National response to passive smoking in enclosed public places and workplaces A Background Paper

National Public Health Partnership

Legislation Reform Working Group

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National response to passive smoking: Background Paper

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Contents

Abbreviations	V
Summary	vii
1 Introduction	1
1.1 Passive smoking: a national response	1
1.2 Aim of this background paper	1
1.3 Why now?	2
2 Passive smoking and environmental tobacco smoke	3
2.1 What is passive smoking?	3
2.2 What is environmental tobacco smoke?	3
2.3 Environmental tobacco smoke as an indoor air pollutant	3
3 Environmental tobacco smoke: effects on health	5
3.1 Adverse health effects	5
3.2 Overview of the evidence	5
3.2.1 Introduction	5
3.2.2 Reports of health and medical organisations	5
3.2.3 Other research studies	7
4 ETS exposure in public places and workplaces	11
4.1 Factors affecting exposure	11
4.2 Measurement of exposure	11
4.2.1 Biomarkers	11
4.2.2 Personal monitoring	12
4.2.3 Direct environmental measurement	12
4.3 High-risk settings	12
4.4 'Acceptable' level of exposure	13
5 The public health risk of ETS	15
5.1 Is ETS a risk to public health?	15
5.1.1 The risk of an adverse health effect occurring	15
5.1.2 The seriousness of the health effects caused by ETS	15
5.1.3 The number of people exposed to ETS	15
5.1.4 The ability of people to avoid ETS exposure	15
5.2 Reducing the risk of ETS to public health	16
5.2.1 Assessing public health risks	16
5.2.2 Exposure standards	16

6 Public opinion on passive smoking	17
6.1 The importance of community attitudes	17
6.2 Relationship between attitudes and behaviour	17
6.3 Public attitudes towards passive smoking	17
6.4 Community support for ETS protection	18
6.5 Public opinion alone has not caused change	19
7 Current ETS management strategies	21
7.1 Personal avoidance of ETS	21
7.2 Current nonlegislative approaches	21
7.2.1 Public sector workplaces	21
7.2.2 Private sector workplaces	21
7.2.3 Other enclosed public places	21
7.3 Current legislative approaches	22
8 Conclusions	25
References	27

List of Tables

Table 1 Public assessment of passive smoking as a health risk	17
Table 2 National public attitudes toward smoking limitation	18
Table 3 Public opinion concerning smoking in public social premises	19
Table 4 Actual provision of nonsmoking areas in restaurants	19
Table 5 Restaurant proprietors' views on providing nonsmoking areas	20

Abbreviations

CVD	cardiovascular disease
ETS	environmental tobacco smoke
HDL-C	high-density lipoprotein cholesterol
IARC	International Agency for Research on Cancer
LRWG	Legislative Reform Working Group
NDSHS	National Drug Strategy Household Survey
NEPC	National Environment Protection Council
NHMRC	National Health and Medical Research Council
NPHP	National Public Health Partnership
RIS	regulatory impact statement
RSP	respirable suspended particles
USEPA	United States Environmental Protection Agency
WHO	World Health Organization

Summary

Passive smoking is the term used for exposure to tobacco smoke, or the chemicals in tobacco smoke, without actually smoking. It usually refers to a situation where a nonsmoker breathes smoke emitted into the environment by other people smoking. This smoke is known as 'environmental tobacco smoke' (ETS).

In 1997, increasing evidence for the adverse health effects of ETS exposure led the Minister for Health and Aged Care, Dr Michael Wooldridge, to announce that passive smoking was a major public health issue requiring a national response.

In response to this directive the Legislative Reform Working Group (LRWG), of the National Public Health Partnership (NPHP) has developed a national response to passive smoking in enclosed public places and workplaces. This component of the overall national response to passive smoking is intended to provide a suite of resources that facilitates best practice legislation in this area. A second component of the overall national response will focus on the issue of children's exposure to ETS.

This background paper forms part of the national response to ETS exposure in public places and workplaces. While the Commonwealth government has taken a leading role in this development, the enactment of legislation regarding passive smoking remains the responsibility of the States and Territories. Information is therefore presented to assist jurisdictions in the continuing development of their own responses to this issue, including:

- definition of passive smoking and composition of ETS;
- the health effects of ETS;
- exposure levels;
- the public health risk associated with ETS;
- public opinion on passive smoking; and
- current ETS management strategies.

With this information, jurisdictions will be able to continue to develop their own responses to the issue of passive smoking, by adapting the national response to local circumstances and systems as they see fit. This approach would also facilitate jurisdictional action in a key strategy identified in the National Tobacco Strategy 1999 to 2002–03. It is noted, however, that by endorsing the national response, jurisdictions do not commit to implementing any of its recommendations.

Composition of ETS

ETS consists of two types of smoke, namely:

- smoke from the burning cigarette, or sidestream smoke, which accounts for most of the tobacco smoke in a room of smokers; and
- smoke exhaled (exhaled mainstream smoke).

Although the chemical composition of ETS differs from the smoke inhaled by active smokers, both contain a similar range of substances with known toxic and carcinogenic effects. Sidestream smoke is produced at lower temperatures than mainstream smoke and contains higher levels of tobacco burned per milligram than mainstream smoke and more of a range of compounds, including a number of carcinogens.

Health effects of environmental tobacco smoke

A review of the research shows that ETS exposure can cause respiratory illness, asthma and can predispose to allergic sensitisation in children, contributes to the risk of sudden infant death syndrome, and significantly increases the risk of cardiovascular disease, stroke, lung cancer and respiratory disease in adults. Maternal ETS exposure is associated with adverse pregnancy outcomes and an increased risk of central nervous system tumours in children. These effects are dose-related.

A number of studies have shown that workplace exposure to ETS is comparable to home exposure, particularly when assessing the risk of lung cancer. Furthermore, the risk to health, for example the risk of lung cancer, may be seriously underestimated in studies where additional sources of ETS exposure were not assessed or accounted for.

Exposure levels

The proportion of total exposure to ETS that occurs in public places and at work differs between one population and another and with changing smoking patterns. In 1997, the NHMRC estimated that, in recent years, workplace exposure in Australia has been equivalent to, or greater than, home exposure. The harm from passive smoking depends on both the time spent in that environment and the concentration of ETS in the airspace. This is in turn affected by the size of the space, the number of cigarettes smoked there in a given time and the ventilation rate. In certain occupational settings, these factors combine to create a substantial risk of harm from ETS. Levels of exposure are hard to measure precisely. Three types of measures have been used:

- biological markers;
- personal monitoring; and
- direct environmental monitoring.

Workers in some industry sectors experience greater health risks due to levels of ETS exposure; for example, casino workers and hospitality industry workers have been identified as high-risk groups.

Researchers in the United States have calculated that typical levels of airborne nicotine in public places range from 1 to 100 micrograms per cubic meter of air (μ g/m³). Exposure to an average of 7.5 μ g/m³ of nicotine for 40 years corresponds to a probability for passive-smoking-induced mortality of 1 per 1000 from lung cancer and 1 per 100 from heart disease. This is considerably higher than the United States Environmental Protection Agency (USEPA)–recommended 'acceptable' risk level of 1 per 1,000,000 for environmental carcinogens and toxins in air, water and food.

Public health risk

Investigation of the potential health risk of ETS highlighted its seriousness because of the large numbers of people affected combined with the high individual risk to health from ETS exposure. Furthermore, there is no evidence of a safe exposure level and there are no national or international exposure standards.

Public opinion

Public opinion on passive smoking has been examined in some detail. Studies have consistently shown that more than 75% of Australians believe that passive smoking causes ill health. The majority of Australians are aware of the health risks of passive smoking and supportive of measures to control ETS exposure in enclosed public places and workplaces. While there is an increasing trend for smokefree workplace policies there are anomalies in the provision of non-smoking areas particularly in the hospitality industry. One reason for the restricted spread of smokefree policies in the hospitality industry is that many hospitality industry proprietors are concerned about the loss of custom, which they believe would be associated with the provision of non-smoking areas.

Current ETS management strategies

Whilst there are a range of current ETS management strategies, including personal avoidance, individual company restrictions on workplace smoking, local incentive schemes and occupational health and safety considerations, these are inconsistent and ineffective in the widespread prevention of ETS exposure. Surveys have shown that businesses would prefer a legislative 'level playing field'. They indicate that legislation would be an effective and publicly well-supported way, of reducing the public health risk of ETS exposure. This indicates that jurisdictions might need to consider the role of legislation in fully addressing the issues of passive smoking.

1 Introduction

1.1 Passive smoking: a national response

The National Public Health Partnership (NPHP), which embodies a commitment by Commonwealth State and Territory governments to work together to address public health issues, includes in its memorandum of understanding the principles that:

- each community or population subgroup should have access to strategies, services and activities which optimise their health;
- each community or population subgroup should have access to a healthy and safe environment, including clean air and water, and adequate food and housing; and
- a supportive legal and political environment is integral to the public health effort.

A national response to passive smoking was foreshadowed by the Minister for Health and Aged Care Dr. Michael Wooldridge when he announced, in response to the 1997 National Health and Medical Research Council's scientific information paper on passive smoking, that passive smoking was a major public health issue warranting a national response.

In response to the Minister's announcement and considering the principles of the NPHP memorandum of understanding, the Legislative Reform Working Group (LRWG) of the NPHP has developed a national response to passive smoking in enclosed public places and workplaces. This component of the national response to passive smoking is intended to provide a suite of resources that facilitates best practice legislation in this area. A second component of the national response will focus on the issue of children's exposure to environmental tobacco smoke.

The development of the national response is consistent with the Commonwealth Government's response to the Report of the Senate Community Affairs Committee (CDHFS 1997), in which the Commonwealth Government agreed to work with State and Territory governments to develop guidelines addressing passive smoking and to promote best practice legislation.

The first component of the national response provides guidelines for jurisdictions considering legislation to restrict ETS in enclosed public places and workplaces, or for jurisdictions wishing to assess the scope and effectiveness of existing legislation. This component supports the *National Tobacco Strategy 1999 to 2002–03* (CDHAC 1999), which provides a framework for national action in

a number of key areas. The strategy highlights the need to reduce people's exposure to environmental tobacco smoke (ETS) as a key area for action and includes as a major objective the establishment of smokefree environments as 'the norm'.

The national response to ETS in enclosed public places and workplaces is in four parts:

- this background paper (National Response to Passive Smoking in Enclosed Public Places and Workplaces: A Background Paper);
- a statement of the guiding principles for developing legislation (*Guiding Principles for Smoke-Free Public Places and Workplaces Legislation*);
- examples of core provisions (Smoke-Free Public Places Legislation: Examples of Core Provisions); and
- Reference Material for Regulation Impact Analysis.

1.2 Aim of this background paper

This background paper complements the other documents in the first component of the national response (ETS in enclosed public places and workplaces) by providing an analysis of the impact of passive smoking on public health. The approach addresses both governmental responsibilities for public ETS exposure¹ and community education to support ETS protection.

The context for this approach is presented by considering:

- definition of passive smoking and composition of ETS;
- the health effects of ETS;
- exposure levels;
- public health risk of ETS;
- public opinion on passive smoking; and
- current ETS management strategies.

With this information, jurisdictions will be able to continue to develop their own responses to the issue of passive smoking, by adapting the national response to local circumstances and systems as they see fit. This approach would also facilitate jurisdictional action in undertaking a key strategy for the National Tobacco Strategy 1999– 2002–03. It is noted, however, that by endorsing the national response, jurisdictions do not commit to implementing any of its recommendations.

