INTERNATIONAL REVIEW OF THE HEALTH AND ECONOMIC IMPACT OF THE REGULATION OF SMOKING IN PUBLIC PLACES

Anne Ludbrook¹, David Cohen², Sheona Bird¹ and Edwin van Teijlingen³, Health Economics Research Unit¹ and Department of Public Health³, University of Aberdeen, and University of Glamorgan².

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EXECUTIVE SUMMARY

INTRODUCTION

0.1 The National Assembly for Wales Committee on Smoking in Public Places was established with terms of reference that included consideration of evidence on relevant issues. This review summarises evidence relating to health and economic impacts.

The aims of the review as stated in the brief are:
- To determine the health impact of smoking bans and smoking restrictions in public places; and
- To determine the economic impact of smoking bans and smoking restrictions in public places.

METHODS

0.2 A literature review has been conducted to establish the impacts of restrictions on smoking in public places. The literature review has had to cover a number of distinct areas: health impacts of exposure to environmental tobacco smoke (ETS) or passive smoking; impact of restrictions on exposure levels; impact of restrictions on tobacco use behaviour; economic impacts of restrictions on the hospitality sector; and costs of workplace smoking.

0.3 Existing high quality reviews of evidence were sought first and primary studies were only reviewed where such studies were lacking or did not provide sufficient information for the nature and quality of the evidence to be judged. Quality assessment of reviews and primary literature was carried out with respect to the study methods and whether or not peer-review had taken place.

HEALTH IMPACTS OF ETS

Lung cancer

0.4 More than 50 epidemiological studies have examined the relationship between passive smoking and lung cancer. The excess risk amongst never-smoking females, who have self-reported exposure to passive smoking in the home from their husbands who are smokers, appears to be about 25%. A dose-response relationship between excess risk of lung cancer and number of cigarettes smoked by husbands is indicated. Although most of the reviews restrict analysis to females only, the most recent meta-analysis found no difference between the relative risk values for men and women. It is generally agreed that exposure in one place acts in similar ways to exposure in others and therefore exposure in public places would elevate the risk of lung cancer. The most recent meta-analysis of studies of workplace exposure reports levels of excess risk that are consistent with those for domestic exposure.
Coronary Heart Disease (CHD)

0.5 More than 20 epidemiological studies have assessed the relationship between CHD (also referred to as ischaemic heart disease (IHD)) and exposure to ETS. The excess risk associated with ever exposure to ETS amongst never smoking women who have a spouse that smokes, appears to be in the region of 25%. The risks associated with exposure in the home range from 1.22 to 1.51. Where risks from workplace exposure are pooled, the estimates are similar, 1.11 to 1.32. There appears to be some evidence of a dose-response relationship in terms of the intensity of ETS to which the individual is exposed.

Stroke

0.6 Only seven studies that look at the association between risk of stroke and exposure to ETS were found and no meta-analysis of the studies has been conducted. Most of the studies were fairly small and whilst most found an excess risk of stroke related to exposure to ETS, the size of the effect varied considerably and the confidence intervals around the estimates were large. Only two of the reported results are statistically significant. The studies also used different endpoints associated with ETS exposure and this makes it unlikely that formal meta-analysis of all the studies could be carried out. Nevertheless, the pattern of association is clear. The relationship between active smoking and stroke is similar to active smoking and CHD. It is plausible that the relationship also holds for passive smoking but this has not been clearly demonstrated because of a lack of studies of sufficient size.

Respiratory Disorders

0.7 Few studies have been conducted in this area (with the exception of lung function). The risks that are presented appear to indicate that a relationship exists between exposure to ETS and poor respiratory health. However, the degree to which passive smoking affects the respiratory system is not clear, as many of the confidence intervals are wide and the risk estimates vary considerably between studies and different health effects. Comparisons between studies are difficult to make due to differences in study design and quality.

0.8 In 1992, the US Environmental Protection Agency (EPA) compared the risks of exposure to ETS to those gained from light active smoking (i.e. less than 10 cigarettes/day). In 1997, the Californian Environmental Protection Agency (CEPA) stated that epidemiological evidence published since the US EPA report had further strengthened the association between the risk of respiratory disorders and exposure to ETS. A more recent review argued that although further research is required, the evidence presented so far indicates that exposure to ETS adversely affects the respiratory system.

Pregnancy

0.9 A number of studies have indicated that exposure to ETS amongst pregnant women may affect the foetal growth and birth weight of the child. In 1997, the Californian Environmental Protection Agency (CEPA) published a comprehensive review of the evidence regarding passive smoking and health risks to the foetus/child with both perinatal and postnatal manifestations. They concluded that small decrements in mean birth weight (25-50
grams) are associated with ETS exposure. Studies that had looked at passive smoking in pregnancy with low birth weight (less than 2500 grams) or small for gestational age had indicated an excess risk of 20 – 40%. Although few studies had examined other health effects, there was a suggestion that exposure to passive smoking may be linked with risk of neonatal mortality rates, spontaneous abortion and congenital malformation. There was no or little evidence linking exposure with stillbirth, sudden infant death syndrome or cognitive and behavioural problems. More recent reviews have generally supported the conclusions made in the CEPA report.

Quality and relevance of the evidence

0.10 The literature relating to lung cancer and CHD is substantial and there are a number of good quality meta-analyses of primary studies. The design of the studies that have been carried out, cohort studies and case-control studies, makes them vulnerable to the possibility of the results being affected by confounding variables and there are other sources of potential bias. However, some of the meta-analyses have taken these factors into account and have adjusted results accordingly. These studies show that there may be an effect on the size of the relative risk for exposure to ETS but adjusting for these factors does not eliminate the excess risk. Some of the potential sources of bias act in opposite directions.

EXPOSURE TO ETS AND ASSOCIATED HEALTH RISKS IN HOSPITALITY SETTINGS

0.11 Several studies have measured exposure levels in hospitality settings. The data indicated exposure levels in hospitality settings where smoking occurred to be higher than in areas that do not permit it. Nicotine concentration levels and cotinine levels related to exposure in hospitality settings appeared to be higher than exposure levels related to exposure at home or other workplaces. High levels of exposure to ETS clearly have implications for the health of hospitality workers.

0.12 A study comparing the exposure of non-smoking hospitality workers to workers in smoke free employment found the mean post-shift salivary cotinine concentrations for workers in bars and restaurants was 3.38 ng/ml compared to 0.08 ng/ml amongst those in smoke-free employment. Other studies of exposure amongst hospitality workers report similar levels of exposure. Studies have reported elevated risks of lung cancer and increased respiratory symptoms amongst hospitality workers.

Quality and relevance of the evidence

0.13 Most studies have been conducted in the US and some studies have taken measurements in only one location. Exposure levels may not accurately reflect wider workplace characteristics and smoking habits/behaviours in the UK. Measurements associated with exposure levels in the 1980s and 1990s may not be representative of current exposure levels as smoking rates change and smoking behaviours in public places become less socially acceptable. Very few studies have assessed the health effects of exposure to ETS amongst hospitality workers.
IMPACT OF SMOKING BANS AND RESTRICTIONS ON EXPOSURE TO ETS

0.14 A number of studies have been conducted that have considered the effectiveness of smoking bans and restrictions on exposure to ETS and these have been the subject of a review for the US Task Force on Community Preventive Services (TFCPS). Ten studies were assessed to be of sufficient quality to include in the review: four evaluated restrictions; four evaluated bans and two considered both. Seven studies evaluated particular worksites and three studies were population-based surveys. Four studies measured air quality and six were based on self-reported exposure to ETS. Smoking bans were generally associated with greater reductions in exposure to ETS.

0.15 Some recent studies relating to the hospitality sector showed a 90% reduction in the level of respirable suspended particles and a reduction in self-reported exposure to ETS following a smoking ban. An observational study found that designated ‘no smoking’ areas in licensed gaming clubs typically produced about 50% reduction in exposure to ETS.

Quality and relevance of the evidence

0.16 The TFCPS review was conducted to a high standard and individual studies were assessed for quality, including the robustness of the design. The studies of specific work settings encompassed the health care sector, government and other public sector workplaces and a university. Whilst this may not be a totally representative sample of workplaces, this is unlikely to bias the measurement or reporting of exposure to ETS.

IMPACT OF SMOKING BANS AND RESTRICTIONS ON SMOKING BEHAVIOUR

0.17 Four reviews have been published relating to the impact of smoking bans and restrictions on smoking behaviour. The literature reviewed included individual workplace studies, population based studies of workplaces and studies of the impact of public laws on smoking behaviour.

Cigarette consumption

0.18 Most of the studies reported reductions in cigarette consumption. The TFCPS review reported a median reduction of 1.2 cigarettes per day (range no change to –4.3 cigarettes per day). A more recent meta-analysis, which included a larger number of studies, gave a pooled estimate of –3.1 cigarettes per day. A third review reported reductions in the quantity smoked of 10-20% from workplace studies. Population based studies have also shown lower consumption by smokers in workplaces with restrictions compared to those without.

0.19 Clean air laws are also effective in reducing cigarette consumption, although these results are based on per capita consumption and will combine reductions in consumption per smoker and reduced smoking prevalence. Clean air laws appear to be more effective than workplace bans in reducing cigarette consumption per capita.
Effect on smoking cessation or quit attempts

0.20 In 3 studies with 12-18 months follow up there were more quitters with a ban than with other workplace restrictions or no restrictions. Results for quit rates were not consistent in prospective cohort studies but they showed little initial effect with greater increases over time. Population based studies have shown higher rates of quit attempts and quitting (10-15%) by workers in workplaces with bans. States with clean air laws had higher quit rates than States without such laws.

Smoking prevalence

0.21 There are fewer studies reporting the effect of smoking bans and restrictions on smoking prevalence and those included in the TFCPS review gave inconsistent results. Prospective cohort studies reported reductions in smoking prevalence of 7-20% and population based studies comparing workplaces with and without restrictions showed 15-20% lower prevalence. Partial restrictions had little or no effect. A recent meta-analysis reported a 3.8% reduction in absolute prevalence (pooled effect) associated with smoke free workplaces.

0.22 The effect of clean air laws on smoking prevalence has only been included in a few studies. Prevalence rates were lower in States with extensive restrictions. In one study, the impact was greatest in the 25 – 44 year old age group. Some studies have examined youth smoking but with variable results. Smoking restrictions in schools appear to be effective and a broader range of smoking restrictions may reduce youth quantity smoked and progression to established smoking.

Quality and relevance of the evidence

0.23 Unlike the studies of impact on exposure to ETS, population studies of smoking behaviour may be affected by selection bias and the specific worksites studied may not be representative of the wider effects of restrictions. Some studies have attempted to control for these problems. One study estimated a 20% reduction in per capita consumption of cigarettes with clean air laws compared with 4-8% without clean air laws, after controlling for smoking sentiment. By contrast, the impact of worksite laws became insignificant when social attitudes were taken into account. The smoking prevalence studies provided a wide range of estimates.

ECONOMIC IMPACTS OF RESTRICTIONS ON SMOKING IN PUBLIC PLACES

General effects on all workplaces

0.24 A small number of studies have been carried out on the costs of workplace smoking. These studies include a range of costs, some of which can be avoided by restrictions on workplace smoking. Other costs, such as absence due to ill health of smokers will only be saved to the extent that smokers reduce or quit smoking.
Productivity loss caused by workplace smoking

0.25 Individual studies of the costs of smoking breaks have produced a wide range of estimates based on alternative assumptions about frequency of smoking breaks and differences in length of smoking breaks. These include a study in Scotland that estimated a gain in productivity from workplaces going smoke free of between £289 million and £605 million (1998 prices); an estimated annual loss of £740 million (Great Britain) based only the productivity losses when any form of smoking policy is introduced to a previously unrestricted workplace; an estimated gain for Ireland of €271 million (2002 prices).

Absenteeism due to passive smoking

0.26 The Health and Safety Executive have estimated costs for sickness absence relating to exposure to ETS for those with asthma and chronic bronchitis to be £83m to £166m per year in Great Britain.

Fire hazards

0.27 The cost of fire damage relating to smoking on business premises has been estimated as £4.5 million for Scotland (1998 prices) and £52 million for Great Britain (1998/9 prices).

Cleaning and redecoration costs

0.28 None of the published studies have estimated the effect of workplace smoking restrictions on cleaning and redecoration costs, although it is recognised that there will be an effect. A US survey estimates a saving of £300 per smoker per year on cleaning and maintenance costs.

Specific effects on the hospitality sector

0.29 Two comprehensive reviews of studies assessing the economic impact of smoke free policies were identified. The reviews included journal publications and grey literature. In addition, the TFCPS review considered six studies that showed no adverse impact of smoking ordinances on businesses or tourism. The reporting of individual studies within the reviews was considered to be limited, in terms of methods, results and conclusions. It was therefore decided to examine the available peer reviewed studies that used objective data.

Restaurants

0.30 A total of 11 studies have been conducted relating to the impact of smoking restrictions in restaurants. Ten of the studies were carried out in the USA and one in Australia. The USA studies covered restrictions in New York (State and City), California and Colorado, Massachusetts, Flagstaff, Arizona and West Lake Hills, Texas. Most of the
study results were not statistically significant and most of the effect sizes, whether positive or negative, were small.

