The proportion of disease occurrence (e.g. lung cancer) potentially eliminated if exposure (e.g. to ETS) was prevented. The proportion is calculated from data on prevalence and relative risk.

**Basal coronary flow velocity:** Normal blood flow in the coronary blood vessels.

**Biological marker:** Any parameter that can be used to measure an interaction between a biological system and an environment agent, which may be chemical, physical or biological.

**Carcinogen:** Any substance capable of producing cancer or a chemical that causes or induces cancer.

**Carcinogenicity:** A property of an agent such as a chemical or chemicals or radiation that cause cancer.

**Cardiovascular:** Pertaining to the heart and blood vessels.

**California's Proposition 65:** California's legislative measure, the Safe Drinking Water and Toxic Enforcement Act of 1986, better known as Proposition 65, requires the Governor of California to publish a list of chemicals that are known to the State of California to cause cancer, birth defects or other reproductive harm. The list is updated at least once a year.

**Causal association:** The relationship between exposure and disease is one of cause and effect.

**Cerebrovascular disease:** Atherosclerosis of the arteries which supply blood and oxygen to the brain can result in cerebrovascular disease also known as stroke.

**Chronic bioassay:** A toxicological study carried out in animals, normally over the complete lifetime of the animal.

**Confounding variable:** A factor that is associated with both the variable of interest (in this case, ETS) and the outcome (e.g. lung cancer or heart disease), and is distributed unevenly in the study groups.

**Coronary heart disease:** Atherosclerosis of the arteries which supply blood and oxygen to the heart muscle results in coronary heart disease.

**Developmental toxicant:** A chemical or other agent that has the property to damage the unborn child.

**DNA:** A polymer composed primarily of units of deoxyribonucleic acids. DNA serves as the primary storage form of genetic information.

**DNA adducts:** Combination of other molecules with DNA.

**DNA sequence:** the arrangement of nucleotides in a gene.

**Endothelial dysfunction:** Impaired functioning of the lining (endothelium) of the blood vessels.

**Epidemiology:** The study of the distribution and determinants of health related states or events in

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specified populations and the application of this study to the control of health problems.

**Environmental tobacco smoke (ETS):** When non-smokers share a space with someone who is smoking they are being exposed to ambient tobacco smoke. This ambient tobacco smoke is called environmental tobacco smoke (ETS), second-hand smoke or passive smoke and is composed of exhaled mainstream smoke and sidestream smoke. ETS is generated by the combustion of tobacco products. ETS is a complex mixture of over 4000 compounds. These include over 50 known or suspected human carcinogens, such as 4-aminobiphenyls, 2-naphthylamine, benzene, nickel, and a variety of polycyclic aromatic hydrocarbons and N-nitrosamines. A number of irritants, such as ammonia, nitrogen oxides, sulphur dioxide, various aldehydes, and cardiovascular toxicants, such as carbon monoxide and nicotine are also present.

**Exposure:** The main routes of exposure for humans are dermal absorption (skin), ingestion (by mouth) and inhalation (breathing).

**Genotoxic:** Toxic to the genome or hereditary material (DNA) of the cell.

**Hazard:** A potential cause of harm to a person.

**Incidence:** The number of new cases of a particular condition arising in a given population in a given time period (usually 1 year).

**Ischaemia/reperfusion:** Inadequate supply of blood to a part of the body, followed by restoration of the blood supply.

**Ischaemia:** The loss of an adequate supply of oxygenated blood.

**Low birth weight:** Abnormally low weight of a new-born infant, usually below 2000g.

**Lung cancer:** Cancer that may appear in the trachea, air sacs and other lung tubes. It may appear as an ulcer in the windpipe, as a nodule or small flattened lump, or on the surface, blocking air tubes. It may extend into the lymphatic and blood vessels.

**Mainstream smoke:** The smoke inhaled and exhaled by smokers directly from tobacco products.

**Mucociliary:** The physiological system of mucus and hairs in the upper respiratory system that is responsible for removal of foreign material that lodges there.

**Mutagenic:** A chemical or other agent that causes mutations in or otherwise damages the hereditary material (DNA) of the cell.

**Myocardial infarction:** Commonly known as a heart attack and is the death of a section of heart muscle when its blood supply is cut off, usually by a blood clot in a coronary artery that has been narrowed by arteriosclerosis.

**One-shot carcinogen:** A carcinogen for which there is considered to be no safe level, i.e. exposure to one molecule could theoretically cause cancer (a non-threshold carcinogen).

**Olfactory sensation:** Ability to smell.

**Passive smoking:** The act of breathing environmental tobacco smoke is called passive smoking.

**Pharmacological:** The property of a chemical substance (normally a drug) to have an effect on a

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normal body function, e.g. blood pressure.

**Platelets:** Blood cells involved in the clotting of blood.

**Polymorphism:** DNA sequence variation that occurs in at least 1% of the population. Variants of genes.

**Prevalence:** The number of cases of a particular condition in a given population at a specified point in time.

**Radionuclide:** A radioactive element.

**Reproductive toxicant:** A chemical or other agent that has the property to impair the fertility or other aspect of the reproduction of men or women, or to damage the unborn child.

**Risk:** The relationship between a hazard and the harm it may cause. Risk can be defined to be a function of three variables: (a) the likelihood of a hazard causing harm to an exposed individual, (b) the severity of the harm and its consequences and (c) the number of persons exposed to the hazard.

**Sidestream smoke:** This is a mixture of the smoke emitted from the smouldering tobacco, contaminants emitted during puffs and contaminants that diffuse through the cigarette paper and the mouth end of the cigarette between puffs.

**Threshold carcinogen:** A carcinogen for which there is considered to be a safe level of exposure, below which cancer is not likely to occur.

**Tidal volume:** The volume of air that is inhaled and exhaled under normal breathing conditions.

**Toxicological:** The property of a chemical substance to have an adverse effect on human health.

**Thrombosis:** The formation of a blood clot within the circulatory system.