^{1 &#}x27;Governments have a responsibility to legislate to control exposure to tobacco smoke in public spaces' (WHO 1999).

1.3 Why now?

Scientific reports on the association between passive smoking and respiratory disease in children began to appear in the 1970s. Public awareness grew during the early 1980s as more studies were published, which linked ETS exposure and disease. A majority of Australians indicated that they supported restrictions on smoking in enclosed public places and workplaces. By the mid-1980s, passive smoking was known to be a health risk for nonsmokers, causing respiratory disease and lung cancer.

Major health and medical authorities called for people to be protected from ETS in enclosed public places and workplaces. For example, both the United States Surgeon General (US Dept of Health and Human Services 1986) and the Australian National Health and Medical Research Council (NHMRC 1987) considered that legislation should be introduced restricting smoking in workplaces and enclosed public places.

The first laws restricting smoking to protect nonsmokers' health were enacted in the early and mid-1970s in the states of Arizona and Minnesota in the United States. Since then, the number and strength of such laws have increased. The Australian Senate Community Affairs References Committee report (1995) recommended that smoking be prohibited in enclosed public places. This included office, factory, shop or other worksites, shopping centres, restaurants, theatres, hotels and sporting venues.

Despite progress since the 1980s, there are anomalies in the provision of nonsmoking areas. Concerns over loss of trade have restricted the spread of smokefree policies in the hospitality industry. Smokefree workplace policies have had a greater impact in large organisations, in the public sector and in white-collar workplaces than in other types of workplaces.

Since the first NHMRC report (1987) on passive smoking and health, a body of evidence has emerged about the health effects of, and the means of reducing, ETS exposure. For example, the World Health Organization (WHO) highlights the 'real and substantial threat to child health' posed by ETS exposure and urges governments to take legislative action to help prevent this (WHO 1999). This has also been a time of extensive public education and debate, with strengthening public support for smokefree environments. In 1994, the Commonwealth, State and Territory ministers for health agreed to target the year 2000 for enclosed public places to become smokefree. They recommended that State and Territory governments introduce legislation to achieve this (CDHSH 1994). In 1997, Dr Michael Wooldridge, the Minister for Health and Aged Care, announced that passive smoking was a major public health issue in need of a national response (Wooldridge 1997). The National Tobacco Strategy (NTS) endorsed by the Ministerial Council on Drug Strategy and released in June 1999 identified reducing exposure to ETS through establishing smoke free public places as the norm as a key strategy.

2 Passive Smoking and Environmental Tobacco Smoke

2.1 What is passive smoking?

Passive smoking is the term used for exposure to tobacco smoke, or the chemicals in tobacco smoke, without actually smoking. It usually refers to a situation where a nonsmoker breathes smoke emitted into the environment by other people smoking. This smoke is known as 'environmental tobacco smoke' (ETS).

2.2 What is environmental tobacco smoke?

ETS consists of two types of smoke: smoke from the burning cigarette (*sidestream smoke*) and smoke exhaled by active smokers (*exhaled mainstream smoke*). Sidestream smoke accounts for most of the tobacco smoke found in a room in which people have been smoking. Although the chemical composition of ETS differs from the smoke inhaled by active smokers, both contain a similar range of substances with known toxic and carcinogenic effects (US Department of Health and Human Services 1986).

Tobacco smoke contains over 4000 compounds of which approximately 60 are known or suspected carcinogens. Of these, at least 50 are present as particles of 0.01–1.0 microgram in size (NHMRC 1997), and the remainder are in a gaseous or vapour phase (Witschi et al 1997). As ETS 'ages' with time, constituents of the particle phase shift to the vapour phase (WHO in press).

Sidestream smoke is produced at lower temperatures than mainstream smoke and contains higher levels of tobacco burned per milligram, and also more of the following compounds (NHMRC 1997, Winstanley et al 1995).

- Noncarcinogenic compounds:
 - ammonia
 - carbon monoxide
 - nicotine
- Carcinogenic compounds:
 - benzene, benz(a)anthracene and benzopyrene (polynuclear aromatic hydrocarbons)
 - 2-napthylamine, 2-naphthylamine, 4-aminobiphenyl (aromatic amines)
 - *N*-nitrosamine
 - nickel
 - polonium-210

2.3 Environmental tobacco smoke as an indoor air pollutant

Indoor air pollution is now considered to be the major source of exposure and risk for many airborne contaminants (Brown 1997, Miller et al 1998). ETS is recognised as a significant component of the toxic air contaminants that people are exposed to indoors.

This has been highlighted by a number of national and international bodies.

- The United Kingdom Building Research Establishment identified ETS as one of the most significant health risks in commercial buildings (after radon, which is not a significant public health problem in Australia) (Raw and Hamilton 1995).
- The United States Environmental Protection Agency (USEPA) found that ETS is the largest source of particulate indoor air pollution, and is the major combustion source for human exposure to mutagens and carcinogens (Lewtas 1990).
- The California Environmental Protection Agency concluded that ETS is an important source of exposure to toxic air contaminants indoors (California Environmental Protection Agency 1997).
- Australia's Environmental Health Strategy, Environmental Health in Australia: Towards a National Strategy (CDHFS 1998), noted that most Australians spend about 90% of their time indoors, and are exposed to serious indoor pollutants including ETS.

There is little scientific information about the effects of ETS exposure in confined outdoor settings, but exposed individuals, particularly those with asthma and other preexisting ailments and conditions, have reported detrimental effects on their health from such exposure.²

² The following explanation of the behaviour of tobacco smoke in the outdoor environment has been provided by James Repace: 'If a smoke plume is significantly hotter than the surrounding air, the plume will rise; however, if the plume has a small cross-section, as for the smoke from a cigarette, it will rapidly cool and lose its upward momentum and then will tend to subside as the combustion gases are heavier than air. Thus, in the case of no wind, the cigarette plume will rise to a certain height and then descend. Where there is wind, the amount of thermally induced plume rise is inversely proportional to the wind velocity – doubling the wind velocity will halve the plume rise. (Repace, Repace Associates Inc., MD, USA, pers comm 1997)

3 Environmental tobacco smoke: effects on health

3.1 Adverse health effects

There is consistent evidence that breathing ETS by nonsmokers can cause bronchitis, pneumonia and other chest illnesses in children, and can increase the risk of cardiovascular disease, lung cancer and other lung diseases in adults (see Section 3.2). The evidence for the harmful effect of ETS exposure in children is particularly strong and children are exposed to ETS in a range of indoor environments, including public places. However, the present discussion focuses mainly on adult exposure in enclosed public places and workplaces.

As well as these serious health effects, ETS also causes 'irritant' effects on the eyes, nose, throat and airways (Winstanley et al 1995). These effects occur after short-term ETS exposure and are due to irritant chemicals in tobacco smoke, such as acrolein, acetaldehyde, formaldehyde, sulfur and nitrogen oxides, ammonia and other volatile organic compounds.

While otherwise healthy adults may experience detrimental effects on ETS exposure, such exposure may have particular significance for people with preexisting conditions.³ For example, people whose respiratory or cardiovascular systems are already compromised (for example, people with chronic bronchitis or ischaemic heart disease) may experience a critical deterioration in function and symptoms as a result of exposure to ETS (NHMRC 1997).

3.2 Overview of the evidence

3.2.1 Introduction

Research evidence for a causal relationship between 'active' smoking and lung cancer dates from the early 1960s. Extensive research over the following 25 years confirmed that smoking affects virtually every organ system. In 1990, the US Surgeon General concluded that 'smoking represents the most extensively documented cause of disease ever investigated in the history of biomedical research' (Davis 1997).

3 The Human Rights and Equal Opportunity Commission (1997) determined that failure to provide an environment free of tobacco smoke for a person with a respiratory disability constituted a breach of the *Disability Discrimination Act 1992* and has stated that 'The capacity for all Australians, with or without a disability, to participate as far as possible in all aspects of community life must be the paramount consideration.' Despite this, substantial research into passive smoking, both by the medical research community and the tobacco companies, only started in the 1970s and the first major reports did not appear until the mid-1980s. This research had the potential to powerfully redefine smoking as a public health issue: if smoking affected nonsmokers as well as smokers, then it could no longer be said that the health issues associated with smoking were exclusively concerns for individual smokers.⁴

3.2.2 Reports of health and medical organisations

Various international reports, 1986

In the mid-1980s, five major reports from authoritative health and medical organisations in the United States, France, Australia and the United Kingdom were published. They all concluded that passive smoking was a cause of disease, including lung cancer, in nonsmokers (US Department of Health and Human Services 1986, US National Research Council 1986, IARC 1986, NHMRC 1987, UK Independent Scientific Committee on Smoking and Health 1988).

United States Environmental Protection Agency, 1992

In 1992, the USEPA published a report on ETS and respiratory disease. By this time, 30 epidemiological studies on ETS and lung cancer had been published, with 24 showing a positive association. The USEPA concluded that the widespread exposure of the population to ETS represented a serious and substantial threat to public health. The USEPA also classified ETS as a 'Class A' human carcinogen, a category that includes other toxic substances such as radon, benzene and asbestos.

California Environmental Protection Agency, 1997

In 1997, a comprehensive report on the health effects of ETS exposure was published by the California Environmental Protection Agency. It agreed with the findings of the USEPA report on the link between ETS exposure, lung cancer and respiratory illness. The report concluded that passive smoking is a cause of heart disease mortality, acute and chronic heart disease morbidity, slowing of

⁴ Concerns about the health effects of ETS generally focus on nonsmokers, although active smokers are also exposed to ETS. However, the added risk to smokers from passive smoking is expected to be relatively small compared to their risk from active smoking (WHO 1999).

fetal growth, sudden infant death, nasal sinus cancer and induction of asthma in children (summarised by Davis 1997).

Wolfson Institute of Preventive Medicine (United Kingdom), 1997

In 1997, two substantial reviews of the scientific data were published from the Wolfson Institute of Preventive Medicine in London, United Kingdom (Hackshaw et al 1997, Law et al 1997). These studies found that, compared with control subjects, nonsmokers married to smokers had a 26% increased risk of lung cancer and a 23% increased risk of ischaemic heart disease. Low-dose exposure to ETS was found to increase the risk of ischaemic heart disease, which may be due to the sensitivity of the body's blood clotting system.

Australian National Health and Medical Research Council (1997)

In late 1997, the Australian NHMRC issued a scientific information paper on *The Health Effects of Passive Smoking*, based on information available up to 25 June 1997. Based on the evidence presented in the report, the NHMRC concluded that passive smoking is a danger to the health of the whole community. The main areas of concern were: asthma and other respiratory illnesses in children; effects on the unborn foetus; association with sudden infant death syndrome; and lung cancer and heart disease in adults. The key findings of this report are summarised as follows.

Children exposed to ETS are 40% more likely to suffer from asthma symptoms than children who are not exposed. An estimated 8% of childhood asthma in Australia is attributable to passive smoking. Passive smoking is estimated to contribute to the symptoms of asthma in 46,500 Australian children a year.

Children exposed to ETS during the first 18 months of life have a 60% increase in the risk of developing lower respiratory illnesses such as croup, bronchitis, bronchiolitis and pneumonia. An estimated 13% of lower respiratory illness in Australian children under 18 months of age (16,300 cases per year) is attributable to passive smoking.