Bars

0.31 One study of the effects of restrictions in California has been conducted. The pooled results for bars showed the impact on bar sales as a fraction of total retail sales was positive but not significant. The effect of the smoke free ordinance on bar sales as a fraction of retail sales was reported as +0.5 with a 95% CI of –0.284 to +1.284.

Hotels and tourism

0.32 Four studies have considered the effect of smoking restrictions on hotel business, as a proxy for tourism, or on tourist numbers directly. Most of the results were not significant. One study showed a positive and significant effect on sales. One location within one study reported a significant negative impact on hotel room revenues. The effect on tourist numbers was either not significant or positive.

Quality and relevance of the literature

0.33 Three papers have provided a commentary on the quality of the literature relating to the hospitality sector. There are a number of valid criticisms relating to the studies carried out in this area and these reflect the difficulties of conducting research into policy impacts. The problems include: the inadequacy of sales tax data to capture all the effects, the timing of the intervention in relation to the data periods; limitations to the smoking restrictions; compliance with the smoking restrictions; selection bias; and the transferability of the results to other settings.

0.34 The failure to find any significant impact on revenues in the sectors analysed does not rule out the possibility of a small negative effect on business but it does weigh against a large negative impact being experienced. If such large effects were experienced and were widespread then it seems unlikely in the extreme that no objective data have been produced to substantiate these effects. However, it is also the case that there has been no analysis of impacts within sectors and no analysis based on measures such as sales volume or profits was reported in the reviews.

CONCLUSIONS

0.35 There is a substantial literature on the health effects of ETS and strong evidence that exposure to ETS increases the risk of mortality and morbidity from lung cancer and CHD. There is less evidence relating to stroke and respiratory disorders but sufficient to suggest a link. There is also evidence to link exposure to ETS with low birth weight and a number of other health problems.

0.36 Smoking bans and restrictions have been shown to reduce exposure to ETS. Smoking bans produce a greater reduction in exposure to ETS than other types of smoking restrictions.
Smoking bans and restrictions have been associated with reductions in the number of cigarettes smoked by continuing smokers, increased quit attempts and reduced smoking prevalence. The precise size of the effect on smoking prevalence is unclear. However, even a modest reduction in active smoking would produce benefits at a population level as large as those associated with reducing passive smoking.

0.37 There are costs associated with smoking at work, some of which would be averted if smoking were banned. A relatively small number of published studies have measured the impact of smoking bans and restrictions on the hospitality industry using objective data. These studies consistently find small effects, most of which are positive. The studies have been carried out in the USA and Australia and results may not transfer exactly to other settings.
1 INTRODUCTION

BACKGROUND
1.1 The National Assembly for Wales Committee on Smoking in Public Places was set up with following terms of reference.

i. consider current evidence on relevant issues, including the health risks of environmental tobacco smoke and the economic impact of restrictions on smoking in public places;

ii. review developments in the UK and Ireland relating to the introduction of restrictions on smoking in public places (including the debates on Baroness Finlay’s and Lord Faulkner’s Private Member’s Bills, the response to the UK Government consultation on devolving powers to local authorities to introduce smoking bans at work and in public places, the outcome of the Scottish Executive consultation on smoking in public places, and the experience of implementing the workplace smoking ban in Ireland).

iii. consider the experiences in other countries where a ban has been introduced; and

iv. report to the Assembly by 25 May 2005 on its conclusions.

The present review was commissioned by NHS Health Scotland on behalf of the Scottish Executive and the National Assembly for Wales to inform the debate on the banning or restricting of smoking in public places.

AIMS AND OBJECTIVES
1.2 The aims of the review as stated in the brief are to determine:
• the health impact of smoking bans and smoking restrictions in public places; and
• the economic impact of smoking bans and smoking restrictions in public places.

Public places are taken to encompass workplaces and the leisure and hospitality sector (including pubs and restaurants).

STRUCTURE OF THE REPORT
1.3 The next section of the report sets out briefly the methods employed in the study and details of the search strategy employed. Thereafter, section 3 provides an overview of the evidence relating to the health impacts of exposure to environmental tobacco smoke (ETS), which would be reduced or eliminated by bans or restrictions on smoking in workplaces or other public places. Section 4 sets out the evidence relating to exposure levels and health effects specific to the hospitality sector.

1.4 The impact of smoking bans and restrictions on exposure to ETS is dealt with in section 5. The impact of bans and restrictions on tobacco use behaviours is reviewed in section 6. Section 7 considers the evidence relating to economic impacts of smoking bans or
restrictions. **Section 8** provides some conclusions and recommendations relating to areas where the evidence base is weak and where further research might be valuable.
2 METHODS

2.1 A literature review has been carried out to determine the impacts of restrictions on smoking in public places. The study has focussed on the main health and economic impacts identified in the literature; therefore, the scope of the study did not include factors such as the costs of any legislative process, implementation costs or compliance costs.

LITERATURE REVIEW

2.2 The literature review has covered a number of distinct areas. The review included evidence relating to the health effects of exposure to ETS, the impact of restrictions on exposure levels and on tobacco use behaviours and the economic impacts of restrictions on workplaces, in general, and on the hospitality sector in particular. The relatively small number of interventions and the short timescale of follow-up, in the context of health benefits, limited the availability of evidence relating to the impact of actual restrictions that have been put in place.

Search strategy

2.3 A search of electronic databases was carried out, using the search strategy set out in Appendix 1. Given the extensive requirements of the review and a short timescale, the evidence was reviewed in a hierarchical manner. Existing high quality reviews of evidence were sought first and primary studies were only examined where such reviews were lacking or where they did not provide sufficient information for the nature and quality of primary evidence to be judged.

Quality assessment

2.4 Quality assessment of reviews and primary literature were carried out with respect to the study methods and whether or not peer review had taken place.
3 HEALTH IMPACTS OF ETS

SUMMARY

This section reviews the literature on the main health impacts of exposure to ETS.

- The excess risk of lung cancer associated with domestic exposure to ETS is about 25%. The range of estimates for workplace exposure is similar to domestic exposure.
- The excess risk of CHD associated with domestic exposure to ETS is also about 25%. The range of estimates for workplace exposure is similar to domestic exposure.
- The risks appear to increase with the extent of exposure to ETS, although in the case of CHD the relationship is not linear.
- There is some evidence to suggest a relationship between exposure to ETS and stroke. Although recent studies report an excess risk of about 34%, further research is required.
- Exposure to ETS has a detrimental effect on lung function and may be associated with poorer respiratory health.
- Exposure to ETS in pregnancy can lead to low birth weight and poor gestational growth.
- The literature relating to lung cancer and CHD is substantial and contains a number of good quality meta-analyses of primary studies.
- Studies that have examined the impact of ETS on health can be affected by bias and confounding. However, good quality studies that have adjusted for these factors still find significant effects.

INTRODUCTION

3.1 Environmental Tobacco Smoke (also known as passive or second-hand smoke) is a diluted mixture of side stream smoke, which is released from a burning cigarette between puffs, and mainstream smoke, which is exhaled by the smoker. ETS contains over 4000 gaseous and particulate compounds, 40 of which are known or suspected human carcinogens. The US Environmental Protection Agency (1992) has classified ETS as a Group A carcinogen and the International Agency for Research on Cancer (IARC) (2002) has defined exposure to second-hand smoke as ‘carcinogenic to humans’.

3.2 Exposure levels can be determined and quantified using markers in air, bodily fluids or through questionnaires/interviews. The most widely used markers for indoor air concentrations are nicotine and RSP (respirable suspended particulates). Cotinine, a metabolite of nicotine is the most common biomarker. Cotinine can be measured in plasma and saliva (measurements are generally equal to each other) and in urine. Cotinine has a half-life in the body of about 15 to 19 hours.

3.3 Exposure levels are dependent on several factors; number of smokers, ventilation properties, size of room and proximity to the source of ETS etc. In addition, exposure levels experienced in workplaces will depend on the working hours and length of shifts etc.
3.4 The health effects of ETS have been mainly examined in epidemiological studies using questionnaires. Few studies have validated self-reported exposure with other measures and there may therefore be concerns about exposure misclassification (see 3.47 – 3.53 for further details). In prospective cohort studies, questionnaires have usually been completed by participants, who were then followed up over a number of years. In case-control studies, questionnaires were issued to patients (or surrogate participants) who suffered/died from a particular disease and exposure to ETS was retrospectively measured. Evidence from these epidemiological studies has led to a number of authoritative bodies supporting associations between exposure to ETS and adverse health effects. According to the Californian Environmental Protection Agency (2004), there is sufficient evidence that a ‘causal’ association exists between ETS exposure in adults and:

- Lung Cancer
- CHD mortality and morbidity
- Low birthweight
- Asthma induction and exacerbation
- Breast Cancer
- Nasal sinus cancer
- Eye and nasal irritation

There is also evidence to ‘suggest’ a causal association between ETS exposure and:

- Stroke
- Spontaneous abortion, preterm delivery and intrauterine growth retardation (IUGR)
- Chronic Respiratory Symptoms
- Exacerbation of cystic fibrosis

3.5 The remainder of this section reviews the epidemiological evidence on lung cancer, CHD, stroke, respiratory disorders and the effects of exposure amongst pregnant women. It was felt that the literature regarding these conditions was extensive and as such, appropriate conclusions could be drawn regarding the quality and relevance of the evidence. Limitations of the time scale of the project restricted further review of other health effects such as those listed above.

LUNG CANCER

Introduction

3.6 More than 50 epidemiological studies have examined the relationship between passive smoking and lung cancer. The excess risk amongst never-smoking females, who have self-reported exposure to passive smoking in the home from their husbands who are smokers, appears to be about 25%. Several meta-analyses have pooled the results of these studies and reported relative risk estimates. These are presented in Appendix 2 Table A2.1a. An overview of the risk of lung cancer, amongst non-smoking females exposed to ETS in the home by smoking husbands, as estimated in meta-analyses over the years is shown in Figure 3.1. The vertical line shows a relative risk of 1; i.e. no excess risk of lung cancer. Values to the right of this line indicate an excess risk. The boxes show the average value for each study.
and the horizontal lines show the 95% confidence intervals around these values. If the confidence interval does not cross the vertical line, this indicates that the result is statistically significant.

**Figure 3.1 – Lung cancer and passive smoking (pooled relative risk estimates)**

<table>
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<td>Wald et al., 1986</td>
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<td>Vels, 1988</td>
<td>N=17</td>
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<td>Saracci and Riboli, 1989</td>
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<td>Lee, 1992</td>
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<td>Tweedie and Wengersen, 1992</td>
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</table>

N = Number of studies included in the meta-analysis
Study characteristics

3.7 Studies included in meta-analyses generally compared the risk of lung cancer between never-smokers according to whether they were exposed to ETS in the home and/or the workplace. Both cohort (prospective) and case-control studies have been conducted to estimate relative risk. Studies have taken place in many countries including USA, UK, China, Sweden, Greece, Japan and India. Lung cancer has mostly been confirmed histologically. The largest case-control study included 651 cases and 1253 controls (Fontham et al. 1994). Recent meta-analyses have not stated which endpoints have been used in each primary study. Risk estimates have therefore been assumed to be indicative of the risk of lung cancer in terms of both morbidity and mortality.

Summary of meta-analyses

3.8 The relative risk estimates for exposure to ETS from spouses ranged from 0.91 to 1.44. The only study that did not report an increased risk was restricted to Chinese women (Wang and Zhou 1997) and the authors noted that risk factors would be different compared with American women. Pooled risk estimates when restricted to US studies only ranged from 1.07 to 1.22. Only a few studies have reported pooled risk estimates from ETS exposure in the workplace (the range was from 1.01 to 1.39).

3.9 A dose-response relationship between excess risk of lung cancer and number of cigarettes smoked by husband was indicated. If the husband smoked more than 15 cigarettes per day, the pooled excess risk ranged from 1.45 to 1.81. This was confirmed by a study conducted by De Waard et al. (1995) who estimated an increased risk of lung cancer amongst passive smokers with high urinary cotinine levels. The risk associated with 9.2 to 23.4 ng/ml was 2.7 (95%CI=0.8 – 9.1).

3.10 A dose-response relationship between excess risk and duration of exposure was less convincing; where exposure has occurred over a period lasting 20 years or more, the excess risks ranged from 1.18 to 1.22.

3.11 Two studies have reported a higher risk of squamous and small cell lung cancer with one study estimating risk at 1.58 and another at 1.38 (for squamous cell only).

3.12 Although most of the reviews restricted analysis to females only, the most recent meta-analysis found no difference between the relative risk values for men and women. The relative risk estimate amongst men exposed to ETS from spouses (number of studies = 9) resulted in a relative risk of 1.25 (95%CI = 0.95 – 1.65). Similarly, Hackshaw et al. (1997) found no significant differences in the risk estimates between men and women (P = 0.31).