Never-smokers who live with a smoker have a 30% increased risk of developing lung cancer. (This does not take into account other sources of ETS exposure, such as work and social settings.)

The risk of heart attack or death from coronary heart disease is about 24% higher in never-smokers who live with a smoker. (This does not take into account other sources of ETS exposure, such as work and social settings.) Passive smoking contributes significantly to the risk of sudden infant death syndrome and may increase the risk of death from all causes.

The NHMRC highlighted several areas in which more research was needed, including the incidence and prevalence of conditions associated with passive smoking (including acute irritant effects), and patterns of exposure to ETS, particularly outside the home and where vulnerable groups within the population may be involved.

The NHMRC acknowledged its reliance on studies of ETS exposure in domestic settings but argued that the available information was relevant for the purposes of policy development. They argued that decisions on the control of ETS in workplaces and confined public places should be based largely on what is known about the health effects of ETS in homes, because:

...studies of lung cancer in non-smoking women exposed to either smoking or non-smoking spouses are likely to provide better evidence with which to assess the causal relationship between lung cancer and exposure to ETS than studies of workplace exposure. This is because more is known about risks to health in the home setting. Moreover, the assessment of exposure to ETS in domestic studies is considered to be a better and more consistent measure than information available from workplace studies. Exposure to ETS at work is likely to vary considerably between workplaces and over time. (NHMRC 1997)

The NHMRC noted that few studies have attempted to separate risks to health from domestic and nondomestic sources of ETS. The characteristics of ETS vary with factors such as ventilation and absorption by internal surfaces. However, the NHMRC found that the nature of the hazard is likely to be similar in all enclosed settings. The available evidence led the NHMRC to conclude that the total population dose resulting from nondomestic exposure to ETS is likely to be similar to that resulting from exposures occurring in the home (NHMRC 1997).

Then NHMRC also noted that studies of natural exposures in human populations can seldom be conducted in the tightly controlled manner of laboratory research. Hence, it is always possible to propose alternative, noncausal explanations for associations between disease and possible risk factors. The NHMRC argued that we should not postpone indefinitely making a conclusion about the possible hazards of passive smoking, since scientific evidence is always incomplete. The same process applies to the management of every other factor in the environment suspected to be hazardous to health.

The NHMRC acknowledged that their findings reflect a conservative approach to the extent and impact of illness

likely to be due to passive smoking in Australia. The conclusions are more likely to underestimate the number of cases of illness attributable to ETS than to overestimate them.

United Kingdom Scientific Committee on Tobacco and Health, 1998

The report of the UK Scientific Committee on Tobacco and Health (UK Department of Health 1998) concluded that passive smoking causes lung cancer and ischaemic heart disease and is an important cause of childhood respiratory infection and chronic lung disease.

United States National Institute of Environmental Health Science, 1998

The National Toxicology Board of Scientific Counsellors of the United States National Institute of Environmental Health Science recommended that ETS should be listed as a known human carcinogen (reported by Reuters, 7 December 1998).

World Health Organization (in press)

The WHO has recently produced updated indoor air quality guidelines, following evaluation of the health risks of ETS exposure (WHO in press). This report summarises the health effects as follows:

ETS has been shown to increase the risks for a variety of health effects in non-smokers exposed at typical environmental levels. The pattern of health effects from ETS exposure produced in adult nonsmokers is consistent with the effects known to be associated with active cigarette smoking. Chronic exposures to ETS increase lung cancer mortality. In addition, the 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). ETS also irritates the eyes and respiratory tract. In infants and young children, ETS increases the risk of pneumonia, bronchitis, bronchiolitis, and fluid in the middle ear. In asthmatic children, ETS increases the severity and frequency of asthma attacks. Furthermore, as with active smoking, ETS reduces birth weight in the offspring of non-smoking mothers. In adults, there is strong suggestive evidence that ETS increases the mortality from sinonasal cancer. In infants, recent evidence suggests that ETS is a risk factor for sudden infant death syndrome.

Specifically in relation to the effects of ETS on children, the WHO found a strong consensus that ETS affects the developing respiratory system and causes an increased risk of:

 lower respiratory tract infections in infants and younger children;

- middle-ear effusion in young children;
- frequency and severity of asthma attacks in asthmatic children;
- irritation of the upper respiratory tract, particularly in infants and preschool children; and
- reduced lung function in children.

The WHO report also described a number of major cardiovascular effects of ETS exposure:

- decreased oxygen-carrying capacity of the blood, most likely attributable to carbon monoxide in ETS;
- increased platelet activation, which could increase the risk of atherosclerosis and thrombosis, and which is nonlinear with dose;
- endothelial damage, which is thought to be a preliminary step in the development of atherosclerosis;
- altered lipoprotein levels, which is associated with an increased risk of atherosclerosis; and
- increased thickening of the arterial walls, which is associated with the progression of atherosclerosis.

3.2.3 Other research studies

Since the publication of the NHMRC report in 1997, researchers have begun to look more broadly at ETS issues, including ETS exposure in work and social settings, effects of ETS on the cardiovascular system and the presence of tobacco carcinogens in exposed nonsmokers.

International Agency for Research on Cancer study (1998)

A multicentre study was conducted for the International Agency for Research on Cancer (IARC) – a WHO agency (Boffetta et al 1998). The study found an increase of 16% in the risk of lung cancer for nonsmoking spouses of smokers and a 17% increase in the risk of lung cancer associated with exposure to passive smoking in the work-place. For both exposures, there was a dose-response relationship and the risk of lung cancer increased with the amount of time spent in smoky environments. Workers who had spent up to 29 years in a smoky environment had a 15% greater chance of developing lung cancer, while people who had worked between 30–38 years in a smoky environment had a 26% greater risk of the disease.

The authors of the IARC study claimed that although the sample size for the study was small and the study on its own was insufficient to establish an association between passive smoking and lung cancer with 95% confidence (i.e. statistically significant), a link was detected that was consistent with many other studies and adds to the robust consensus that is developing in this area. Meta-analyses of many studies have revealed a statistically significant association. They also noted that when science is being used to inform decisions about policy – for example a ban on smoking in restaurants – the confidence demanded should reflect the circumstances in which a decision is

being made (Paolo Boffetta, Chief, Unit of Environmental Cancer Epidemiology, IARC: pers comm to Clive Bates, Director, Action on Smoking and Health, 10 April 1999).

Passive smoking and heart disease meta-analysis (He et al 1999)

He et al 1999, conducted a meta analysis of passive smoking and heart disease, with a total sample size of more than 600,000 people. The studies were weighted in terms of their strengths and weaknesses. The review found that passive smoking was consistently associated with an increased relative risk of coronary heart disease in cohort studies, in case-control studies, in men, in women, and in those exposed to smoking at home or in the workplace. A significant dose-response relationship was identified (risk ranging from 23% to 31% depending on the number of cigarettes per day to which the nonsmoker was exposed). Overall, the relative risk of coronary heart disease of exposed nonsmokers was 25%. This is consistent with the findings of a previous meta-analysis that showed that the effect of ETS exposure was to increase the risk of ischaemic heart disease by 23% (Law et al 1997).

This study looks at the effect of ETS on cardiovascular disease. The most widely accepted explanation is that the large effect seen from low-dose exposure to ETS may be attributed to platelet aggregation (ie a thickening of the blood is stimulated by low exposures to ETS). Researchers at the Wolfson Institute of Preventive Medicine, London, UK reached similar conclusions to those in this study (Law et al 1997) and noted that nonlinearity of heart disease risk also characterises active smoking. For example, people who actively smoke just five cigarettes a day increase their risk of heart disease by 40%, whereas people who smoke four times as many cigarettes only double their risk of heart disease (interview with Malcolm Law, 'Smoke gets in your eyes', *New Scientist* 29 May 1999).

The explanation given by the UK Department of Health (1998) is that the main causal factor appears to be an increase in platelet aggregation (a major step in the formation of thrombi), which may occlude in the arteries. The dose–response relationship between ETS exposure and platelet aggregation is nonlinear and is consistent with result from other studies of the effects of tobacco smoke on platelet aggregation.

Other key findings in ETS research

Recent information on ETS and health, from both primary research and meta-analyses, includes the following key findings:

Passive, as well as, active smoking has a significant effect on lung growth in adolescents. This effect is dose-related (Bono et al 1998).

- ETS exposure in the workplace results in an increased lung cancer risk similar to that resulting from household exposure (Wells 1998b, Brown 1999, Reynolds 1999, Kreuzer et al 2000, Zhong et al 2000). Furthermore, lung cancer risk may be seriously underestimated where exposure, other than that studied, exists (Johnson and Repace in press).
- Working in a smoke-free bar or tavern is associated with a rapid improvement of the respiratory health of bar workers (Eisner et al 1998). There is a significant correlation betweens children's ETS exposure and upper respiratory tract infections, otitis media, asthma, asthma wheeze or wheeze (Benninger 1999,Gryczynska et al 1999, Lister and Jorm 1998, Wahlgren et al 2000, Young et al 2000).
- Children exposed to ETS show a cellular infiltration into the nasal mucosa that resembles an allergic response (Vinke et al 1999). This effect may predispose the children to allergic sensitisation of the airways.
- Chronic exposure to ETS doubles the risk of stroke, based on exposure to spousal smoking (You et al 1999, Bonita et al 1999).
- ETS exposure is associated with a 20% increase in the progression of atherosclerosis. Current smokers have a 50% increase and past smokers have a 25% increase. Some of these effects may be cumulative and irreversible (Howard et al 1998b).
- In women exposed to workplace ETS, those in the top third of exposure levels have an increased risk of low blood levels of high-density lipoprotein-cholesterol (HDL-C)(Mizoue et al 1999). Since HDL-C is a preventive factor for coronary heart disease, this indicates that ETS exposure may increase the risk of coronary heart disease.
- Children born to women exposed to ETS in pregnancy have a higher risk of developing central nervous system tumours after birth (Filippini et al 2000).
- Overall studies showed an association between exposures to ETS during pregnancy with low birth weight and concluded that there is consistent evidence that ETS exposure in pregnancy is associated with adverse pregnancy outcome (Misra and Nguyen 1999). One study, (Sadler et al 1999) showed that exposure to ETS during pregnancy had no significant association with low birth weight babies.).
- Specific language impairments in children do not correlate with levels of ETS exposure (Tomblin et al 1998).

Small doses of ETS can have large effects on arterial function, which helps to explain the large effects, relative to dose, of ETS exposure on the risk of heart disease. Ending ETS exposure is important if this dysfunction is to be corrected. The maximum improvement in vascular function was observed more than two years after cessation of passive smoking, suggesting only partial reversibility of passive smoking-related arterial injury. This has important implications in terms of disease (Raitakari et al 1999).

Studies over the past 15–20 years have resulted in broadly consistent findings about the effects of ETS exposure on health. As researchers have acknowledged, studies of passive smoking are necessarily conducted under less than ideal conditions, as, for example, no methods are available to quantify all aspects of previous exposure to tobacco smoke in a comprehensive fashion. Direct experimental studies are logistically impossible because cessation of passive smoking at home or in the workplace often cannot be controlled voluntarily by the exposed person (Raitakari et al 1999).

4 ETS exposure in public places and workplaces

4.1 Factors affecting exposure

The proportion of total exposure to ETS that occurs in public places and at work differs between populations. Within any country, this fraction also varies over time, as smoking patterns change and smokefree policies take effect. The NHMRC report (1997) estimated that, in recent years in Australia, workplace exposure has been equivalent to, or greater than, home exposure. Similar assessments have been made in the United States (Repace and Lowrey 1985, Fontham et al 1991, USEPA 1992, Fontham et al 1994, Wells 1998b).

There are two groups of people who are exposed to ETS in public places and workplaces: employees and customers. In occupational settings, the harm from passive smoking depends on the time spent in that environment and the concentration of ETS in the airspace, which is in turn affected by the size of the space, the number of cigarettes smoked there in a given time and the ventilation rate. In certain occupational settings, these factors combine to create a substantial risk of harm from ETS (Davis 1998).

A meta-analysis, which looked at relative risks for heart disease from passive smoking in the home and in the workplace (and which took account of methodological problems in the original studies), concluded that the risks for heart disease from passive smoking at work are roughly equal to those from home-based exposure (Wells 1998a).

A study of more than 32,000 nonsmoking women found that women exposed to ETS at home or at work had a 71% increased risk of ischaemic heart disease; the risk increased to 91% for those with regular exposure (Kawachi et al 1997). A large population-based case-control study that examined the effect of ETS exposure on nonsmoking women aged 20–79 found a statistically significant lung cancer risk associated with exposure to ETS in work and social settings (Fontham et al 1991, Fontham et al 1994).

Air sampling studies at worksites with and without smoking policies found that occupational exposure to ETS presents a substantial risk to workers where there is no policy restricting or banning smoking (Hammond et al 1995). Using biological markers, researchers have found levels of nicotine in the hair of nonsmoking hospitality industry workers which were relative to ETS exposure (Dimich-Ward et al 1997).

4.2 Measurement of exposure

Because ETS is a dynamic, complex mixture of thousands of compounds, in particulate and vapour phases, it is difficult to measure exposure. Instead, various markers are used to quantify environmental exposure (WHO in press). Measurement of exposure to ETS presents particular difficulties, as noted in the discussion document for Australia's National Environmental Health Strategy:

...there is no absolute standard by which to measure exposure to ETS: biomarkers such as urinary cotinine provide precise measures, but quantify only one facet of exposure to smoke, and it is not yet known which constituents of ETS are most important in the aetiology of disease. Direct environmental measurement and personal monitoring are being used increasingly and may in the future provide accurate and relevant data on exposure to ETS suitable for use in aetiological studies.' (CDHFS 1998)

Researchers have also pointed out that the statistically significant health risks to nonsmokers from ETS exposure may appear smaller than they are because it is difficult to find a truly unexposed control group. This is because a lung cancer appearing in 1990 is the product of decades of exposure and it is difficult to find a pristine group of nonsmokers who are unexposed to second-hand smoke. Discriminating between exposed and unexposed subjects and estimating past exposures is also difficult (Johnson and Repace in press), and may lead to a serious underestimation of the risk of lung cancer.

The NHMRC (1997) found that these difficulties have implications for the ability to assess exposure of a general population. Unlike shared environmental exposures, such as urban air pollution, drinking-water lead levels and latitude-dependent ultraviolet irradiation, ETS is predominantly a 'personal environmental' exposure. Its major components include exposures in the home, recreational settings and in the workplace. To date, there has been no attempt at a standardised national survey using large representative samples.

4.2.1 Biomarkers

ETS exposure has been measured by a variety of biological markers. Tests of breath (for carbon monoxide), the hair (for nicotine) and the saliva, blood and urine (for cotinine, a nicotine derivative) have verified the presence of tobacco smoke chemicals in the bodies of nonsmokers exposed to ETS (WHO in press), including metabolites of three major classes of carcinogens in tobacco smoke (NHMRC 1997):

- polynuclear aromatic hydrocarbons (eg benzene)
- aromatic amines (eg 2-napthylamine)
- nitrosamines (eg N-nitrosamine)

Such studies have shown levels of these biological markers in nonsmokers that are 30% of those of active smokers (Dimich-Ward et al 1997, Eliopoulos et al 1994). Urinary cotinine, in particular, is a very reliable marker, with a consistent linear relationship to degree of ETS exposure (Matt et al 1999, Lindgren et al 1999).

Lung cancer and heart disease have been associated with cotinine levels in nonsmokers as well as with passive smoking in the workplace. Therefore, air nicotine and cotinine in body fluids appear to be useful markers for estimating ETS exposure and dose and for assessing nonsmokers' risk of heart and lung mortality from passive smoking the workplace (Repace et al 1998).

In one recent study, blood samples from volunteers in control groups and groups exposed to ETS in public places were analysed for plasma cotinine (Howard et al 1998). The exposed group (who were not exposed to ETS at home) had cotinine levels 65% higher than that found in the control group. The study also found a marked increase in DNA damage in the exposed group. The authors concluded that exposure to ETS in the workplace caused an increase in oxidative DNA damage and hence an increased risk for the development of a number of diseases.

4.2.2 Personal monitoring

Covance Laboratories in the United Kingdom conducted recent studies of personal exposure levels, for the Centre for Indoor Air Research in the United States (which is financially supported by the tobacco industry). ETS exposure was examined for randomly selected volunteer nonsmokers in urban centres in Europe using personal air monitors for 24 hours (Phillips et al 1998ab). In one group more than half the sample were not exposed to ETS at home or at work. Salivary cotinine was measured to check that the subjects were not smokers.

The results suggested that nonsmokers' exposure to ETS was about 0.1% that of a smoker, or about 6 cigarettes per year. These findings were at variance with biological measurements of ETS exposure. Problems have also been identified with the 'cigarette equivalents' approach to assessing exposure. As the NHMRC (1997) noted, estimates relating to the inhalation of tobacco smoke constituents are highly variable according to the particular substances considered and do not provide an adequate single index of total exposure to ETS. The 'cigarette equivalents' approach is said to have serious limitations

because it ignores differences in the relative proportions of various chemicals in sidestream and mainstream smoke, the physical nature (particulate versus vapour) of these chemicals, and breathing patterns of passive versus active smokers (NHMRC 1997). There are also unresolved questions about whether air monitors provide accurate readings of cumulative exposures and if they reflect how the body deals with low-level chronic exposure versus short-term acute exposures.

4.2.3 Direct environmental measurement

There have been many attempts to measure ETS levels in indoor air. Measurements of indoor air pollutants in the form of respirable suspended particles (RSP) indicate that tobacco smoke accounts for more than 80% of RSP (Ott et al 1996). However, ETS is a complex mixture of pollutants that changes rapidly with time, preventing most of its constituents from being used as indoor air quality indicators (Brown 1997). It is therefore difficult to find a suitable indicator for ETS in indoor environments, particularly where smoking is less heavy. There are some concerns that nicotine might underestimate, and RSP might overestimate, ETS levels where smoking is moderate to light (Brown 1997).

Following a review of methods used to estimate ETS exposure, Woodward and Al-Delaimy (1999) concluded that no current method gives a comprehensive picture. However, measurements of cotinine in conjunction with questionnaires may be suitable for epidemiological studies of disease aetiology.

Regardless of what methods are used to measure ETS exposure, there is no accepted standard that answers the question, 'How much is too much?' An exposure standard for ETS has not been determined by national or international authorities. Exposure standards are discussed further in Section 5.2.2.

4.3 High-risk settings

Workers in some industry sectors experience greater health risks because of higher levels of ETS exposure in their workplace. Airline flight attendants represent one high-risk group and the separation of smoking and nonsmoking in the aircraft cabin did not protect nonsmoking passengers or flight attendants from ETS exposure. Legal action brought by flight attendants in the United States was settled for US\$350 million.

Casino workers and hospitality industry workers have been identified as two other high-risk groups. Studies of ETS exposure in casinos have found that workers at nonsmoking tables were receiving ETS exposure levels similar to those received by employees working at tables where smoking was permitted (Trout et al 1998). A study of nonsmoking employees working in licensed premises in central New South Wales found that, after at least of four hours work, employees had four-times the carbon monoxide levels of workers in a smokefree workplace, and about one-third of the nonsmoking employees had carbon monoxide levels consistent with 'light smoker' status (Tutt and Harris 1990).

A British study of nonsmoking adults working in bars showed nicotine intakes equivalent to half a cigarette per day. Exposure to other smoke components could be higher (as in the case of some nitrosamines) or lower. These findings were based on salivary cotinine measurements taken from premises where doors and windows were open to provide natural ventilation (Jarvis et al 1992).

The recent prohibition on smoking in bars in California provided an opportunity for a before-and-after study of bar workers. Researchers found that 74% of bartenders initially reported respiratory symptoms when bartenders' median exposure was 28 hours per week. At follow-up, ETS exposure at work had declined to a median of two hours per week and 59% of the initial group no longer had symptoms. Of the 77% initially reporting sensory irritation symptoms, 78% had no symptoms at follow-up. Complete cessation of workplace ETS exposure was associated with the greatest improvements in respiratory health (Eisner et al 1998).

4.4 'Acceptable' level of exposure

Researchers in the United States have calculated that typical levels of airborne nicotine in public places range from 1 to 100 micrograms per cubic meter of air (μ g/m³). Exposure to an average of 7.5 μ g/m³ of nicotine for 40 years corresponds to a probability for passive-smoking-induced mortality of 1 per 1000 from lung cancer and 1 per 100 from heart disease (Repace et al 1998, Repace and Lowrey 1993, Johnson and Repace in press).

The USEPA defines 'acceptable' risk levels for environmental carcinogens and toxins in air, water and food as 1 per 1,000,000. The typical excess population risk generated by passive smoking range is about 2 per 1000 for lung cancer and about 2 per 100 for heart disease. This represents 200 times more than the acceptable risk level for lung cancer, and 2000 times the acceptable risk level for heart disease (Repace and Lowrey 1993).

Given that researchers believe that the risk of heart disease mortality from ETS exposure is about 10 times the risk of lung cancer mortality, analysis of cotinine levels has produced estimates that suggest that more than 95% of nonsmoking workers – including all workers in office workplaces with unrestricted smoking – would exceed the current United States 'significant risk' level due to passive smoking (Repace et al 1998, Johnson and Repace in press). Furthermore, focussing on either lung cancer or heart disease underestimates the risks of other adverse effects of ETS on health. The significance of the public health risk associated with ETS exposure is discussed further in Section 5.

5 The public health risk of ETS

5.1 Is ETS a risk to public health?

When assessing a public health risk, the following three factors are important:

- the risk of an adverse health effect occurring;
- the seriousness of the health risk; and
- the number of people likely to be exposed to ETS (and the ability of people to avoid exposure).

5.1.1 The risk of an adverse health effect occurring

Based on the amount and consistency of evidence over the past 15 years, health and medical authorities have concluded that indoor exposure to ETS is a cause of lung cancer, heart disease and other serious illnesses in otherwise healthy nonsmokers (see Section 3). The effects are more serious for children and people with pre-existing health conditions (WHO 1999).

The significance of the risk to public health is due to the large number of people exposed to ETS, and to the fact that there is no evidence of a safe exposure level (Davis 1998, WHO in press). In comparable situations of health risk, action is commonly taken to limit exposure where the risk for an individual is low, but where large numbers of people are exposed. Although attention has focused on the risk of lung cancer and heart disease, the impact of ETS on health includes the risk of initiation or exacerbation of many other symptoms of ill health that affect significant numbers of people.