Recent Publications

3.13 Two large case-control studies, which have previously been included in meta-analyses, were pooled together in a recent publication (Brennan et al. 2004). The odds ratio for ever exposure to ETS from spousal smoking was 1.18 (95%CI=1.01 – 1.37). For workplace exposure the pooled risk was 1.13 (95%CI=0.97 – 1.31).
3.14 A publication by Enstrom and Kabat (2003) did not support an excess risk associated with exposure to ETS. Amongst female never smokers exposed to ETS from a spouse who smokes, the age-adjusted risk presented was 0.93 (0.65–1.33). Enstrom and Kabat’s risk estimate based on exposure in 1972 was slightly higher at 1.00 (95%CI=0.52-1.92) and although an excess risk was still not indicated, the confidence intervals included the risks presented in other recent studies and meta-analyses. A number of concerns have been expressed and published with regard to this paper. (see Letters in BMJ, 327, 30 August 2003). Enstrom and Kabat’s estimate was based on a 40 year follow up and the implications of long follow up periods are discussed in more detail towards the end of this section (3.50). Hackshaw (2003) has stated that even if Enstrom and Kabat’s estimate were included in a recent IARC meta-analysis (2002), the pooled relative risk value would be 1.23

Home versus Workplace Exposure

3.15 The majority of studies undertaken have included pooled relative risks for lung cancer among never smoking females exposed to ETS in the home. Levois and Layard (1994) reported risk estimates for workplace exposure that were considerably lower than those found from spousal exposure. They argued that this was due to the effects of bias and confounding that are associated with spousal studies. More recent analyses (i.e. Zhong et al. 2000; Boffeta, 2002) that have included a higher number of studies have however, reported higher relative risk estimates from exposure in the workplace and these are more consistent with the estimates found in spousal studies.

CORONARY HEART DISEASE

Introduction

3.16 More than 20 epidemiological studies have assessed the relationship between coronary heart disease (CHD) and exposure to ETS. The excess risk associated with ever exposure to ETS amongst men and women who have a spouse that smokes, appears to be in the region of 25% (see Appendix 2 Table A2.2a). An overview of the risks associated with exposure from a smoking spouse, as calculated by meta-analyses over time, can be seen in Figure 3.2. The vertical line shows a relative risk of 1; i.e. no excess risk of lung cancer. Values to the right of this line indicate an excess risk. The boxes show the average value for each study and the horizontal lines show the 95% confidence intervals around these values. If the confidence interval does not cross the vertical line, this indicates that the result is statistically significant.
3.17 Studies were conducted in many countries; Japan, Scotland, England, New Zealand, China, Argentina, Italy and Australia. In the most recent meta-analysis, half of all the studies included were conducted in the US. The largest study, with a cohort of 479,680 men and women, was conducted by Steenland et al. (1996). Studies used different endpoints. These generally included admission to hospital or death with coronary heart disease / ischemic heart disease as the underlying cause, and fatal and non-fatal myocardial infarction.

3.18 Both cohort and case-control studies have been conducted and included in meta-analyses. Cohort studies have mainly used death as the endpoint whereas case-control studies have mostly used non-fatal incidence as the endpoint. In the review by He et al. (1999) the pooled risk estimate for studies that used death as an endpoint was slightly smaller than the combined estimate. This was also the case in other meta-analyses.
Summary of meta-analyses

3.19 The risks associated with exposure in the home ranged from 1.22 to 1.51. Where risks from workplace exposure were pooled, the estimates were similar; 1.11 to 1.32. There appeared to be some evidence of a dose-response relationship in terms of the intensity of ETS the individual was exposed to. In He et al.’s (1999) pooled analysis the risks associated with exposure to 1 – 19 cigarettes/day was 1.23 (95%CI= 1.13 – 1.34) and with over 20 it was 1.31 (95%CI= 1.21-1.42) P = 0.006 for linear trend. This has been supported by the recent report by Whincup et al. (2004), which measured plasma cotinine levels in 1978/80 amongst 945 never-smoking British men between the ages of 40 and 59. The study found that high plasma cotinine levels were associated with higher risks. However, this was only apparent before multivariate adjustments were made. Cotinine acts as a biomarker of exposure to ETS (see 3.2 for further details)

3.20 The review by He et al. (1999) pooled data from several studies that had calculated risks associated with risk of CHD and duration of exposure and reported increasing risk with longer durations.

<table>
<thead>
<tr>
<th>Duration</th>
<th>Risk Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-9 years</td>
<td>1.18 (95%CI = 0.98 – 1.42)</td>
</tr>
<tr>
<td>10 – 19 years</td>
<td>1.31 (95%CI = 1.11 – 1.55)</td>
</tr>
<tr>
<td>20 years +</td>
<td>1.29 (95%CI = 1.16 – 1.43)</td>
</tr>
</tbody>
</table>

P = 0.01 for linear trend

More recently, Pitsavos et al. (2002) published estimates of risk over 20 years, which were also indicative of increased risk (see Appendix 2 Table 2.2b).

3.21 Where only studies from the US were pooled, the risk estimate was very similar. There also appears to be little difference between excess risk estimates for men only and for both sexes combined.

3.22 The estimated risk estimates amongst passive smokers exposed to ETS are similar to those who smoke about one cigarette a day. Glantz and Parmely (1991) suggested that the reason cigarette smoking had no linear dose-response effect on CHD risk was because the effects of smoking may reach saturation point. Law et al. (1997) looked at the risks of ischaemic heart disease associated with smoking one cigarette per day and estimated a risk of 1.39 (95%CI= 1.18 – 1.64) as weighted over five studies. Surprisingly, the risk of smoking 20 cigarettes a day was only 1.78 (and not 20 times the risk) and a linear dose-response relationship is clearly not evident. They reported that this pattern was similar to that with ETS. The exposure from ETS was only about 1% of smoking yet the risk was nearly half. They concluded that this was not fully explained by bias or confounding but that such a large effect from such a small dose results from platelet aggregation that occurs as an immediate response to exposure to ETS. Epidemiological data indicate that there may be both acute and chronic effects of ETS on cardiovascular health. Laboratory data suggest that low levels of exposure can trigger myocardial infarction through platelet aggregation. Increasing levels of exposure can further increase this risk with adaptations to HDL cholesterol and carboxyhaemoglobin levels (Pechacek and Babb, 2004). The dose-response relationship is believed to be non-linear with the risk of CHD increasing rapidly with low exposure to ETS. This is consistent with the effects of smoking cessation upon which the risk of myocardial infarction (MI) quickly declines within a short period of time (US Dept of Health, 1990).
3.23 A study published in 2004 (Sargent et al.) looked at the admissions to the local hospital in Helena, Montana, before and after a ban on smoking in public places was introduced. Comparisons were made with admission patterns in previous years and with admissions from outside Helena where legislation on passive smoking was not in place. During the six months that the law had been in effect, the number of admissions for acute MI dropped by 16 cases in comparison to the average number of admissions to previous years. Within the same period, there was an increase of 5.6 admissions per month from outside Helena.

3.24 Although this report indicated an association between reduced levels of ETS exposure and admission for MI, a number of concerns were expressed regarding study limitations (see Rapid Responses, http://bmj.bmjournals.com/cgi/eletters/328/7446/977, 30/11/04). The study was small with only 24 admissions occurring in the six months, and therefore limited in its power to detect significant differences. In addition, the authors suggested that compliance with the ban was high but no measures were taken with regard to differences in exposure levels before and after the ban. Furthermore, demographic and other information regarding patient characteristics was not provided.

3.25 Whincup et al. (2004) indicated that the risk of CHD (associated with cotinine levels greater than 0.7 ng/ml) calculated after 5 years of follow up was considerably higher than those estimated after 10, 15 and 20 years. Although these high risks could be attributed to acute effects of exposure to ETS, it is also likely that fewer incidents of CHD occurred as exposure levels decreased during the follow-up period (see 3.49 – 3.50).

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Risk Estimate</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-4 years</td>
<td>3.45*</td>
<td>(95%CI = 1.36 – 8.80)</td>
</tr>
<tr>
<td>5 – 9 years</td>
<td>1.90*</td>
<td>(95%CI = 1.09 – 3.31)</td>
</tr>
<tr>
<td>10-14 years</td>
<td>1.27*</td>
<td>(95%CI = 0.72 – 2.22)</td>
</tr>
<tr>
<td>15-20 years</td>
<td>1.09*</td>
<td>(95%CI = 0.62 – 1.76)</td>
</tr>
</tbody>
</table>

*After adjusting for age and town

**Recent Publications**

3.26 Three studies that have been published since the most recent meta-analysis are presented in **Appendix 2 Table 2.2b**. The case control studies by Pitsavos et al. (2002) and Rosenlund et al. (2001) generally supported the risk estimates that had been previously reported (although the risk estimates presented by Rosenlund are not statistically significant). Enstrom and Kabat (2003), however, reached different conclusions about the risk of CHD associated with passive smoking. An age-adjusted risk estimate of 1.01 (95%CI = 0.93 – 1.09) for Californian females exposed to ETS from a spouse who smokes was presented, based on an initial measurement of exposure taken in 1959 and followed up over about 40 years. The risks based on a later measurement (1972) are slightly higher: 1.06 (95%CI=0.90-1.25). Although there are methodological issues surrounding long follow-up periods (see 3.49 and 3.50), Enstrom and Kabat concluded that the risk of CHD associated with passive smoking may be weaker than previously thought. A number of criticisms of the paper have been published (see Letters in BMJ, 327, 30 August 2003) and, at present, no meta-analysis has included the Enstrom and Kabat findings on the risks of CHD.
3.27 The study conducted by Whincup et al. 2004 conflicts with the conclusions of Enstrom and Kabat. Cotinine levels were associated with incidence of fatal and non-fatal CHD over about 20 years of follow up. This study can be compared with that of Tunstall-Pedoe et al. (1995), which looked at never smokers in Scotland (786 men and 1492 women) between the ages of 40 and 59 in 1984/86. Serum cotinine levels were associated with current diagnosed non-fatal CHD. It is possible that the calculated hazard ratios from cotinine studies are less subject to bias from exposure misclassification and misclassification as never smokers than those which use self reporting in questionnaires.

<table>
<thead>
<tr>
<th>Table 3.1</th>
<th>Comparison of studies based on cotinine measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Whincup et al. (2004)</strong>*</td>
<td><strong>Tunstall-Pedoe et al. (1995)</strong>**</td>
</tr>
<tr>
<td>Cotinine concentration</td>
<td>Hazard Ratios</td>
</tr>
<tr>
<td>0.8 – 1.4</td>
<td>1.32 (95%CI=0.78–2.25)</td>
</tr>
<tr>
<td>1.5 – 2.7</td>
<td>1.44 (95%CI=0.83–2.50)</td>
</tr>
<tr>
<td>2.8 – 14.0</td>
<td>1.55 (95%CI=0.90–2.69)</td>
</tr>
</tbody>
</table>

* Adjusted for age and town. Hazard ratios after adjusting for further potential confounding variables were mostly higher but the dose-response pattern was less consistent.

** Adjusted for age, housing tenure, cholesterol and diastolic blood pressure

**Home versus Workplace Exposure**

3.28 Most studies included in the meta-analyses have been based on spousal exposure. Some studies, however, have reported risk from workplace exposure and exposure at home separately. The study conducted by Kawachi et al (1997) presented risk estimates associated with exposure at home only and at work only.

<table>
<thead>
<tr>
<th>Table 3.2</th>
<th>Exposure at home and work - Kawachi et al. (1997)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of Exposure</td>
<td>Home only (95%CI)</td>
</tr>
<tr>
<td>Occasional</td>
<td><strong>1.19</strong> (95%CI=0.63-2.23)</td>
</tr>
<tr>
<td>Regular</td>
<td><strong>2.11</strong> (95%CI 1.03 – 4.33)</td>
</tr>
</tbody>
</table>

The risks in the Kawachi study were higher than might be expected (especially in the workplace) and could be attributed to the intensity of exposure experienced at the time of measurement. A large (32,046) cohort of female nurses in the US were asked about exposure to ETS in the home and work in 1982. At this time there would have been few restrictions on smoking and relaxed social attitudes to smoking in the presence of others.
STROKE

3.29  Only seven studies that looked at the association between risk of stroke and exposure to ETS have been found and no meta-analysis of the studies has been conducted. Most of the studies were fairly small and whilst most found an excess risk of stroke related to exposure to ETS, the size of the effect varied considerably and the confidence intervals around the estimates were large. Only two of the reported results were statistically significant. The studies also used different endpoints associated with ETS exposure and this makes it unlikely that formal meta-analysis of all the studies could be carried out. Nevertheless, the pattern of association is clear from **Figure 3.3**. The vertical line shows a relative risk of 1; i.e. no excess risk of lung cancer. Values to the right of this line indicate an excess risk. The boxes show the average value for each study and the horizontal lines show the 95% confidence intervals around these values. If the confidence interval does not cross the vertical line, this indicates that the result is statistically significant.

**Figure 3.3 – Stroke and passive smoking (relative risk estimates)**

![Figure 3.3](image)

*With home exposure of more than 20 hours week
** Serum cotinine concentration = 0.8 – 1.4 ng/ml

N= Number of participants. Note: Participants in the studies by Lee et al., Donnan et al., You et al. and Bonita et al. are stroke cases. Participants in all other studies are cohorts.