The WHO has observed that 'Because of the extensive prevalence of ETS exposure and the high incidence of some of the detrimental health effects associated with ETS exposure (such as heart disease in adults and lower respiratory tract infections in children), even small increases in relative risks can translate into substantial mortality and morbidity on a population basis' (WHO in press).

5.1.2 The seriousness of the health effects caused by ETS

ETS exposure is a serious public health risk. A large proportion of people who are exposed to ETS show some symptoms of ill health. For some people, exposure can lead to life-threatening illness such as lung cancer and heart disease. For a significant proportion of the population who experience cumulative exposures to ETS, there is a measurable increase in the likelihood of ill health over the longer term.

5.1.3 The number of people exposed to ETS

There is widespread exposure to ETS in enclosed public places and workplaces. These exposures are often involuntary. Nonsmokers may also be unaware of ETS exposure. For example, a 1996 study that examined the extent of ETS exposure in a large sample of the United States population (Pirkle et al 1996), found that 88% of nonsmokers had measurable exposure to ETS (based on the level of cotinine, a metabolite of nicotine, found in the blood), but only 37% reported ETS exposure.

5.1.4 The ability of people to avoid ETS exposure

It has been argued that ETS exposure may, in some circumstances, be voluntary. However, this view is inconsistent with accepted approaches to other public health issues relating to voluntary activities for which the community expects protection. The public expects that there will be systems in place to prevent contamination of the environment where there is the potential for widespread exposure to a health risk that may cause serious effects.

The assertion that people can avoid ETS exposure because they are aware of its presence is not entirely substantiated, and there is also the possibility that nonsmokers may not always be aware of ETS exposure. On many occasions, exposures to ETS are involuntary. Examples include children's exposure, exposures in workplaces, and exposures where there is no realistic alternative without incurring a high social or professional cost. In social and family situations, the onus is typically on the nonsmoker to ask the smoker to refrain from smoking or to ask the group to sit in a nonsmoking area. Despite not wanting to breathe other people's smoke, many nonsmokers feel unable to do this.

During the past decade, there have been gradual shifts in public attitudes about whether the smoker or the nonsmoker is responsible for nonsmokers' avoidance of ETS. When information about the health consequences of ETS exposure first came to light, health and medical authorities advised that nonsmokers should avoid ETS exposure. While this was important advice, it placed the onus on nonsmokers to ask for special provision and did not alter the situation of smoking as the 'norm'. It is now accepted that the interests of public health are better served if nonsmoking is taken as the 'norm' and special provision is sought by people who wish to smoke, rather than those who do not.

5.2 Reducing the risk of ETS to public health

5.2.1 Assessing public health risks

There is no agreed formula for determining what is a risk to public health (Reynolds 1998). In order to determine a reasonable response to a probable risk, we must take into account the magnitude of the risk in terms of its seriousness and the number of people potentially affected, the likelihood of the risk occurring and the cost and difficulty of alleviating the risk.

The 'precautionary principle' is commonly applied to public health matters. This means that policy makers are justified in erring on the side of caution to limit activities where full scientific certainty does not exist. Thus, where there is possibility of harm, lack of complete scientific certainty cannot be used as a reason to postpone preventive measures. This principle gives rise to debate on various public health issues where the epidemiology shows a relatively small effect.

Applying the precautionary principle to passive smoking means that preventive action is justified. Failure to take such action would mean that large numbers of people continue to be exposed to the detrimental health effects of ETS. The accumulated evidence about the health effects of ETS strongly indicates that the elimination of ETS exposure in indoor public places is a valid public health objective.

5.2.2 Exposure standards

Health and environmental regulations in Australia are the responsibility of specific State and Territory government departments. Occupational exposure standards from Worksafe Australia are adopted by most State governments and some States develop specific regulations; for example, concerning the use of asbestos in buildings or in industrial processes (Brown 1997).

Although control of pollutant emission from sources is widely accepted as the optimum response for improving air quality, in comparison to regulation of outdoor air quality and industrial workplace air, indoor air quality concerns have not been systematically addressed, or regulatory actions taken, by any health or environment agency in Australia (Brown 1997).

While the National Environment Protection Council (NEPC) has set ambient (outdoor) air-quality standards, they have not set indoor air-quality standards. The NHMRC has developed goals for indoor air quality for formaldehyde and radon, but not for other pollutants. (CDHFS 1998).

Standards for mechanical ventilation such as AS1668.2,⁵ which rely on dilution ventilation, have been found to require impractically high ventilation rates or uneconomical filtration equipment to control ETS (Brown 1997). Such standards are engineering and design standards based on occupant 'comfort'.

The WHO, in its recent development of indoor air quality guidelines, concluded that, 'There is no evidence for a safe exposure level (of ETS)' (WHO in press).

⁵ Australian Standard AS 1668.2: 1991. Australian Standard for Mechanical Air Conditioning and Ventilation, Australian Standards, Sydney.

6 Public opinion on passive smoking

6.1 The importance of community attitudes

Although ETS protection is primarily an issue of public health, rather than customer preference, consideration of community attitudes is important for several reasons:

- if the provision of nonsmoking areas has not kept pace with community attitudes and preferences, this suggests that nonregulatory approaches have failed to meet community expectations;
- awareness of community attitudes can both ease concern about nonsmoking arrangements; and neutralise claims by lobby groups that such arrangements would be unpopular; and
- community attitudes, and their associated education and implementation strategies, are an important factor in the success of various policy options.

For example, following introduction of the *Smokefree Areas* (*Enclosed Public Places*) *Act 1994* (ACT), community support for nonsmoking in enclosed public places substantially contributed to widespread self-enforcement. In cases like this, legislation serves to validate the community norm (Jacobson and Wasserman 1997, Kagan and Skolnick 1993, Goodin 1995). Where support is more divided, but where protective health measures are required, understanding public attitudes is instrumental in successful community education.

6.2 Relationship between attitudes and behaviour

The relationship between public attitudes and the provision of nonsmoking areas in the community works both ways: attitudes change behaviour and vice versa. Community support facilitates the implementation of nonsmoking policies; the existence of these policies is associated with increased approval of smokefree provision (Wakefield et al 1999b, Pederson et al 1991). This association was reflected by National Drug Strategy Household Survey (NDSHS) results in 1993 (CDHHS 1993) and 1995 (AIHW 1996), which indicated that 8 in 10 workers in a workplace with a total smoking ban supported such restrictions, compared to fewer than 5 in 10 in a workplace with no restrictions. Workplace smoking bans, once implemented, can be expected to generate widespread support among workers (Makkai and McAllister 1998).

6.3 Public attitudes towards passive smoking

Surveys have consistently shown that more than 75% of Australians believe that passive smoking is a cause of ill health in nonsmokers (Table 1).

Other surveys have also found high levels of awareness of the health risks of passive smoking (Mullins et al 1992,

Survey statement Percentage of respondents that agree		l Source	
The health of nonsmokers can be damaged by other people's tobacco smoke	84% in NSW (1990), including:69% current smokers90% nonsmokers	Winstanley et al (1995)	
	 86% in Victoria (1991), including: 73% current smokers 92% never smokers 87% past smokers 		
Passive smoking is a 'high' or 'medium' health risk	78%	McAllister (1993)	
Other people's smoke affects nonsmokers' health	89%	Health Department of Western Australia (1998)	
Nonsmokers who live with smokers83%, including:might develop health problems because69% of smokers*of exposure to tobacco smoke85% of ex-smokers91% of nonsmokers		AIHW (1999)	

Table 1 Public assessment of passive smoking as a health risk

* Regular and occasional smokers

Wakefield et al 1996). Surveys by the Health Department of Western Australia (1998, p77) showed that at least 80% of people have been aware of the health risks of passive smoking since 1984.

6.4 Community support for ETS protection

A review of surveys from 1985–1995 (Makkai and McAllister 1998) shows that national public opinion on passive smoking and smoking restrictions was largely shaped in the late 1980s and early 1990s, when there was considerable publicity about passive smoking. This is consistent with data from Victoria, which show that the biggest change in attitudes about smoking at work occurred between 1988 and 1989. There was a further increase by 1990, with a doubling of the proportion of people supporting a total smoking ban (Mullins et al 1992).

Results from the 1998 NDSHS (AIHW 1999) shown in Table 2, are consistent with findings from other surveys and show strong, increasing support for prohibiting smoking in enclosed public places and workplaces.

Surveys in Western Australian have found a steady increase in support for smoking restrictions in restaurants and cafes since the mid-1980s (Health Department of Western Australia 1998, pp 90–91). The strength and consistency of community preference for nonsmoking in public social premises has been revealed in numerous surveys (Table 3). Hospitality industry employees held similar opinions to those of patrons (Trotter 1998).

The situation is different in outdoor dining areas. The majority of people prefer no restrictions on smoking in these areas, with nearly half of the respondents favouring smoking in restricted areas (Health Department of Western Australia 1998, p 93).

The Western Australian results for enclosed areas of hotel bars and taverns (Health Department of Western Australia 1998, pp 96–101) were broadly consistent with the 1998 NDSHS results for pubs and clubs (AIHW 1999). In

Western Australia, 48% of respondents supported there being no smoking at all in these areas, with another 26% supporting smoking being limited to separate areas with separate air conditioning. Support among smokers for making these premises smokefree (19%) was consistent with smokers' response nationally to the NDSHS question (17%). The Western Australian data indicate a substantial increase in the proportion of people supporting a smoking ban in these premises, from 15% in 1984 to 48% in 1997. Among smokers and nonsmokers, support for banning smoking and for limiting smoking to separate areas more than trebled during this time, and support for unrestricted smoking more than halved.

In 1998, the ACT Department of Health and Community Care commissioned a survey to gauge public attitudes in the lead-up to the implementation of smoking restrictions in licensed premises (ACT Department of Health and Community Care 1998), finding that:

the majority of smokers (85%) and nonsmokers (75%) patronise pubs and clubs;

- similar proportions of smokers and nonsmokers patronise large and small pubs and clubs;
- more than three-quarters of respondents approved of prohibiting or restricting smoking in pubs and clubs;
- 85% of current patrons would continue to patronise licensed premises if these were smokefree; and
- 38% of nonpatrons said a smokefree policy would encourage them to patronise these premises.

While restrictions on smoking are widely supported by the public, survey results suggest that support is more likely among nonsmokers, people with tertiary education qualifications, those employed in professional positions and those with young children (Makkai and McAllister 1998). When interpreting survey responses, it is important to acknowledge that the options presented in the survey and the specific wording of the questions can influence people's responses. In particular, people may indicate that they would prefer certain types of premises to be nonsmoking, without necessarily supporting legislation to this effect.

Table 2 National public attitudes toward smoking limitation

Survey statement	Percentage of respondents
Support, or strongly support, prohibition in enclosed shopping centres	81%
Support prohibition in restaurants	74%
Support prohibition in workplaces	76%
Support prohibition in pubs and clubs	44%, including 45% of regular drinkers

Source: AIHW 1999

6.5 Public opinion alone has not caused change

Since the 1970s, public opinion has indicated strong support for restricting smoking in enclosed public places and workplaces. This strong public opinion has not been matched, however, by actual nonsmoking arrangements made through either voluntary or regulatory means (WA Task Force 1997). In South Australia in 1997, a majority of people reported that they had been bothered by tobacco smoke when dining out (Wakefield et al 1999a). Despite strong public opinion, individuals may not always communicate their preferences, either due to uncertainty about their rights or fear of confrontation.