3.30  Note that these were individual studies and the numbers were much smaller than those providing the pooled estimates for lung cancer and coronary heart disease. The smaller numbers make it less likely that a statistically significant result will be found. The relationship between active smoking and stroke is similar to active smoking and CHD. It is
plausible that the relationship also holds for passive smoking and this has not been clearly demonstrated because of a lack of studies of sufficient size.

RESPIRATORY DISORDERS

Summary

3.31 In 1992, the US Environmental Protection Agency concluded that exposure to ETS was associated with a 2-5% decrease in FEV₁ (Forced Expiratory Volume), which is a measure of lung function, and a 30 – 60% increase in respiratory symptoms. They compared these risks to those gained from light active smoking (i.e. less than 10 cigarettes/day). In 1997, the Californian Environmental Protection Agency stated that epidemiological evidence published since the US EPA report had further strengthened the association between the risk of respiratory disorders and exposure to ETS. Similarly, a more recent review (Chan-Yeung and Dimich-Ward, 2003) agreed that although further research is required, the evidence presented so far indicates that exposure to ETS adversely affects the respiratory system.

3.32 Figures 3.4, 3.5 and 3.6 show the risk estimates from studies that have looked at the association between passive smoking and certain respiratory disorders. Further information can be found in Appendix 2, Section A2.4. The vertical line shows a relative risk of 1; i.e. no excess risk of lung cancer. Values to the right of this line indicate an excess risk. The boxes show the average value for each study and the horizontal lines show the 95% confidence intervals around these values. If the confidence interval does not cross the vertical line, this indicates that the result is statistically significant. Note that these are individual studies and the numbers are much smaller than those providing the pooled estimates for lung cancer and coronary heart disease. The smaller numbers make it less likely that a statistically significant result will be found.

3.33 Nine out of ten studies that have looked at the relationship between passive smoking and lung function have reported a deficit in FEV₁ amongst those exposed. This appears to range from –0.55% to –3.8%. Two studies found significant deficits amongst men only. A meta-analysis (Carey and Cook, 1999) of 21 studies reported a pooled deficit in FEV₁ of –1.7% (95%CI= -2.8% to –0.6%).

3.34 Only a few studies (see Appendix 2) have examined the association between exposure to ETS and chronic cough, exacerbation of pre-existing asthma, chronic bronchitis and sputum production.

3.35 Overall, the risk estimates from various studies appear to support a relationship between exposure to ETS and poor respiratory health. However, the degree to which passive smoking affects the respiratory system is not clear, as few studies have been conducted, many of the confidence intervals are wide and risk estimates vary considerably between studies and different health effects.
Figure 3.4 –Wheezing and passive smoking (relative risk estimates)

Comstock et al. 1981 (N=1,802)**
Comstock et al. 1981 (N=1,802)**
Kaufman et al. 1989 (N=6,075)
Ng et al. 1993 (N=1,436)
Leuenberger et al. 1994 (N=4197)

N = Number of participants
Figure 3.5 – Dyspnea and passive smoking (relative risk estimates)

N = Number of participants

*Risk estimate is for women only from a cohort of 1,802 men and women
**Risk estimate is for men only from a cohort of 1,802 men and women.

Figure 3.6 Adult onset asthma and passive smoking (relative risk estimates)

* Associated with exposure to 150 cigarette years or more
N = Number of participants
3.36 Comparisons between studies are difficult to make due to differences in study design and quality. For example, some studies have controlled for many potential confounding variables where others have only controlled for age and sex. All of the studies can be affected by bias, especially bias from misclassification as never smokers, exposure misclassification and perhaps publication bias. These issues are discussed fully below in reference to risk of CHD and lung cancer with exposure to ETS.

PREGNANCY

3.37 A number of studies have indicated that exposure to ETS amongst pregnant women may affect the foetal growth and birth weight and of the child. In addition, exposure may increase the risk of spontaneous abortion, sudden infant death syndrome, and development of childhood cancers and cognition or behavioural problems.

3.38 In 1997, the Californian Environmental Protection Agency published a comprehensive review of the evidence regarding passive smoking and health risks to the foetus/child with both perinatal and postnatal manifestations. They concluded that small decrements in mean birth weight (25-50 grams) were associated with ETS exposure. Studies that had looked at passive smoking in pregnancy with low birth weight (less than 2500 grams) or small for gestational age had indicated an excess risk of 20 – 40%. Although few studies had examined other health effects, there was a suggestion that exposure to passive smoking may be linked with risk of neonatal mortality, spontaneous abortion and congenital malformation. There was no or little evidence linking exposure with stillbirth, sudden infant death syndrome or cognitive and behavioural problems.

3.39 More recent reviews (i.e. Windham et al. 1999; Windham, 2001) have generally supported the conclusions of the CEPA report. For example, a meta-analysis of 19 studies (Windham et al. 1999) found a pooled birth weight decrease of 31 grams (95%CI = -42 grams to -20 grams). There was also an increase in the risk of low birthweight or intrauterine growth retardation (IUGR); the pooled estimate reported was 1.2 (95%CI=1.1 – 1.3). More recently, a study conducted in the Czech Republic (Dejmek et al. 2002) which compared non-smoking mothers who were ETS exposed (N=3,713) with those not exposed (N=1,797) gave an adjusted odds ratio amongst those exposed of 1.43 (95%=1.04 – 1.97) for low birth weight. For IUGR, the odds ratio presented was 1.19 (95% CI =0.96 – 1.47). A study conducted in Norway (Haug et al. 2000) compared mean birth weights of children with smoking fathers (N=4934) and non-smoking fathers (N=11,496) born to non-smoking mothers. This study found an adjusted decrease of just 1 gram in mean birth weight. However, the authors of the study noted that fathers may not smoke in the pregnant mother’s presence because of public attitudes towards passive smoking. Similar conclusions were arrived at in a study conducted in New Zealand (Mitchell et al. 2002) that did not find any increased risk of small for gestational age where the father was a smoker and the mother a non-smoker.

3.40 A study conducted in Hong Kong (Lam et al. 2001) found higher rates of doctor consultations (adjusted OR = 1.26) and hospitalisations (adjusted OR = 1.18) amongst infants whose mothers had been exposed to ETS during pregnancy compared to those who were not exposed. The same authors found never or short duration of breast-feeding was more common (OR =1.10) amongst mothers that had been exposed to ETS in pregnancy compared to those who had not (Leung et al. 2002).
3.41 A study conducted in 2002 by the IARC (Filippini et al. 2002) in seven countries found no association between risk of childhood brain tumour and exposure to ETS at home or work during pregnancy.

3.42 Conclusions drawn from studies that have looked at passive smoking in pregnancy are limited by several factors:

- Variations in design, location and extent to which adjustments have been made for bias or confounding factors make between-study comparisons difficult to make. Despite this, consistency in an association between different studies (as in birth weight) can strengthen the evidence for a causal effect further.
- In addition, it can be difficult to accurately identify the contribution of maternal ETS exposure to postnatal manifestations when the child continues to be exposed after birth.
- Where passive exposure is from paternal tobacco smoke there may be a confounding effect from spermatogonia abnormalities, which can increase the risk of adverse health outcomes.
- Misclassification as smokers is a particular concern in these studies due to the social stigma attached to pregnant women who smoke. Dejmek et al. (2002) concluded that less than 10% of smoker/nonsmoker misclassification could be attributed to their study population based on data from cotinine studies.

QUALITY AND RELEVANCE OF THE EVIDENCE

3.43 The literature relating to lung cancer and CHD is substantial and contains a number of good quality meta-analyses of primary studies. The design of the studies that have been carried out, cohort studies and case-control studies, makes them vulnerable to the possibility of the results being affected by confounding variables and there are other sources of potential bias. However, some of the meta-analyses have taken these factors into account and have adjusted results accordingly. These studies show that there may be an effect on the size of the relative risk for exposure to ETS but adjusting for these factors does not eliminate the excess risk. Some of the potential sources of bias act in opposite directions. These issues are discussed in more detail below and apply generally to the literature on most health effects.

3.44 None of the reviews obtained included comprehensive details regarding the search strategy undertaken. The authors usually stated that studies had been located through databases such as Medline and by hand searching reference lists. Very few authors reported whether articles published in languages other than English had been excluded.

Potential sources of bias

Misclassification as never smokers

3.45 Subjects who take part in studies on passive smoking are usually required to self-report their smoking status. Research indicates that some current smokers or ex-smokers may misclassify themselves as never smokers. As smoking inflates the risk of lung cancer, this
bias will overestimate the actual risk of lung cancer. Smokers tend to marry other smokers so those who are misclassified are also more likely to be in the ETS exposed group, which in turn intensifies the effect of any misclassification bias. Biochemical markers (such as nicotine and cotinine concentrations) and repeated questionnaires have been used to test the association between actual smoking status and self-reporting. In one analysis (Wagenknecht et al., 1992), misclassification rates were estimated using data from 10 studies that had measured the cotinine levels in body fluids and compared results with self-reported smoking status. Amongst US majority females, misclassification rates amongst those who reported themselves as never smokers were 6% as occasional smokers and 0.8% as regular smokers. The misclassification rates amongst minority US females were considerably higher. Hackshaw et al. (1997) estimated that the relative risk of lung cancer associated with passive smoking of 1.24 (95%CI = 1.13 – 1.36) is reduced to 1.18 (95%CI = 1.06 – 1.30) after making adjustments for misclassification bias. Similarly, the US EPA report (1992) and Gross (1995) also adjusted their pooled relative risks for lung cancer downwards for misclassification bias.

3.46 With respect to coronary heart disease, the risk of heart diseases falls dramatically as smoking ceases and it is therefore felt that the bias from ex-smokers misclassified as never-smokers is minimal and would not explain the overall excess risk. Very few studies have therefore adjusted for smoker misclassification.

**Exposure misclassification**

3.47 The reference group in the studies are assumed to have no exposure to ETS and consequently, no increased risk of lung cancer. Urinary cotinine measures have indicated that exposure amongst non-smokers married to non-smokers is not zero. The increased exposure in the reference group will therefore reduce the relative risk estimates. Hackshaw et al. (1997) take exposure in the reference group into account and adjustments to relative risks are based on the findings that urinary cotinine concentrations amongst non-smokers living with smokers are approximately three times that of non-smokers living with non-smokers. The pooled relative risk estimate is increased to 1.42 (95%CI = 1.21-1.66).

3.48 Exposure from spousal smoking is usually measured by questionnaire. This is a crude measurement, which does not measure whether the spouse smokes in their presence, or whether there is exposure to ETS from another member of the household or exposure at work or other public places. Using cotinine as a biomarker of exposure to ETS is therefore a more valid measure of exposure.

3.49 As most studies use exposure from spouses, deaths, divorces/ separations etc. may alter exposure levels. This is a particular concern in studies that have long follow-up periods.

3.50 Another concern with studies that have long follow-up periods is that risks to health may alter over time as smoking becomes less prevalent in society, smoking behaviours change and restrictions on smoking in public places become more widespread. Many of the individual cohort studies have assessed ETS exposure only once. Risk estimates are therefore likely to be indicative of exposure levels that have actually decreased during the follow-up period.
3.51 Several of the studies that have been included in the meta-analyses have taken marriage to an ever-smoker (as opposed to a current smoker) as an indicator of exposure. Inclusion of these relative risks is likely to decrease the pooled risk estimate. The study conducted by Levois and Layard (1995), which analysed the Cancer Prevention Study (CPS) I and II dataset was heavily criticised for including never smokers married to ex-smokers. They reported no relationship between heart disease and passive smoking and used this finding to infer that publication bias had led to negative studies not being published. This prompted the American Cancer Society to conduct their own analysis of the CPS II dataset and Steenland et al. (1996) estimated an excess risk of 1.2. The analysis conducted by Steenland is used in meta-analyses in favour of Levois and Layard’s. Due to the large cohort, the study carries considerable statistical weight.

3.52 In case-control studies, it is argued that cases are more likely to over-estimate actual ETS exposure because of an increased perception as to the potential causes of their illness. Exposure levels in the workplace are difficult to assess over time (due to job changes, workplace legislation changes, recall bias etc.) and may therefore give an inaccurate picture of the association between ETS and lung cancer. There may have also been exposure to ETS in childhood. The degree to which childhood exposures inhibit or promote the risk of lung cancer is uncertain (CEPA, 1997).

3.53 The extent to which the risks associated with exposure in the home are indicative of risks associated with workplace exposure is unclear. It is generally agreed, however, that exposure in one place acts in similar ways to exposure in others and therefore exposure in the workplace (or in leisure settings) would also elevate the risk of lung cancer. The risk estimates between studies that differ in terms of design, size and location of exposure appear to be quite similar. It is also very difficult to accurately assess the degree to which exposures in different locations contribute to overall exposure. The pooled risk estimates presented in Appendix 2, which are based on spousal exposure and/or exposure from the workplace, are from studies which may or may not have excluded those who reported exposure elsewhere. For example, a study that measured exposure in the home may not have controlled for additional exposure in the workplace.