Restrictions on smoking are a particular problem in the hospitality industry. Many surveys have highlighted the disparity between the views of proprietors (Table 4) and the opinions and preferences of the public.

Other studies have shown that most pub and nightclub proprietors think that their patrons favour unrestricted smoking and that more than 75% of their patrons smoke (Semmonds et al 1995, Right Marketing 1997).

Public opinion	Percentage of respondents	Source
Want smoking restricted or prohibited	90%	Schofield et al (1993)
in restaurants	97%	National Heart Foundation (ACT) (1992)
	96%	Lee (1997)
	97%	Trotter (1998)
	95%	NSW Health Promotion Survey (1995)
	97%, including 94% of smokers	Health Department of Western Australia (1998, pp 87–92)
Want to dine in nonsmoking room	85% (nonsmokers) 33% (smokers)	Mullins (1991)
Want separate smoking areas in pubs	60%	Semmonds et al (1995)
Want smoking restricted or prohibited in pubs and clubs	82%	NSW Health Promotion Survey (1995)
Want smokefree bars in pubs and clubs	43%	WA Task Force (1997, Appendix 3)
Want totally smokefree pubs	10%	Semmonds et al (1995)
	44%	AIHW (1999)
	48%	Health Department of Western Australia (1998, pp 79–81)
Want smoking prohibited in indoor recreational and sporting venues	87%, including 79% of smokers	Health Department of Western Australia (1998, pp 79–81)
Expected the hospitality industry to be smokefree in five years	68%	WA Task Force (1997, Appendix 3)

Table 3 Public opinion concerning smoking in public social premises

Table 4 Actual provision of non-smoking areas in restaurants

Provision of non-smoking areas	Percentage of premises	Source
Either separate smoking and non-smoking areas or a total ban	<40% (national) 23%	Schofield et al (1993) Trotter (1998)
Totally smokefree	<2% (national)	Schofield et al (1993)
Either separate smoking and non-smoking areas or a total ban	24%	Northern Sydney Area Health Service (1994)

Views on providing non-smoking areas	Percentage of proprietors (premises with unrestricted smoking)	
Would cause loss of custom	50%*	
Would have no effect	31%*	
Not enough space	40%*	
Needs total government ban	50%*	
Total ban would cause loss of custom	50%*	
Would prefer consistent legislation	70%*	
Would support legislation prohibiting smoking	68%†	

Table 5 Restaurant proprietors' views on providing non-smoking areas

• Jones et al (1999), Turnbull et al (1996)

† QUIT Tasmania, 1996

Even where proprietors are aware of a demand for smoking restrictions in public social premises, various factors may prevent them from taking action (Schofield et al 1993). For example, in the Northern Sydney Area Health Service survey (1994) cited in Table 4, 62% of proprietors thought there was a demand for smokefree dining. Studies of restaurant proprietors (Table 5) have highlighted the various factors that deter proprietors from establishing nonsmoking areas. These results suggest that many of these factors could be overcome by a consistent legislative approach.

Studies of hospitality industry attitudes toward passive smoking for the National Heart Foundation NSW (Elliot Shanahan Research 1997ab) found that:

- understanding of the health effects of ETS varied widely;
- passive smoking was seen as merely unpleasant, not as a health issue;
- smoking was viewed as part of the ethos of attending their venues; and
- the risk of legal action was considered remote.

Survey responses from other areas (for example Jones et al 1999) also suggest that proprietors who believe that the industry will become smokefree as a result of government action are less likely to take action on their own. Conversely, those who believe that government action is unlikely to come about are unmotivated to introduce policies that they do not support. This mixed attitude suggests that consistent, widespread legislative action may be necessary to prevent the public health risk of ETS exposure in enclosed public places and workplaces.

To conclude, a number of recent studies have highlighted the factors that deter proprietors from taking action to establish nonsmoking areas in their premises. These studies also suggest that many of these barriers could be overcome by a legislative 'level playing field' to allay concerns of adverse affects on custom caused by smoking restrictions.

7 Current ETS management strategies

7.1 Personal avoidance of ETS

While public opinion alone is not enough to eliminate ETS exposure in enclosed public places and workplaces, the 1998 NDSHS showed that a majority of people are taking action to avoid places where they might be exposed to other people's smoke.

In answer to the question 'Do you avoid places where you may be exposed to other peoples' cigarette smoke?' respondents in all States and Territories replied that they did, as follows:

ACT	72%
New South Wales	69%
Victoria	65%
Queensland	65%
South Australia	66%
Western Australia	66%
Tasmania	63%
Northern Territory	63%

Nationally, the proportion of people saying they take such action increased from 59% to 66% between 1995 and 1998, including a quarter of smokers and a quarter of people who drink regularly.

7.2 Current nonlegislative approaches

Some of the most detailed information about workplace ETS exposure comes from a 1996 Tasmanian survey (Australian Bureau of Statistics 1996). This survey found that just over 9% of employed people were nonsmokers who worked where other people smoked. A 1998 survey of 2400 workers in Western Australia found that 60% of blue-collar workers, and 30% of white-collar workers, had no workplace ETS protection (Pryer 1998).

7.2.1 Public sector workplaces

The Commonwealth health department became completely nonsmoking in December 1986, and all Commonwealth government departments and buildings became smokefree by March 1988. The Commonwealth has also implemented smoking restrictions in a number of areas where it has the power to do so, including prohibiting smoking in: buses and coaches registered under the Federal Interstate Registration Scheme (1988); on domestic airlines flights and commuter services (1987); on domestic sectors of international flights (1990); on all flights of Australian carriers anywhere in the world; and on overseas carriers flying with Australia (1996). Following, or coinciding with, Commonwealth action, other public sector organisations also introduced smokefree workplace arrangements during the late 1980s and early 1990s. These included the Australian Broadcasting Corporation, the Australian Federal Police and a number of State and Territory public services.

7.2.2 Private sector workplaces

In 1991, a survey of 455 of Australia's top companies found that just under half operated a ban on smoking at work. Twenty-three per cent of companies had no policy on smoking (Richmond et al 1993). These results are consistent with other studies, which show that the rate of nonsmoking policy implementation has slowed considerably since the early 1990s (Wakefield et al 1999b, Borland et al 1997).

The Western Australian Task Force on Passive Smoking in Public Places (WA Task Force 1997) found a lack of information about policies in small businesses, even though these account for the majority of private sector employment. This report found that 63% of small businesses had a complete smokefree policy, with 11% having some restrictions and 26% with no policy. Among small business respondents who had not introduced a smokefree policy, 97% acknowledged the public health risk of ETS and the majority favoured legislation to control ETS exposure.

7.2.3 Other enclosed public places

There is a lack of information concerning ETS exposure in other enclosed public places which makes assessment of the situation difficult. However, considering the types of enclosed public places controlled by legislation in the ACT, South Australia, Western Australia, New South Wales and Victoria, these jurisdictions have accepted that ETS can pose a public health risk in the following situations:

- shops and shopping centres;
- professional and trade premises offering good or services to the public;
- restaurants, cafes and other eating areas;
- bars, nightclubs, cabarets, hotels, taverns, casinos and other licensed premises;
- educational facilities;
- community centres, halls, places of public meeting and clubrooms of sporting and community organisations;
- theatres, cinemas, libraries and galleries;
- sporting and recreational facilities;

- public transport waiting areas;
- common areas of multiunit residential facilities such as hostels and nursing homes; and
- common areas of short-stay facilities such as motels.

Many local councils, health authorities, health promotion and public health units have used a variety of initiatives to extend smokefree areas within their communities. Measures have included smokefree policies for premises under council control, conducting education and awareness programs, and promoting award and incentive schemes for restaurants that include nonsmoking dining areas. Incentive schemes normally include, in the qualifying criteria, the presence of nonsmoking dining areas. Although there has been little formal evaluation of these schemes, discussion with health officers indicates that restaurateurs are reluctant to introduce nonsmoking areas, even in response to these positive schemes.

The Tiwi Islands community of Milikapati has successfully introduced a smokefree policy for enclosed public places using incentive funding from the Northern Territory Health Services. This includes offices, meeting rooms, recreation rooms, shops, takeaway food premises and the women's centre. However, little further information is available about the extent of nonsmoking areas in Aboriginal and Torres Strait Islander communities.

In Western Australia, 55% of local councils had introduced comprehensive smokefree policies in all venues under council control, 37% had some smoking restrictions and 8% had no smoking policy (WA Task Force 1997). Councils reported high compliance levels with their no-smoking policies.

Some outdoor sporting venues have designated nonsmoking spectator seating areas. Some of these arrangements were a result of sponsorship and assistance from health agencies such as Healthway and QUIT, and other policies have been prompted by the results of customer surveys. Threatened legal action led the Melbourne Cricket Club to designate a nonsmoking seating area, and similar concerns led to the designation of nonsmoking seating at Waverley Park stadium.

Therefore, in the absence of legislation, the control of ETS exposure in enclosed public places appears to remain largely arbitrary.

7.3 Current legislative approaches

Laws that address public health issues are one component of an overall public health response. Tobacco control legislation is an example of public health law designed to work together with other approaches, forming an overall tobacco control strategy. During the debate in the early 1990s over the proposed passive smoking legislation in the ACT, editorials in the *Canberra Times* highlighted the government's role in protecting people's rights: 'nonsmokers have a right to go to restaurants and bars too, and need not have it insisted that it be entirely on other people's terms' (*Canberra Times*, 30 May 1992) and 'The Government does not need to cite the risks of disease from passive smoking to justify a wider ban on smoking in confined public places. The protection of people's comfort, especially that of people who find cigarette smoke highly irritating, is sufficient justification for widening the ban on smoking in enclosed public spaces' (*Canberra Times*, 18 February 1993).

While the implications of the law of nuisance and other remedies in civil law have not been fully explored in Australia, remedies that involve leaving redress to the civil law system are generally not as effective as legislation, which provides a public and statutory remedy to the problem.

Currently, three jurisdictions (ACT, Western Australia and South Australia) have enacted and implemented legislation to reduce ETS exposure in enclosed public places and workplaces. New South Wales has enacted, but not implemented, legislation.

The ACT was the first Australian jurisdiction to enact comprehensive legislation prohibiting smoking in a range of enclosed public places (*Smokefree Areas (Enclosed Public Places) Act 1994*). Coinciding with this legislation, a code of practice for smokefree workplaces was also introduced, in line with the *Occupational Health and Safety Act 1984* (ACT). All enclosed public places in the ACT are now completely nonsmoking except for a small number of exempt restaurants (where smoking is limited to no more than 25% of the dining area) and a larger number of licensed premises such as pubs and clubs (where smoking is limited to no more than 50% of the public area).

Following recommendations of the WA Task Force (1997), Western Australia enacted legislation that came into effect in March 1999: the Health (*Smoking in Enclosed Public Places*) *Regulations 1999*. This prohibits smoking in enclosed public places including shopping centres, indoor sporting venues, food halls and restaurants. Hotels, bars, cabarets, nightclubs and the casino are exempt. Staff smoking is prohibited in enclosed workplaces and employers cannot require employees to work in designated smoking areas.