Selection bias

3.54 In some of the studies, survival bias may have influenced the results. Where subjects were too ill to respond or were deceased, the authors had to rely on surrogates to provide information regarding ETS exposure that may have affected the validity of responses.

Publication Bias

3.55 It is sometimes felt that studies (especially small studies) which report positive results (i.e. an association between passive smoking and lung cancer) are more likely to be written up than studies that report inconclusive or negative results. This results in a publication bias that skews the true estimate of risk. Copas and Shi (2000) report that just a small degree of publication bias, if it is present, would be able to ‘substantially’ reduce the risk estimates. Tweedie et al. (1996) report that publication bias could account for as much as 43% of the increased risk. It appears unlikely however, that adjustment for publication bias would rule out the effect of excess risk exposure altogether though it may reduce the overall estimate.
Law and Wald (2003) consider this issue with respect to CHD. They noted that of the 19 studies included in their meta-analyses (1997), eight were significant. They calculated that 320 studies would have to have been conducted in order to generate eight statistically significant studies by chance. In addition, they state that the inclusion of smaller studies, with less than 100 events, did not alter the risk estimates. He et al. (1999) also reported no evidence of publication bias in their meta-analysis; the correlation between variance and log relative risk was low at 0.24 (p=0.16), which indicates that small studies with negative results were not less likely to be published.

The publication of negative studies of ETS and CHD and lung cancer would, at present, have little effect on the current pooled findings as the dataset is relatively large. If there were no association between passive smoking and CHD, publication of more negative studies would be expected in the time elapsed since the first publication of an effect in 1984.

Misakian and Bero (1998) found that a publication delay for studies with non-significant (P = .001) results was evident. The median time for publication for statistically non-significant studies was 5 years compared to 3 years for statistically significant studies. There was no evidence to suggest publication bias in terms of study size, source of funding or health outcome.

Confounding factors

Dietary Confounding

In terms of health risks, there may be a protective effect from diet if it includes certain nutrients (beta-carotene, vitamin C etc.). Similarly, a poor diet (i.e. high fat) may increase the risks. It has been argued that non-smoking wives may share the same poor dietary habits as their smoking husbands. If this were true, the risk estimates of lung cancer may be elevated. Epidemiological data from around the world has, however, indicated a relationship between lung cancer, CHD, stroke, respiratory disorders and passive smoking despite wide variations in diet. The evidence regarding diet as a confounding factor is inconsistent and associations are difficult to measure due to individual and cultural differences and the complexities in assessing dietary intake accurately. Hackshaw et al. (1997) essentially rule out the effect of dietary confounding by estimating that only 2% of the excess risk of lung cancer is due to dietary confounding.

Age

Most of the reviews included the age-adjusted relative risk estimate from individual studies if it was available. For example, Zhong et al. (2000) found that 33 out of 43 studies on lung cancer had adjusted for age and these were used to calculate the pooled relative risk.
Heat sources and cooking with oil

3.61 These potential confounding factors have mainly been associated with studies conducted in Asia and may give support to separating pooled results within meta-analyses by geographical areas. The meta-analyses performed by Wang and Zhou (1997) looked at the risk of lung cancer amongst Chinese women and found no statistically significant association. The authors report that lung cancer risk factors between Chinese and American women differ widely and that other factors must be taken into consideration. The US EPA paper (1992) reports positive associations between risk of lung cancer and the use of kerosene, coal and wood for heating and cooking, although these were not always statistically significant. In addition, deep-frying, stir-frying, boiling and smokiness when cooking have also been found to be associated with an increased risk.

Pooling results

3.62 Studies that are included in a meta-analysis differ in terms of whether they present crude results only, results adjusted for confounding factors or sources of bias only, or both crude and adjusted values. Reviewers have approached the decision whether to include the crude or adjusted results differently. The US EPA report (1992) on lung cancer for example decided to include the smaller of the crude and adjusted values where both were available. Occasionally, adjustments (for publication bias, misclassification as smokers etc.) by the authors of the meta-analysis are made before or after pooling. Where the same adjustment has been made to all of the studies in the meta-analysis, a description is given in the final columns of the tables presented in Appendix 2.

3.63 All studies included in the CHD meta-analyses controlled for age and sex (though this is not true of the lung cancer studies). Despite this, there was no consistency in controlling for other potentially confounding variables. In order to assess the effects from confounding variables it is worth looking at the large studies that have been conducted; for example, when the Nurse’s Health Study (Kawachi et al. 1997) adjusted for 12 factors including diet, alcohol consumption, physical activity, body mass index etc. the RR estimate for all MI events decreased from 1.97 (95%CI= 1.20 – 3.24) to 1.71 (95%CI= 1.03 – 2.84). However, when Wells (1998a) pooled together the five studies that controlled for confounding variables most extensively, a higher RR for passive smoking and CHD mortality was found; 1.7 (95%CI= 1.3 – 2.3) for both sexes compared to 1.2 (95%CI= 1.1 – 1.4) when 12 studies were pooled. Law et al. (1997) estimate that confounding may account for a small proportion of the excess risk of heart disease in smokers. This is due to higher LDL cholesterol levels and lower intakes of fruit and vegetables amongst smokers. As never smokers married to smokers tend to share some (but not all) lifestyle characteristics it is reasonable to assume that confounding may account for up to 6% of the excess risk of heart diseases in never smokers exposed to ETS from spouses.

3.64 Although there may be confounding factors that have not been adjusted for these factors would probably affect the association between ETS and CHD in different directions. It therefore seems unlikely that any adjustment would greatly affect the risk estimates. In the most recent meta-analysis, He et al. (1999) pooled together ten out of eighteen studies that they believed controlled for important CHD risk factors. Their estimate is very similar to the combined estimate for all studies, which suggests confounding has a very small effect. Similarly, Hackshaw et al. (1997) concluded that adjusting the pooled relative risk estimate
(lung cancer) for misclassification as smokers, exposure misclassification and dietary confounding alters the unadjusted estimate only very slightly and therefore assume the original unadjusted risk estimate to be valid of the true risk.
4. EXPOSURE TO ETS AND ASSOCIATED HEALTH RISKS IN HOSPITALITY SETTINGS

SUMMARY

- Levels of exposure to ETS in hospitality settings that permit smoking are higher than those that do not allow it.
- Exposure levels in hospitality settings, when measured by air nicotine and bodily cotinine concentrations, are often higher than those found in homes and other workplaces that permit smoking.
- High exposure levels to ETS are associated with an increased risk of lung cancer, CHD, stroke and other illnesses.
- Few studies have specifically looked at health effects of hospitality workers.
- One study shows an improvement in respiratory health amongst bar workers associated with a smoking ban.

INTRODUCTION

4.1 Several studies have measured exposure levels in hospitality settings. Comparisons with other locations can therefore be made. The data indicated exposure levels in hospitality settings where smoking occurred to be higher than in areas that do not permit it. Nicotine concentration levels and cotinine levels related to exposure in hospitality settings appeared to be higher than exposure levels related to exposure at home or other workplaces. High levels of exposure to ETS clearly have implications to the health of hospitality workers. The health effects of exposure to ETS are fully explained in section 3. Studies that have looked at the health effects of a ban on smoking within the hospitality sector, show improved respiratory function and decreased reported respiratory disorder.

EXPOSURE TO ETS IN HOSPITALITY SETTINGS

4.2 A recent study by Ott et al (2003) check if this is the same reference given as in press in the reference list reported residential RSP concentration levels to be 300 µg/m³ in a bedroom where one cigarette was smoked and Klepeis et al (1999) not in reference list found mean concentrations in a parlour where one cigarette was smoked to be 65 µg/m³. Levels found in residences vary considerably and seem dependent on number of smokers, ventilation properties etc. CEPA (2004) estimate RSP concentration levels in homes to be between 300 and 5,500 µg/m³. Studies that have measured RSP concentration levels in hospitality settings before and after smoking bans are discussed in section 5. A study conducted in the UK (Carrington et al. 2003) not in reference list found median RSP concentrations in the smoking areas of pubs to be 57.3 µg/m³ where mechanical ventilation was used and 109.4 µg/m³ where extractor fans were switched on. Klepeis et al (1999) found RSP concentration levels to be 68µg/m³ above background levels in a smoking restaurant/bar in San Francisco. Maskarinec et al (2000) not in reference list measured mean RSP levels (where smoking was permitted) of 135 µg/m³ in bar areas and 29.4 µg/m³ in non-bar areas.
4.3 A review conducted by Siegel (1993) found weighted mean nicotine concentration levels in residences to be about 4.3 µg/m³ and in offices 4.1µg/m³. Studies which measured nicotine concentration levels in bars, bowling alleys, billiard halls, betting establishments and bingo parlours in the US were reviewed by Siegel and Skeer (2003). The weighted mean nicotine concentrations in bars was 31.1 µg/m³. Mean values in the other locations ranged from 9.8 µg/m³ to 76.0 µg/m³. Studies included in the review were conducted between 1975 and 2002. Mean values may therefore not be indicative of current exposure levels in these locations. A review by Hammond (1999), which focussed on studies conducted between 1984 and 1999, found nicotine concentrations levels to be 1.5 – 5.8 µg/m³ in smokers homes, 2 – 6 µg/m³ in offices in which smoking is permitted and 3 - 8 µg/m³ in restaurants that allow smoking. In 1998, Trout et al. reported exposure levels in casinos to be between 6-16 µg/m³ (geometric mean).

4.4 Mean plasma cotinine levels amongst non-smokers who live with smoking partners are about 1.67 ng/ml (95%CI=1.58-1.77) (Jarvis et al. 2001). In a small study conducted in New Zealand (Bates et al. 2002), salivary cotinine levels were measured over the course of a shift. A comparison was made between those working in bars and restaurants (permitting smoking) to workers in smoke-free environments. The mean post-shift salivary cotinine concentration for workers employed in bars and restaurants was 3.38 ng/ml compared to 0.08 ng/ml amongst those in smoke-free employments. Bergman and Johnson (1996) found similar exposure levels (mean = 3.4 ng/ml) amongst nightclub musicians in the United States. Measurements were taken over 15 performance nights. Trout et al (1998) measured plasma cotinine levels amongst casino employees in the United States. Post exposure levels were about 1.85 ng/ml. Besaratinia et al (2002) looked at students visiting a smoky pub in the Netherlands. Measurements ranged from 1.72ng/ml to 3.92ng/ml. Maskarinec et al (2000) measured salivary cotinine levels amongst bartenders and wait staff who were not exposed at home. Mean values amongst the bartenders were 2.61 ng/ml and amongst wait staff 3.67 ng/ml.

4.5 In all studies, the cotinine concentrations varied considerably between participants. This may have been due to differences in the exact location of work within a particular establishment or to individual differences in the metabolic process that influence cotinine concentration levels. Nevertheless, on average, these levels are high compared to those not exposed to ETS and also to those exposed to ETS in other places.

THE HEALTH EFFECTS OF ETS EXPOSURE AMONGST HOSPITALITY WORKERS

4.6 Epidemiological studies (see section 3) have associated exposure to ETS with an increased risk of lung cancer, CHD, stroke and certain respiratory disorders in addition to adverse health effects to the child where pregnant women are exposed. Although these associations are mainly based on exposures in the home, there are a number of studies that relate health risks to exposure in the workplace (see Appendix 2 for further details). Workers in the hospitality industry appear to be exposed to higher levels of ETS than many others and this may have both acute and long-term consequences. Few studies have looked specifically at the health effects of exposure amongst hospitality workers.
4.7 Levels of respirable particulates in the air are associated with increased rates of mortality from all causes and risk from cardiovascular and respiratory illnesses (Samet et al. 2000).

4.8 Studies by Whincup et al (2004) and Tunstall Pedoe et al (1995) (see table 3.1 and Appendix A2.3) have looked at the association between risk of heart disease and stroke with plasma cotinine levels. According to the Whincup study, cotinine levels of about 3.5 ng/ml would be associated with a risk of CHD of 1.55 (95%CI=0.90-2.69) and a risk of stroke of 2.16 (95%CI=0.80-5.80). These risks are higher than those that have been previously associated with workplace or home exposures in meta-analyses of case control and cohort studies (see Appendix A2.2a).

4.9 Siegel (1993), a reviewed five case control studies and one retrospective cohort study, relating to the risk of lung cancer amongst food service workers. All studies controlled for active smoking and other potential confounding variables. Combined, the studies indicated that there was an excess risk of lung cancer of 50% (range from 10% to 90%) amongst food service workers. These studies were, however, mainly conducted in the 1980s where few restrictions on passive smoking would have been in place and exposure levels in areas serving food would have been high.

4.10 Bates et al. (2002) asked hospitality sector workers about various respiratory symptoms experienced in the previous four weeks. The prevalence ratio was then calculated as a comparison between hospitality workers employed in places that allow smoking with unexposed workers.