Next, South Australia enacted legislation, with effect from January 1999, prohibiting smoking in enclosed public dining areas and with specific provisions for exemptions, relating to licensed premises (*Tobacco Products Regulation Act 1997*). A further amendment was made to the

legislation, effective April 1999, to extend the provisions to apply for an exemption to unlicensed premises. This legislation also prohibits smoking in auditoria and places of public entertainment (defined as buildings where seating is in rows) and represents an historical ban concerning fire safety in picture theatres.

Two surveys have evaluated the effects of the South Australian legislation (South Australian Department of Human Services 1999ab). Proprietors reported 98% compliance, with smoking restriction or prohibition in 94% of venues. Most proprietors were in favour of, or indifferent to, the legislation and found that very little effort or cost was required to implement it. The majority of both smoking and nonsmoking patrons agreed with the legislation and found that they enjoyed dining out equally, or more, following the smoking bans in enclosed public dining areas or cafes.

As noted previously, since drafting of this paper commenced, there have been further developments in other jurisdictions including Victoria, New South Wales, Tasmania and Queensland.

8 Conclusions

In this background paper, research into the health effects of ETS exposure has been reviewed, with a focus on exposure in enclosed public places and workplaces. This evidence indicates that ETS exposure in these environments represents a significant public health risk. From analysis of public opinion to passive smoking, it is clear that the general public is aware of the health risks of passive smoking and supportive of measures to restrict smoking in enclosed public places and workplaces. This demand for restrictive measures is not matched by the provision of such measures, in particular, in some sectors such as the hospitality industry.

The information contained in this paper clearly outlines the impact of passive smoking on public health and provides a rationale for the development of a legislative basis for the national response to passive smoking in enclosed public places and workplaces. Therefore, jurisdictions may need to consider further enactment and implementation of legislation to fully address the public health risk of ETS exposure.

References

ACT Department of Health and Community Care (1998). Licensed premises survey. Unpublished paper.

AIHW (Australian Institute of Health and Welfare) (1996). *1995 National Drug Strategy Household Survey.* AIHW, Canberra.

AIHW (Australian Institute of Health and Welfare) (1999). *1998 National Drug Strategy Household Survey: First Results.* AIHW (cat no. PHE 15), Canberra.

Anderson HR, Katsouyanni K and Spix C (1998). Reply to: Hasford B and Fruhmann G. Air pollution and daily admissions for chronic obstructive pulmonary disease in six European cities: results from the APHEA project. Air Pollution and Health, a European Approach. *European Respiratory Journal* 11:993

Australian Bureau of Statistics (1996). *Health Issues – Tasmania.* ABS Cat. No. 4396.6

Australian Senate, Community Affairs References Committee (1995). *The Tobacco Industry and the Costs of Tobacco-Related Illness.* Parliament House, Canberra

Bailar JC 3rd (1999). Passive smoking, coronary heart disease, and meta-analysis. *New England Journal of Medicine* 340: 958–959

Benninger MS (1999). The impact of cigarette smoking and environmental tobacco smoke on nasal and sinus disease: A review of the literature. *American Journal of Rhinology* 13:435–438.

Boffetta P, Agudo A, Ahrens W, Benhamou E et al (1998). Multicenter case-control study of exposure to environmental tobacco smoke and lung cancer in *Europe. Journal of the National Cancer Institute* 90:1440–1450.

Bonita R, Duncan J, Truelsen T, Jackson RT and Beaglehole R (1999). Passive smoking as well as active smoking increases the risk of acute stroke. *Tobacco Control* 8:156–160.

Bono R, Nebiolo F, Bugiani M, Meineri V, Scursatone E, Piccioni P, Caria E, Gilli G and Arossa W (1998). Effects of tobacco smoke exposure on lung growth in adolescents. *Journal of Exposure Analysis and Environmental Epidemiology* 8: 355–345.

Borland R, Morand M and Mullins R (1997). Prevalence of workplace smoking bans in Victoria. Australian and *New Zealand Journal of Public Health* 21: 694–698.

Brown KG (1999). Lung cancer and environmental tobacco smoke: occupational risk to nonsmokers. *Environmental Health Perspectives* 107:885–890.

Brown SK (1997). *Indoor air quality, Australia: State of the Environment Technical Paper Series (Atmosphere).* Commonwealth Department of the Environment, Sport and Territories, Canberra

California Environmental Protection Agency (1997). *Health effects of exposure to environmental tobacco smoke.* Office of Environmental Health Hazard Assessment. California Environmental Protection Agency, Sacramento, USA.

CDHAC (Commonwealth Department of Health and Aged Care) (1999). *National Tobacco Strategy 1999 to 2002–3.* CDHAC, Canberra.

CDHFS (Commonwealth Department of Health and Family Services) (1997). *Government Response to the Report of the Senate Community Affairs References Committee: the Tobacco Industry and the Costs of Tobacco Related Illness.* CDHFS, Canberra.

CDHFS (1998). *Environmental Health in Australia: Towards a National Strategy.* CDHF, Canberra.

CDHHCS (Commonwealth Department of Health, Housing and Community Services) (1993). National Campaign against Drug Abuse: Social Issues Survey 1993, Social Science Data Archives, Australian national University, Canberra.

CDHSH (Commonwealth Department of Human Services and Health) (1994). *Better Health Outcomes for Australians: National Goals, Targets and Strategies for Better Health Outcomes into the Next Century.* Australian Government Publishing Service, Canberra.

Davis RM (1997). Passive smoking: history repeats itself. *British Medical Journal* 315: 961–962.

Davis RM (1998). Exposure to environmental tobacco smoke: identifying and protecting those at risk. *Journal of the American Medical Association* 280:1947–1949.

Dimich-Ward H, Gee H, Brauer M, and Leung V (1997). Analysis of nicotine and cotinine in the hair of hospitality workers exposed to environmental tobacco smoke. *Journal of Occupational and Environmental Medicine* 39:946–948. Eisner MD, Smith AK and Blanc PD (1998). Bartenders' respiratory health after establishment of smokefree bars and taverns. *Journal of the American Medical Association* 280:1909–1914.

Eliopoulos C, Klein J, Phan MK, Knie B, Greenwald M, Chitayat D and Koren G (1994). Hair concentrations of nicotine and cotinine in women and their newborn infants. *Journal of the American Medical Association* 23:621–623.

Elliott and Shanahan Research (1997a).

Elliott and Shanahan Research (1997b).

Filippini G, Farinotti and Ferrarini M (2000). Active and passive smoking during pregnancy and risk of central nervous system tumours in children. *Paediatric and Perinatal Epidemiology* 14:78–84.

Fontham ET, Correa P, WuWilliams A, Reynolds P et al (1991). Lung cancer in non-smoking women: a multicenter case-control study. *Cancer Epidemiology, Biomarkers and Prevention* 1:35–43.

Fontham ET, Correa P, Reynolds P, Wu-Williams A et al (1994). Environmental tobacco smoke and lung cancer in non-smoking women. A multicenter study. *Journal of the American Medical Association* 271:1752–1759.

Goodin M (1995). Clean indoor air legislation in Australia (letter). *Tobacco Control* 4:294–296.

Gryczynska D, Kobos J and Zakrzewska A 1999. Relationship between passive smoking, recurrent respiratory tract infections and otitis media in children. *International Journal of Paediatric Otorhinolaryngology* 49 Suppl 1:S275–S278.

Hackshaw AK, Law MR and Wald NJ (1997). The accumulated evidence on lung cancer and environmental tobacco smoke. *British Medical Journal* 315: 980–987.

Hammond SK, Sorensen G, Youngstrom R and Ockene JK (1995). Occupational exposure to environmental tobacco smoke. *Journal of the American Medical Association* 274: 956–960

He J, Vupputuri S, Allen K, Prerost MR, Hughes J and Whelton PK (1999). Passive smoking and the risk of coronary heart disease – a meta-analysis of epidemiologic studies. *New England Journal of Medicine* 340:920–926.

Health Department of Western Australia (1998). Smoking and Health in Western Australia 1998 Resource Book. Development and Support Branch, Public Health Division, Health Department of Western Australia. Howard DJ, Ota RB, Briggs LA, Hampton M and Pritsos CA (1998a). Environmental tobacco smoke in the workplace induces oxidative stress in employees, including increased production of 8-hydroxy-2'-deoxyguanosine. *Cancer Epidemiology, Biomarkers and Prevention* 7:141–146.

Howard G, Wagenknecht LE, Burke GL, Diez-Roux A, Evans GW, McGovern P, Nieto FJ and Tell GS (1998b). Cigarette smoking and progression of atherosclerosis: the atherosclerosis risk in communities (ARIC) study. *Journal of the American Medical Association* 279:119–124.

Human Rights and Equal Opportunity Commission (1997). Decision by Commissioner Mr Graeme Innes AM, 25 Sept. 1997, cases nos. H97/50 and H97/51 between Neil Francey and Sue Meeuwissen (complainants) and Hilton Hotels of Australia Pty Ltd (respondent).

IARC (International Agency for Research on Cancer) (1986). *Tobacco Smoking*. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Volume 38, IARC, Lyon, France.

Jacobson PD and Wasserman J (1997). *Tobacco Control Laws: Implementation and Enforcement.* RAND, Santa Monica, USA.

Jarvis M, Foulds J and Feyeraband C (1992). Exposure to passive smoking among bar staff. *British Journal of Addiction* 87:111–113.

Johnson KC and Repace J (in press). Turning over the wrong stone (letter). *British Medical Journal.*

Jones K , Wakefield M, Turnbull DA (1999). Attitudes and experiences of restaurateurs regarding smoking bans in Adelaide, South Australia, *Tobacco Control* 8:62–66

Kagan RA and Skolnick JH (1993). Banning Smoking: Compliance Without Enforcement. In: Rabin RL and Sugar man SD (eds). *Smoking Policy: Law Policy, and Culture.* OUP, New York, US.A

Kawachi I, Colditz GA, Speizer FE, Manson JE, Stampfer MJ, Willett WC and Hennekens CH (1997). A prospective study of passive smoking and coronary heart disease. *Circulation* 95:2374–2379.

Kreuzer M, Krauss M, Kreienbrock L, Jockel KH and Wichmann HE (2000). Environmental tobacco smoke and lung cancer: a case-control study in Germany. *American Journal of Epidemiology* 151:241–250.

Law MR, Morris JK and Wald NJ (1997). Environmental tobacco smoke exposure and ischaemic heart disease: an evaluation of the evidence. *British Medical Journal* 315:980–988.

Lee N (1997). Survey of Parramatta Restaurant Patrons Attitudes to Smokefree Dining. Western Sydney Area Health Promotion Centre.

Lewtas J (1990). Indoor air – health. Human exposure and dosimetry of environmental tobacco smoke. Office of Research and Development, United States Environmental Protection Agency. Health Effects Research Laboratory, North Carolina, USA.

Lindgren T, Willers S, Skarping G and Norback D (1999). Urinary cotinine concentration in flight attendants, in relation to exposure to environmental tobacco smoke during intercontinental flights. *International Archives of Occupational and Environmental Health* 72:475–479.

Lister SM and Jorm LR (1998). Parental smoking and respiratory illnesses in Australian children aged 0–4 years: ABS 1989–90 National Health Survey results. *Australian and New Zealand Journal of Public Health* 22:781–786.

Makkai T and McAllister I (1998). *Public Opinion Towards Drug Policies in Australia 1985–95.* Department of Health and Family Services, Canberra.