<table>
<thead>
<tr>
<th>Respiratory Symptom</th>
<th>Prevalence Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wheezing</td>
<td>1.21</td>
</tr>
<tr>
<td>Frequently cough</td>
<td>1.88</td>
</tr>
<tr>
<td>Frequently phlegm</td>
<td>2.53</td>
</tr>
</tbody>
</table>

4.11 Some studies have used modelling to estimate the risk of poor health with exposure to ETS amongst hospitality workers. For example, Siegel and Skeer (2003) reviewed 13 studies that had measured ambient nicotine concentrations in various places of employment in the US. They then used the mean values to estimate lung cancer mortality risk (based on a model devised by Repace and Lowrey in 1993). Their calculations were based on a 40 hour week (Bingo = 4 hours). Table 4.1 shows lung cancer deaths attributable to workplace exposure to ETS estimated over a 40 year working period.

Table 4.1

<table>
<thead>
<tr>
<th>Place of Employment</th>
<th>Lung Cancer Mortality Estimates (of every 1000 workers)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Betting establishments</td>
<td>1.3</td>
</tr>
<tr>
<td>Bowling alleys</td>
<td>1.4</td>
</tr>
<tr>
<td>Billiard halls</td>
<td>1.7</td>
</tr>
<tr>
<td>Bars</td>
<td>4.1</td>
</tr>
<tr>
<td>Bingo parlours</td>
<td>1.0</td>
</tr>
</tbody>
</table>
4.12 Other studies which have modelled mortality risks for lung cancer amongst hospitality workers have also estimated excess risks associated with workplace exposure (reported in Dimich-Ward and Brauer, 2001). These are mostly based on exposure levels in the 1980s and, therefore, may not be indicative of current levels.

**IMPACT OF SMOKING BANS AND RESTRICTIONS ON HEALTH**

4.13 A study carried out by Eisner et al. (1998) looked at a cohort of 53 bartenders who were interviewed and provided information about respiratory symptoms before and after the prohibition of smoking in bars in California. Although 45% of the bartenders were current smokers, there was no change in smoking prevalence or quantity that could have influenced respiratory health. Forty percent of bartenders felt ETS had a slight effect on their health and 40% of bartenders felt ETS had a moderate to severe effect on their health.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Baseline (%)</th>
<th>Follow-up (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any respiratory symptom</td>
<td>74</td>
<td>32</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Wheezing</td>
<td>32</td>
<td>15</td>
<td>.02</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>19</td>
<td>8</td>
<td>.06</td>
</tr>
<tr>
<td>Phlegm production</td>
<td>53</td>
<td>11</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Cough (morning)</td>
<td>53</td>
<td>23</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

In the Eisner et al. (1998) study the bartenders also underwent spirometric assessment. Forced expiratory volume (FEV), forced vital capacity (FVC) and maximal midexpiratory flow rate (FEF) were all measured to assess changes in lung function.

<table>
<thead>
<tr>
<th></th>
<th>Mean value at baseline</th>
<th>Mean value at follow-up</th>
<th>% Change</th>
<th>% Change after controlling*</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV&lt;sub&gt;1&lt;/sub&gt;</td>
<td>3.38</td>
<td>3.42</td>
<td>1.2</td>
<td>4.5**</td>
</tr>
<tr>
<td>FVC</td>
<td>4.43</td>
<td>4.62</td>
<td>4.2</td>
<td>6.8**</td>
</tr>
<tr>
<td>FEF&lt;sub&gt;25-75&lt;/sub&gt;</td>
<td>3.37</td>
<td>3.18</td>
<td>-5.7</td>
<td>+ (but not significant)</td>
</tr>
</tbody>
</table>

*Controlling for current smoking, daily cigarette consumption and recent upper respiratory tract infections.

**QUALITY AND RELEVANCE OF THE EVIDENCE**

4.14 Measurement of all the components in ETS is not achievable due to its complex composition. Markers of exposure (i.e. RSP, nicotine and cotinine) are therefore limited in the extent to which accurate representations of ETS can be made. Nevertheless, these methods are the most favoured means of quantifying exposure levels.
4.16 Most studies have been conducted in the US and some studies have taken measurements in only one location. Exposure levels may not accurately reflect wider workplace characteristics and smoking habits/behaviours in the UK.

4.17 Measurements associated with exposure levels in the 1980s and 1990s may not be representative of current exposure levels as smoking rates change and smoking behaviours in public places become less socially acceptable.

4.18 Very few studies have assessed the health effects of exposure to ETS amongst hospitality workers. Further research in this area is required in order to fully understand the effects of exposure to ETS and the consequences of restrictions on smoking within the hospitality industry.
5. IMPACT OF SMOKING BANS AND RESTRICTIONS ON ETS EXPOSURE

SUMMARY
This section reviews evidence of the effect of smoking bans and restrictions on levels of exposure to ETS

- Studies of the introduction of workplace bans and restrictions show a reduction in exposure to ETS.
- Complete bans are associated with greater reductions in exposure to ETS than other forms of restrictions.
- Before and after studies of smoking bans in the hospitality sector have recorded reductions in exposure to ETS.

INTRODUCTION

5.1 A number of studies have been conducted that have compared workplaces with and without restrictions or have followed up (workplace) smoking bans and restrictions. A systematic review has been carried out by the US Task Force on Community Preventive Services (TFCPS) (Hopkins et al 2001). This identified 17 studies that had considered effectiveness in terms of exposure to ETS.

MEASURING EXPOSURE TO ETS

5.2 Exposure to ETS can be measured in three ways; in the air (i.e. through respirable suspended particulates (RSP) or nicotine), in biomarkers such as cotinine and through self reported exposure from questionnaires/interviews. Cotinine is a metabolite of nicotine and can therefore be used as a biomarker of exposure to environmental tobacco smoke. Cotinine can be measured in plasma, saliva and urine and has a half-life in the body of about 15 to 19 hours. Measurements are indicative of exposure over the previous 2 – 3 days. In the studies reviewed by Hopkins et al (2001), measures of environmental air quality and self-reported exposure were used. Workplace exposures will vary according to the type of occupation, working hours and length of shifts so different employees will experience different levels of exposure. In addition, exposure levels are also dependent on other factors, such as number of smokers, ventilation properties, size of room and proximity to the source of ETS etc.

IMPACT OF RESTRICTIONS AND BANS

5.3 An assessment of study quality reduced the number of eligible studies included in the Task Force report from 17 to 10 (Hopkins et al 2001). Nine of the 10 studies reported reductions in exposure. Four studies evaluated restrictions; four evaluated bans and two considered both. Seven studies evaluated changes introduced in particular worksites, and three studies were population-based surveys comparing exposure to ETS for those working in places with restrictions to those working in places without restrictions. Four studies measured air quality and six were based on self-reported exposure to ETS.
5.4 The median reduction in self reported exposure to ETS measured between 4 and 18 months after restrictions were imposed was 60%. The results for bans had values above the median (range 64% to 83%). Environmental measurements of ETS decreased by a median of 72% measured 6 to 12 months after restrictions. Two studies of bans reported reductions of 96% and 97% and one reported a reduction of 44%. Only one study of lesser restrictions reported environmental measurements and the reduction was 48%. From this small number of studies there was no clear difference in the size of reductions between measures based on air quality or self reported reductions in exposure but some indication that bans were more effective than lesser restrictions.

5.5 No studies of bans or restrictions in public settings outside the workplace were found by Hopkins et al for their review. However, some more recent studies relating to the hospitality sector have been published. Travers et al (2004) measured air quality in 20 hospitality venues in western New York before and after enactment of the Clean Indoor Air Law in 2003. In bars and restaurants (n=14) where smoking was permitted before the law took effect, the level of respirable suspended particles (RSPs) fell by 90% on average (range 73% to 98%). In 2 restaurants where smoking was permitted in an adjacent bar area, the reductions were 87% and 28%; the second venue already had low levels of RSPs measured at baseline. The 4 other venues (2 bowling alleys, a pool hall and a bingo hall) had a mean reduction of 76%. Similar results have been reported from a study in Delaware (Repace 2004). Albers et al (2004) carried out a telephone survey of self-reported exposure to ETS in Massachusetts. Residents in towns with strong regulations were more than twice as likely to report no exposure to ETS (odds ratio 2.74; 95% CI 1.97 to 3.80). In an observational study, Cains et al (2004) found that designated ‘no smoking’ areas in licensed gaming clubs in New South Wales typically produced about 50% reduction in exposure to ETS. Separate rooms were only marginally better.

QUALITY AND RELEVANCE OF THE EVIDENCE

5.6 The TFCPS review (Hopkins et al 2001) was conducted to a high standard and individual studies were assessed for quality, including the robustness of the design. The studies of specific work settings encompassed the health care sector, government and other public sector workplaces and a university. Whilst this may not be a particularly representative sample of workplaces, this is unlikely to bias the measurement or reporting of exposure to ETS with and without restrictions or bans.
6 IMPACT OF SMOKING BANS AND RESTRICTIONS ON TOBACCO USE BEHAVIOURS

SUMMARY
This section reviews evidence from studies that have related smoking bans and restrictions to changes in cigarette consumption and smoking cessation rates.

- Workplace bans and restrictions are associated with reductions in number of cigarettes smoked by continuing smokers, increases in quit attempts and successful quitting and reductions in smoking prevalence.
- Complete bans are associated with greater reductions in smoking than other forms of restriction.
- At a population level, the health gains from reductions in active smoking may be at least as great as those from reduced passive smoking.

INTRODUCTION

6.1 Four systematic reviews have been carried out on the effect of workplace smoking bans and restrictions on tobacco use behaviours, including cigarette consumption, cessation or quitting attempts, actual quitting and smoking prevalence (Chapman et al, 1999; Hopkins et al, 2001; Fichtenberg and Glantz, 2002; Levy and Friend, 2003). Levy and Friend also compare the impact of workplace restrictions with clean indoor air laws.

6.2 An assessment of study quality reduced the number of eligible studies in the Task Force review from 50 to 9, including both bans and restrictions (Hopkins et al 2001). The other studies are larger with 19 studies included by Chapman et al, 26 studies included by Fichtenberg and Glantz and 35 by Levy and Friend. The literature reviewed includes individual workplace studies, population based studies of workplaces and studies of the impact of public laws on smoking behaviour.

Cigarette consumption by continuing smokers

6.3 All of the studies reviewed by Hopkins et al (2001) measured self-reported cigarette consumption. Eight studies reported reductions in smoking with one study reporting no change. The median reduction was 1.2 cigarettes per day (range: no change to –4.3 cigarettes per day) with follow up periods up to 2 years. The individual study results were a mixture of statistically significant and non significant changes, with some studies not reporting significance levels. The statistically significant changes tended to be larger. The largest effect reported was for a smoking ban (-4.3 cigarettes per day), but in this small number of studies there was no clear difference between the size of the effect for bans or lesser restrictions.

6.4 Chapman et al (1999) pooled data from 6 prospective cohort studies; this design was considered to be the most robust. These studies showed a reduction of 3.5 cigarettes per day (20.7% of baseline level). A more recent meta-analysis, which included a larger number of
studies, gave a similar statistically significant pooled estimate of –3.1 cigarettes per day (95% CI -2.4 to -3.8) (Fichtenberg and Glantz 2002) based on comparisons of smoke free workplaces with unrestricted workplaces. Levy and Friend (2003) report that retrospective studies of individual worksites show a reduction in amount smoked of 10-20% after 6-13 months. Reductions in quantity smoked appeared to decline with length of follow up but this result could be due to light smokers being more likely to quit and being excluded from the follow up data for continuing smokers. Population based studies comparing workplaces with and without restrictions at a point in time show that 7-15% fewer cigarettes are smoked per smoker where restrictions are in place. In two population based studies, which had compared different levels of restrictions, the difference in quantity smoked per smoker in smoke free workplaces compared to workplaces with no ban was larger than the difference in quantity smoked per smoker in workplaces with lesser restrictions compared to workplaces with no ban.

6.5 Clean air laws are also effective in reducing cigarette consumption, although these results are based on per capita consumption and will combine reductions in consumption per smoker and reduced smoking prevalence. Chaloupka and Saffer (1992) estimate a 20% reduction in per capita consumption of cigarettes with clean air laws compared with 4-8% without clean air laws, after controlling for smoking sentiment. By contrast, the impact of worksite laws became insignificant when social attitudes were taken into account. Clean air laws appear to be more effective than workplace bans in reducing cigarette consumption per capita.

**Effect on smoking cessation or quit attempts**

6.6 Hopkins et al (2001) identified 4 studies that had self-reported smoking cessation data with follow up periods between 4 weeks and 18 months. Four week follow up showed no quitters but in 3 studies with 12-18 months follow up there were more quitters with a ban compared with workplaces that were not smoke free. Two of the studies show increases in quitting of 7.9 and 9.6 (not significant) percentage points. The third study reported results in terms of a relative risk of quitting of 1.7.

6.7 Chapman et al (1999) and Levy and Friend (2003) reported that results for quit rates were not consistent in prospective cohort studies of individual worksites. However, Levy and Friend found some limited evidence that quit rates show little initial effect but show greater increases over time. Some population based studies showed higher rates of quit attempts and successful quitting lasting at least 3 months. Three studies reported higher cessation rates in workplaces with bans (10-15%). However, three other studies have reported small or not significant effects.