Matt GE, Wahlgren DR, Hovell MF, Zakarian JM, Bernert JT, Meltzer SB, Pirkle JL and Caudill S (1999). Measuring environmental tobacco smoke exposure in infants and young children through urine cotinine and memory-based parental reports: empirical findings and discussion. *Tobacco Control* 8:282–289.

McAllister I (1993). *Knowledge, attitudes and policy preferences concerning drugs in Australian society.* NCADA National Household Survey report series. Australian Government Publishing Service, Canberra.

Miller SL , Branoff S and Nazaroff WW (1998). Exposure to toxic air contaminants in environmental tobacco smoke: an assessment for California based on personal monitoring data. *Journal of Exposure Analysis and Environmental Epidemiology* 8:287–311.

Misra DP and Nguyen RH (1999). Environmental tobacco smoke and low birth weight: a hazard in the workplace? *Environmental Health Perspectives* 107(Suppl 6):897–904.

Mizoue T, Ueda R, Hino Y and Yoshimura T (1999). Workplace exposure to environmental tobacco smoke and high-density lipoprotein cholesterol among nonsmokers. *American Journal of Epidemiology* 150:1068–1072.

Mullins RA (1991). *Survey of patrons of Melbourne restaurants on the provision of smokefree dining.* Centre for Behavioural Research on Cancer, Melbourne. Mullins R, Borland R and Hill D (1992). *Smoking knowledge, attitudes and behaviour in Victoria, results from the 1990–91 household surveys.* QUIT Evaluation Studies No. 6, 1990–91. Victorian Smoking and Health Programme, Melbourne.

NHMRC (National Health and Medical Research Council) (1987). *Effects of Passive Smoking on Health: Report of the NHIMRC Working Party on the Effects of Passive Smoking on Health.* Adopted by the 101st session of the NHMRC, Australian Government Publishing Service, Canberra.

NHMRC (1997). *The Health Effects of Passive Smoking: a scientific information paper.* Commonwealth Department of Health and Family Services, Canberra.

National Heart Foundation (ACT) (1992). *Survey of Attitudes Towards Smoking in Public Places: Summary and Review of Key Findings.* National Heart Foundation (ACT), Canberra.

Northern Sydney Area Health Service (1994). *Healthy Restaurants Resource Kit.* Manly Health Promotion Unit, Northern Sydney Area Health Service, Sydney.

NSW Health Promotion Survey (1995).

Ott W, Switzer P and Robinson J (1996). Particle concentrations inside a tavern before and after prohibition of smoking: evaluating the performance of an indoor air quality model. *Journal of the Air and Waste Management Association* 46: 1120–1134.

Pederson LL, Wanklin M, Bull SB and Ashley MJ (1991). A conceptual framework for the roles of legislation and education in reducing exposure to environmental tobacco smoke. *American Journal of Health Promotion* 62: 105–111.

Phillips K, Bentley MC, Howard DA and Alvan G (1998a). Assessment of environmental tobacco smoke and respirable suspended particle exposure for nonsmokers in Prague using personal monitoring. *International Archives of Occupational and Environmental Health* 71:379–390.

Phillips K, Bentley MC, Howard DA and Alvan G (1998b). Measured exposures by personal monitoring for respirable suspended particles and environmental tobacco smoke of housewives and office workers resident in Bremen, Germany. *International Archives of Occupational and Environmental Health* 71:201–212.

Pirkle JL, Flega KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR (1996). Exposure of the US population to environmental tobacco smoke: The Third National Health and Nutritional Examination Survey, 1988 to 1991. *Journal of the American Medical Association* 275(16):1233–1240.

Pryer W (1998). Smoking study sees gap in law. *The West Australian* 26 November 1998.

QUIT Tasmania (1996). *Smokefree Dining Survey.* QUIT Tasmania.

Raw GJ and Hamilton RM (eds) (1995). Building Regulation and Health, Building Research Establishment Report BR289, London, UK.

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

Raitakari OT, Adams MR, McCredie RJ, Griffiths KA and Celermajer DS (1999). Arterial endothelial dysfunction related to passive smoking is potentially reversible in healthy young adults. *Annals of Internal Medicine* 130: 578–581.

Repace JI and Lowrey AH (1985). A quantitative estimate of nonsmokers' lung cancer risk from passive smoking. *Environment International* 11:3–22.

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

Reynolds C 1998, Forms of Public Health Law – a general outline. In: *Public Health Law in Australia: New Perspectives, Australian Institute of Health, Law and Ethics.*

Reynolds P (1999). Epidemiological evidence for workplace ETS as a risk factor for lung cancer among nonsmokers: specific risk estimates. *Environmental Health Perspectives* 107:865–872.

Richmond R, Heather N, Holt P and Hu W (1993). Workplace Policies and Programs for Tobacco, Alcohol and Other Drugs in Australia. NCADA Monograph No. 24. AGPS, Canberra.

Right Marketing (Aust) Pty Ltd (1997). *Provision and Use of Smokefree Facilities in the Hospitality Industry.* Appendix 2 in: Western Australian Task Force Report on Passive Smoking in Public Places (1997), Perth, WA.

Sadler L, Belanger K, Saftlas A, Leaderer B, Hellenbrand K, McSharry JE and Bracken MB (1999). Environmental tobacco smoke exposure and small-for-gestational-age birth. *American Journal of Epidemiology* 150:695–705.

Schofield MJ, Considine R, Boyle CA, and Sanson-Fisher R (1993). Smoking control in restaurants: the effectiveness of self-regulation in Australia. *American Journal of Public Health* 83: 1284–1288.

Semmonds A, Bailey K, Bentley S, Chase V, Fernando S, Guruge A, King M, Tan OM and Walsh R (1995). Smoking in hotels: prevalence, and opinions about restrictions. *Australian Journal of Public Health* 19: 98–100.

South Australia Department of Human Services (1999a). *Smoke-free Dining Legislation Evaluation. Community Survey – 1999.* South Australia Department of Human Services, Adelaide.

South Australia Department of Human Services (1999b). Smoke-free Dining Legislation Evaluation. Dining Venue Survey and Inspection 1999. South Australia Department of Human Services, Rundle Mall.

Sweet M (1998). Tobacco companies try to sow seeds of doubt. *Sydney Morning Herald*, 3 February.

Tomblin JB, Hammer CS and Zhang X (1998). The association of parental tobacco use and SLI. *International Journal of Language and Communication Disorders* 33:357–368.

Trotter L (1998). *Environmental tobacco smoke: surveys of restaurant patrons and hospitality industry personnel. In: QUIT Evaluation Studies No. 9: 1996–97*, pp. 43–59. Centre for Behavioural Research on Cancer, Melbourne.

Trout D, Decker J, Mueller C, Bernert JT and Pirkle J (1998). Exposure of casino employees to environmental tobacco smoke. *Journal of Occupational and Environmental Medicine* 40: 270–276.

Turnbull D, Jones K, Teusner D (1996). *Results of a study into the provision of no-smoking policies in metropolitan restaurants and cafes and other enclosed public places throughout S.A.*, Report to the South Australian Health Commission, University of Adelaide

Tutt D and Harris W (1990). Where there's smoke – carbon monoxide exposures in smoking and smokefree workplaces. *Community Health Studies* 14: 297–301.

UK Department of Health (1998). *Report of the Scientific Committee on Tobacco and Health.* London, UK.

UK Independent Scientific Committee on Smoking and Health (1988). *Fourth Report of the Independent Scientific Committee on Smoking and Health.* (Chairman: Sir Peter Froggat), HMSO, London, UK. US Environmental Protection Agency (1992). *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders.* Document EPA/600/6-90/006F. Office of Health and Environmental Assessment, Office of Research and Development, Washington DC, USA.

US Department of Health and Human Services (1986). *The Health Consequences of Involuntary Smoking: A Report of the Surgeon General.* Publication no. (CDC) 87-8398, Office on Smoking and Health, Center for Health Promotion and Education, Centers for Disease Control, Public Health Service, US Dept. of Health and Human Services, Rockville, MD, USA.

US National Research Council (1986). *Environmental Tobacco Smoke – Measuring Exposures and Assessing the Health Effects.* Committee on Passive Smoking, Board on Environmental Studies and Toxicology, National Research Council, National Academy Press, Washington, USA.

Vinke JG, KleinJan A, Severijnen LW and Fokkens WJ (1999). Passive smoking causes an 'allergic' cell infiltrate in the nasal mucosa of non-atopic children. *International Journal of Paediatric Otorhinolaryngology* 51:73–81.

Wahlgren DR, Melbourne FH, Meltzer EO and Meltzer SB (2000). Involuntary smoking and asthma. *Current Opinion in Pulmonary Medicine* 6:31–36.

Wakefield M, Roberts L and Miller C (1999a). Public perceptions of the effect of an impending restaurant smoking ban on dining out experience. In: *Evaluation and Research Report No. 5 1995–1998*, SASHP, Adelaide.

Wakefield M, Roberts L and Miller C (1999b). Population monitoring of tobacco control progress in South Australia. In: *Evaluation and Research Report No. 5 1995–1998.* SASHP, Adelaide.

Wakefield M, Roberts L and Owen K (1996). Trends in prevalence and acceptance of workplace smoking bans among indoor workers in South Australia. *Tobacco Control* 5:1–4.

WA Task Force (Western Australian Task Force on Passive Smoking in Public Places) (1997). *Task Force Report.* Perth. WA.

Wells AJ (1998a). Heart disease form passive smoking in the workplace, *Journal of the American College of Cardiology* 31:1–9.

Wells AJ (1998b). Lung cancer and passive smoking at work. *American Journal of Public Health* 88: 1025–1029.

WHO (World Health Organization) (1999). *International Consultation on Environmental Tobacco Smoke (ETS) and Child Health, Consultation Report.* Division of Noncommunicable Diseases, WHO, Geneva.

WHO (World Health Organization) (in press). *Air Quality Guidelines for Europe: Volume 5 – Indoor.* WHO Regional Office for Europe, Copenhagen, Denmark.

Winstanley M, Woodward S and Walker N (1995). *Tobacco in Australia: Facts and Issues*, 2nd edition. Victorian Smoking and Health Program (Quit Victoria), Melbourne.

Witschi H, Espiritu I, Maronpot RR, Pinkerton KE and Jones AD (1997). The carcinogenic potential of the gas phase of environmental tobacco smoke. *Carcinogenesis* 18: 2035–2042.

Woodward A and Al-Delaimy W (1999). Measures of exposure to environmental tobacco smoke. Validity, precision, and relevance. *Annals of the New York Academy of Sciences* 895:156–172.

Wooldridge M (1997). Scientific evidence mounts against passive smoking. Media release, 24 November.

You RX, Thrift AG, McNeil JJ, Davis SM and Donnan GA (1999). Ischemic stroke risk and passive exposure to spouses' cigarette smoking. Melbourne Stroke Risk Factor Study (MERFS) Group. *American Journal of Public Health* 89:572–575.

Young S, Arnott J, O'Keeffe PT, Le Souef PN and Landau LI (2000). The association between early life lung function and wheezing during the first 2 years of life. *European Respiratory Journal* 15:151–157.

Zhong L, Goldberg MS, Parent ME and Hanley JA (2000). Exposure to environmental tobacco smoke a meta-analysis. *Lung Cancer* 27:3–18.