6.8 Two studies in the Levy and Friend review provided results for the effect of clean air laws on quit rates. One reported a 12% higher mean quit rate in States with extensive clean air laws and the other reported a 38% higher 6-month cessation rate in areas with strong local ordinances.
Smoking prevalence

6.9 Hopkins et al (2001) concluded that studies reporting smoking prevalence gave inconsistent results. Three of the studies reviewed reported reductions in smoking prevalence and three reported increases. The results did not appear to be related to differences in study design. However, the meta-analysis by Fichtenberg and Glantz (2002) reported a statistically significant 3.8% reduction in absolute prevalence (pooled effect based on 19 studies) associated with smoke free workplaces (95% CI 2.8% to 4.7%). Levy and Friend (2003) found that prospective cohort studies reported reductions in smoking prevalence of 7-20% one year or more after restrictions were put in place. Population based studies comparing employees in workplaces with bans and workplaces without restrictions showed 15-20% lower prevalence. Partial restrictions had little or no effect. Other studies had reported greater differences but did not control for confounding factors.

6.10 The effect of clean air laws on smoking prevalence has only been included in a few studies. More studies have considered per capita consumption, which combines reduced prevalence and reduced consumption per smoker. As reported above, Chaloupka and Saffer (1992) estimated a 20% reduction in per capita consumption of cigarettes with clean air laws. Two other studies have estimated effects between 6% and 10% on per capita consumption. Two studies reported results for prevalence rates. One study reported mean prevalence rates were 14% lower in States with extensive restrictions and the other study reported a 13% lower smoking rate for males and a smaller effect for females. The same study reported that the impact was greatest in the 25 – 44 age group.

6.11 Some studies reviewed by Levy and Friend have looked at youth smoking but with variable results. Smoking restrictions in schools appear to be effective and a broader range of smoking restrictions may reduce youth quantity smoked and progression to established smoking. Results have been found to depend on demographic subgroup.

QUALITY AND RELEVANCE OF THE LITERATURE

6.12 All four reviews adopted a systematic approach to the literature but used different inclusion criteria. Therefore, the reviews have considered different studies, although there is considerable overlap between Chapman et al., Levy and Friend and Fichtenberg and Glantz. Unlike the studies of impact on exposure to ETS, population studies of smoking behaviour may be affected by selection bias and confounding factors. Most of the studies controlled to some degree for confounding factors. However, there may still be some degree of selection bias, in terms of either areas or workplaces that have decided to adopt restrictions differing from those that do not or in terms of the ability of individuals to exercise choice between workplaces with greater or lesser restrictions on smoking.

6.13 The specific worksites studied provide before and after information on the impact of interventions but may not be representative of the effects of restrictions in a wider workplace setting. The health care sector, in particular, was over represented in individual workplace studies.

6.14 The smoking prevalence studies provided a wide range of estimates and some of the differences may be due differences in study design and the extent to which confounding
factors have been controlled for. In particular, smoking bans may have been accompanied by other smoking cessation interventions.
7 ECONOMIC IMPACTS OF RESTRICTIONS ON SMOKING IN PUBLIC PLACES

SUMMARY
This section presents a review of literature relating to the economic impacts of smoking bans or restrictions on workplaces, in general, and on the hospitality sector in particular.

- Economic impacts for all workplaces include the net productivity effect from smoking breaks and improved air quality and reductions in sickness absence; savings on fire damage; and cleaning costs
- Only a small number of studies have estimated these costs and these are not based on direct measurement but rely on a number of assumptions.
- Studies of the impact of smoking restrictions on the hospitality sector (hotels, bars and restaurants), using objective data such as sales tax and employment, have failed to find any statistically significant effect.
- The evidence from these studies is not as robust as the evidence relating to health effects, in terms of quantity of published studies, study design and sample size. However, the findings are consistent in demonstrating small and mainly positive effects.
- These studies were designed and carried out in the context of claims that there would be a negative impact of 30% and have a very high power to detect effects of this size. The studies demonstrate that impacts of this size have not occurred in any of the locations studied.
- The possibility of a small overall negative effect cannot be ruled out, nor can the possibility of negative impacts for some businesses or small areas that are balanced out by gains elsewhere.

GENERAL EFFECTS ON ALL WORKPLACES

Introduction
7.1 A ban on smoking in enclosed public places would be expected to have an economic impact on workplaces through the avoidance of some of the costs of workplace smoking. A small number of studies have been carried out on the costs of workplace smoking. These studies include a range of costs, such as the effect of smoking breaks on productivity, the effect of air quality on productivity, fire damage caused by smoking materials and the additional cleaning and redecoration costs related to smoking. Absence due to ill health of smokers will only be saved to the extent that smokers reduce or quit smoking. Estimates reported here from this literature will consider gains only from a reduction in passive smoking. The impact of bans and restrictions on smoking behaviour has been considered in section 6.

7.2 The studies that have been carried out on the costs of workplace smoking are not based on direct measurement of costs but rely largely on modelling the costs on the basis of available data and a range of assumptions. The studies generally use the same methodology but with some variability in the assumptions made and data used. The costs reported here
should therefore be considered to be indicative of the type and magnitude of costs that could be avoided if all enclosed workplaces were smoke free.

**Productivity effects of workplace smoking**

7.3 The impact of a ban on smoking in enclosed public places on productivity in the workplace depends upon the number of smoking breaks taken and the current smoking restrictions in place. If a workplace has no restrictions in place, the introduction of a ban may result in workers taking additional breaks to smoke and a loss of productivity. However, if smoking is currently permitted in designated areas or ‘smoke rooms’ workers may already be taking more breaks and the length of these breaks may be reduced by having to smoke outside. The study by Parrott et al (2000) estimated the costs of smoking breaks in Scotland using survey data to adjust for the number of employees already covered by restrictions or bans on smoking. They produced a wide range of estimates based on alternative assumptions about frequency of smoking breaks and differences in length of smoking breaks from £289 million to £605 million (1998 prices). This annual productivity gain is based on the net effect of, first, a reduction of 20 minutes per smoking break when a building changes from having a separate room for smokers to take breaks to being smoke free, and, secondly, an increase of 10 minutes per smoking break when a building goes smoke free compared with no restrictions.

7.4 By contrast, the Health and Safety Executive (HSE) (1999) estimated only the productivity losses relating to increased time on smoking breaks when any form of smoking policy is introduced to a previously unrestricted workplace. They estimated this annual loss as £740 million but argued that this is offset by productivity gains relating to improved indoor air quality.

7.5 A study from Ireland (Madden 2003) based its approach on Parrott et al but assumed that the number of permitted smoking breaks will be more restricted following legislation to ban smoking in workplaces. The estimate for Ireland was €271m (2002 prices) per annum. A further study from Canada (Health Canada) estimated costs of smoking breaks but did not produce an estimate for the effect of a change in smoking policy.

**Absenteeism due to passive smoking**

7.6 Smoking in the workplace affects the health of both active smokers and those exposed to passive smoking and this results in increased absenteeism due to ill health. The study by Parrott et al (2000) produced estimates of the annual cost of sickness absence in Scotland due to active smoking of £40 million (1998 prices) but this total amount would not be saved by workplace smoking restrictions. The HSE (1999) have estimated costs for sickness absence relating to exposure to ETS for those with asthma and chronic bronchitis as £83m to £166m per year in Great Britain, which would imply costs in the range of £8.3m to £16.6m. These figures appear to be high in comparison to the estimate for active smoking.
Fire hazards

7.7 A ban on smoking in enclosed public places will reduce the fire hazard in workplaces from careless disposal of smoking materials. The annual cost of fire damage relating to smoking on business premises was estimated in the study by Parrott et al (2000) as £4.451 million (1998 prices). This is based on insurance claims data for the UK and estimates of the proportion attributable to smoking materials. The HSE (1999) have used a similar approach, with more recent data on insurance losses, to produce an estimate of £52 million for fire damage in Great Britain. Administration costs for the claims may add 10% to this figure. They also noted that 2 deaths and 139 injuries per year are associated with the fires. Costs to the fire service may be reduced by around £0.2 million.

Cleaning and redecoration costs

7.8 None of the published studies have estimated the effect of workplace smoking restrictions on cleaning and redecoration costs, although it is recognised that there will be an effect. The HSE (1999) cites a US survey that estimates a saving of £300 per smoker per year on cleaning and maintenance costs.

SPECIFIC EFFECTS ON THE HOSPITALITY SECTOR

Introduction

7.9 Separate consideration of the effect of smoking restrictions on the hospitality sector is based on the potential impact that might be felt on trade in this sector. However, there has been little attempt within the literature to develop a proper economic framework for analysis of the hospitality industry. From the perspective of smokers, a ban on smoking within enclosed venues can be considered either as an additional ‘cost’ of visiting the venue (having to forgo smoking or go outside) or a reduction in the enjoyment or benefit of the visit. Either of these interpretations would lead to a predicted reduction in demand. On the other hand, for non-smokers the removal of exposure to ETS provides a potential increase in the enjoyment or benefit of each visit or a reduction in ‘cost’ associated with the negative effects of tobacco smoke. This would lead to a predicted increase in demand. For some smokers, a smoke free environment may also be preferred, if the ‘benefit’ of smoking is outweighed by the ‘cost’ of being in a smoky atmosphere.

7.10 The net effect on the hospitality sector will depend on the relative strength of these effects. The effect on different parts of the hospitality sector may vary with different characteristics of venues or of patrons if these determine the effect on demand but the literature gives no insight into these factors. Rather, the studies that have been conducted have proceeded to estimate aggregate effects for specific geographic areas and specific sectors; restaurants, bars and hotels.

7.11 In one of the few theoretical contributions to the literature, Dunham and Marlow (2000) proposed an economic model in which owners adjust to customer preferences in terms of smoking restrictions. They estimate an optimal balance based on survey data. However, their approach ignores the practical problem of how potential patrons signal their preference
for smoke free environments if there are none offered. The problem of being first to go
smoke free has been addressed by Shiell and Chapman (2000), who demonstrated a classic
‘Prisoner’s Dilemma’. They demonstrate that even if it is in the individual best interest of
every owner to go smoke free, in the face of potential compensation claims from workers
exposed to ETS, individual decision making will result in none of the owners going smoke
free because they cannot be certain that all other owners will do the same.

Reviews of studies

7.12 Two reviews of studies assessing the economic impact of smoke free policies were
identified (Scollo and Lal 2004; VicHealth Centre for Tobacco Control 2002). The more
recent study appears to be an update and expansion of the former; therefore the results of
most recent review are reported. The review includes journal publications and grey literature.
A commentary on the quality of the literature reviewed has also been published (Scollo et al
2003).

7.13 Studies were reported separately according to whether they had used objective
measures of outcomes, such as sales receipts or taxes, number of businesses, number of
employees, or had used subjective outcomes, such as opinions of proprietors, predicted
frequency of visiting. The review also classified the studies by funding source using four
categories; identified source other than the tobacco industry, funding source unknown,
organisations with links to the tobacco industry at the time of the study and funded by
tobacco companies or industry groups.

7.14 The review takes into account whether the studies controlled for confounding factors
relating to economic conditions and whether the studies were peer reviewed. The studies
vary in the nature of the smoking restrictions examined; some studies refer to smoking
restrictions that may include both smoking bans and other restrictions. The majority of the
studies were concerned with the impact of restrictions affecting restaurants.

7.15 All of the objective studies have been carried out in locations in the USA, Canada or
Australia; the studies identified as having links with the tobacco industry were all conducted
in the USA and the one study in which the funding source is unknown was carried out in
Canada. None of the studies funded by sources other than the tobacco industry found
evidence of negative effects; all of the studies linked to the tobacco industry found negative
effects.

7.16 The same dichotomy of conclusion by funding source was apparent in the studies with
subjective measures of outcome (eg proprietor opinions of effect on business; predicted
frequency of visits). In some cases, studies had both objective and subjective outcome
measures and appear in both categories. In addition to studies carried out in USA, Australia
and Canada, subjective studies have been carried out in New Zealand, Hong Kong and UK.
These studies generally take the form of opinion surveys of owners or users of facilities and
may be prospective (asking what people think will happen if a restriction is put in place) or
retrospective (asking how business or behaviour has changed). As such, these studies relate
opinions about what may happen or what has happened, rather than information about what
has happened, and surveys may be subject to response bias and reporting bias. They are
discussed here for completeness but conclusions about the economic impact are drawn only
from the studies with objective data.
7.17 The views of patrons or the general public were reported in a number of prospective surveys. In studies with no tobacco industry funding, the proportions likely to increase their use were generally higher than the proportion reporting that they would reduce use. In some cases the positives and indifferent were reported together. More negative findings were reported from surveys with tobacco industry funding. Surveys of proprietors’ views were overwhelmingly negative regardless of source of funding. In the retrospective studies, with no tobacco industry funding, some studies reported more customers reducing their visits than increasing them, but the difference was only large in one study. Most of the retrospective studies funded by the tobacco industry reported proprietors’ views. More proprietors reported reductions in business than reported increases.

**Review of peer reviewed studies**

7.18 A review of the literature by Durkan and McDowell (2004) commissioned by the Office of Tobacco Control (Ireland) was critical of the quality of the objective literature. The literature has also been criticised by Dunham and Marlow (2000) and these criticisms are reflected in the discussion on quality below. The authors of this current review considered that the reporting of individual studies was limited, in terms of methods, results and conclusions. It was therefore decided to examine in more detail the available peer reviewed literature that used objective data to provide results.

**Restaurants**

7.19 A total of 11 studies had been conducted relating to the impact of smoking restrictions in restaurants. Ten of the studies had been carried out in the USA and one in Australia. The USA studies cover restrictions in New York (State and City), California and Colorado, Massachusetts, Flagstaff, Arizona and West Lake Hills, Texas. The studies were variable in the size of the geographic areas covered and the number of years of data available. Most of the study results were not statistically significant and most of the effect sizes, whether positive or negative, were small. The studies are summarised in table 7.1.

**Bars**

7.20 One study of the effects of restrictions in California has been conducted (Glantz and Smith 1997). The pooled results for bars showed that the impact on sales as a fraction of total retail sales was positive but not significant. The ratio of bar sales in cities with smoke free ordinances to bar sales in comparison cities was sensitive to the form of the model but neither result was significant. The effect of the smoke free ordinance on sales as a fraction of retail sales was reported as +0.5 with a 95% CI of –0.284 to +1.284 calculated from the standard error (linear model). Although there was no evidence of significant positive serial correlation in the residuals for the pooled estimates, results were also reported for a quadratic model. This showed an effect of 0.3 with a 95% CI of –0.68 to +1.28.
<table>
<thead>
<tr>
<th>Study and location</th>
<th>Smoking restriction</th>
<th>Results</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glantz and Smith (1994) California and Colorado</td>
<td>100% smoke-free restaurants – not necessarily including bar areas.</td>
<td>Pooled effect on either sales as a fraction of retail sales or sales as a ratio to comparison cities negative but not significant.</td>
<td>Ordinances were repealed in 2 cities; sales did not increase in one and fell in the other.</td>
</tr>
<tr>
<td>Huang et al (1995) West Lake Hills (suburb of Austin) Texas</td>
<td>100% smoke-free in all commercial premises to which public has access including restaurants and restaurants with bar areas</td>
<td>No decrease in sales.</td>
<td>Very small study of eight restaurants</td>
</tr>
<tr>
<td>Glantz and Smith (1997) California and Colorado</td>
<td>100% smoke-free restaurants – not necessarily including bar areas.</td>
<td>Pooled results: fraction of total retail sales for restaurants no change and still not significant, ratio of sales compared with comparison city still negative but smaller and still not significant.</td>
<td>Follow up of Glantz and Smith (1994) study with additional data.</td>
</tr>
<tr>
<td>Sciacca and Ratliff (1998) Flagstaff, Arizona.</td>
<td>Smoke-free restaurants. Bar areas excluded if separately enclosed.</td>
<td>Trends in restaurant sales before and after the introduction of the ordinance showed no significant change.</td>
<td>Ratios with restaurant sales in other areas and retail sales produced similar results.</td>
</tr>
<tr>
<td>Bartosch and Pope (1999) Massachusetts</td>
<td>100% smoke-free including bar areas or a separately enclosed and ventilated room.</td>
<td>Estimated effect on meal sales for all restaurants +1.6% (95% CI: -4.0% - +7.2%). Estimated effect on meals sales for restaurants serving alcohol +3.7% (95% CI: -19.5% - +26.9%).</td>
<td>Free-standing bars where food is incidental not covered in 15 areas. 20 areas allowed appeals for variances. When these are excluded, effect is more positive but has greater variance.</td>
</tr>
<tr>
<td>Hyland et al (1999) New York City (NYC)</td>
<td>Indoor area of restaurants smoke-free unless fewer than 35 seats. Smoking permitted in bar areas of restaurants – six feet separation required or ceiling to floor partition or wall.</td>
<td>Positive but not significant effects for restaurants</td>
<td>Stand-alone bars exempt if alcohol sales 40% or more of total revenue.</td>
</tr>
<tr>
<td>Hyland and Cummings (1999) NYC</td>
<td>Restrictions in dining areas of restaurants: NYC applies only if 35 seats or more</td>
<td>Growth in number of restaurants in NYC similar to rest of state. 3 times the rate of employment growth. Other 2 counties adopting restrictions: slightly lower growth in number of restaurants. Similar employment growth to rest of state.</td>
<td></td>
</tr>
<tr>
<td><strong>Study</strong></td>
<td><strong>Location</strong></td>
<td><strong>Description</strong></td>
<td><strong>Results</strong></td>
</tr>
<tr>
<td>-----------</td>
<td>--------------</td>
<td>----------------</td>
<td>-------------</td>
</tr>
<tr>
<td><strong>Hyland et al (2000)</strong>&lt;br&gt;Erie County, New York State</td>
<td>1990 New York State restricts smoking to 30% seated capacity (or sufficient area to meet customer demand) – capacity less than 50 exempt&lt;br&gt;1st Jan 1997 Erie County reduces capacity to 20% and removes exemption&lt;br&gt;1st Jan 1998 ban implemented – smoking permitted in bar area or separate room.</td>
<td>Small reductions in employment and number of restaurants not related to the phasing in of ban – effects disappear when adjusted for changes in population and underlying economic trends.</td>
<td>Short term follow up to end of 1998.</td>
</tr>
<tr>
<td><strong>Bartosch and Pope (2002)</strong>&lt;br&gt;Massachusetts</td>
<td>Smoking restrictions could include separate ventilated area</td>
<td>All establishments: effect on rate of growth in sales 0.25% (95% CI: -1.32 to +1.81%)&lt;br&gt;Alcohol serving establishments: effect on rate of growth in sales 2.5% (95% CI: -1.48 to +6.48%)</td>
<td>Follow up to <strong>Bartosch and Pope (1999)</strong>. 12 localities required physical segregation or separate ventilation. Authors tested the effect of leaving out these 12 and results did not change.</td>
</tr>
<tr>
<td><strong>Wakefield et al (2002)</strong>&lt;br&gt;South Australia</td>
<td>Smoke free restaurants but with exemptions</td>
<td>No significant effect of smoking ban on any models</td>
<td>Not able to detect differential impact with and without exemptions. 10% had exemptions at Sept 2000 mostly in hotels and licensed clubs. Data from these not included in restaurant and café data.</td>
</tr>
<tr>
<td><strong>Hyland et al (2003)</strong>&lt;br&gt;New York State</td>
<td>Hotels and restaurants. 100% smoke free dining areas/no smoking in restaurants unless separate ventilation</td>
<td>Negative but not significant impact on restaurant employment. Positive and significant impact on per capita sales for both hotels and restaurants.</td>
<td></td>
</tr>
</tbody>
</table>
Hotels and tourism

7.21 Four studies have considered the effect of smoking restrictions on hotel business, as a proxy for tourism, or on tourist numbers directly. Hyland et al (1999) examined the effect of restrictions in New York (NY) and found a positive but not significant effect for hotel sales. There was a negative but not significant effect for the ratio of hotel sales in NY City to the rest of NY State. A further study (Hyland et al 2003) found a positive but not significant impact on hotel employment and a positive and significant effect on sales.

7.22 Glantz and Charlesworth (1999) considered the impact of smoke-free restaurants on hotel room revenues as an indicator of impact on tourism and also looked at visitor numbers from destinations that it had been claimed would be affected by smoking restrictions (Japan/Asia and Germany/Europe). The smoke-free ordinances may allow smoking in bar areas of restaurants. Nine locations were included; 3 states and 6 cities chosen on the basis that impact on tourism had been used to argue against the implementation of the ordinance.

7.23 Most of the results were either not significant or positive. Only one location reported a significant negative impact on hotel room revenues; when revenues were considered as a percentage of retail sales (to control for local economic conditions) this remained significant and negative. However, an earlier study on the same location (Sciacca and Ratliff 1998) found no significant difference in the trend of hotel sales before and after introduction of the ordinance. Compared with US hotel revenues as a whole there were 2 localities with negative effects, 2 with positive effects and 5 not significant. The figures were estimated over a fairly short time series. The effect on tourist numbers was either not significant or positive. Note that no attempt was made to adjust for prevailing economic conditions in the countries of origin for the tourists.

7.24 The smoking restrictions allowed for an exemption for bar areas, provided food areas were smoke-free. One of the locations, Boulder, allowed the construction of a separately ventilated smoking room but officials reported the use of such rooms as rare. This was despite the fact that this was one of the locations that showed a negative and significant effect on hotel revenues compared with national revenues. Flagstaff and Mesa allowed for applications for hardship exemptions. Flagstaff had granted none (despite negative and significant results on all measures); Mesa had granted applications to 3.5% of businesses (positive and significant results on all measures).

QUALITY AND RELEVANCE OF THE LITERATURE

7.25 There are a number of valid criticisms relating to the quality of the studies carried out in this area and these reflect the considerable difficulties of conducting research into policy impacts. These issues have been raised in commentaries on the literature by Dunham and Marlow...
(2000), Scollo et al (2003) and Durkan and McDowell (2004). The key issues are discussed below and these raise concerns about the precision of the results in this literature. However, it is important to bear in mind that the studies were not designed to provide precise estimates of the size of any impact but to investigate the likelihood that there would be a negative impact as great as 30%, a figure that has been proposed in a number of settings but has no empirical foundation. To quote the authors of one study:

“A power analysis indicates low power to detect an association if one truly exists using the taxable sales data for the regression models. The main problem is that the taxable sales data obtained for this study are subject to too much misclassification and bias to detect small effects. However, the real question of interest to policymakers is whether smoke-free restaurant legislation is associated with large decreases in business.” [emphasis added]

Hyland et al (1999 p20)

7.26 Furthermore, Glantz and Smith (1997) indicate that their study of bars and restaurants was powered to detect a reduction of 30% in revenues. A fair summary of the evidence, therefore, would be that the failure to find any significant impact on revenues in the sectors analysed does not rule out the possibility of a small negative effect on business but it does weigh against a large negative impact being experienced. If such large effects were experienced and were widespread then it seems unlikely in the extreme that no objective data have been produced to substantiate these effects. However, it is also the case that there has been no analysis of impacts within sectors, implying that individual businesses or smaller geographic areas could be adversely affected, and no analyses based on measures such as volume or profits were reported in the reviews. (One study examining the profitability of restaurants and bars has been published since the reviews were conducted (Alamar and Glantz 2004). Studies have not reported on industry issues, such as the degree of competition in local markets, which might explain local variations in the impact of smoking restrictions.

Inadequacy of sales tax data

7.27 The majority of studies using objective data have relied on aggregate sales tax data to examine sector activity before and after the introduction of smoking restrictions or bans. A number of criticisms have been made relating to these data. First and foremost, these data measure expenditure in the sector and not the volume of activity. Thus, it has been suggested that the lack of change in sales tax revenue could be the result of a combination of volume and sales changes producing level expenditure. Whilst this is certainly possible, these suggestions are not entirely plausible in terms of economic theory. If business volume fell following the imposition of a restriction or ban, then the finding that
expenditure levels remained the same would imply either that businesses raised their prices to offset the loss of revenue or that they cut prices sufficiently to induce additional demand that would both replace the lost business resulting from the smoking restriction and generate additional business to replace the revenue lost through cutting prices. The first scenario, a price rise, would be expected to reduce demand further. The second scenario seems unlikely and no empirical evidence has been offered in support. Where studies have been able to look at price data, these have failed to show any change in relevant prices (Hyland et al 1999; Wakefield et al 2002).

7.28 A further possibility exists, and this relates to a second criticism of the data, which is that it cannot detect different effects in different parts of the sector. If there are differential effects, and if more expensive establishments gain business and less expensive establishments lose business then this could produce a situation in which the total volume of business is reduced and the effect is masked by the shift within the market. Again, there has been no analysis to support this hypothesis and price data that are based on expenditure per meal would capture this effect. A small number of studies have used measures that might be more closely associated with volume of business, such as employment levels, and the findings from these studies do not differ from those that use sales tax data. However, it is the case that there is a lack of research on intrasectoral effects of smoking restrictions and bans.

**Timing of the intervention**

7.29 The studies generally adopt a before and after design, comparing periods before restrictions with periods after restrictions or model the effect by including a dummy variable for the months or years when legislation is in force. However, the intention to legislate has usually been announced and debated for some time before it is enacted and comes into force. It has been argued that the impact of the legislation may be spread over this period and is, therefore, underestimated. Again, there is no empirical evidence to support this view and it seems likely that a very large impact on businesses would still be detected even if it were diffused over a period of time.

**Limitations of the restrictions**

7.30 In terms of the relevance of the peer reviewed objective studies, it is important to note that many of the restrictions examined are described as ‘smoke-free’ but may fall some way short of a complete smoking ban. Typical exemptions relating to indoor areas of restaurants are: that smoking may be permitted in bar areas provided these are separated from the food serving area either by a specified distance or by a
floor to ceiling partition or wall; smoking may be permitted in a separately enclosed and ventilated room if the establishment has more than one room; smaller establishments may be excluded; and exemptions may be granted for ‘hardship’.  

7.31 However, a few areas have added bar restrictions to earlier restaurant restrictions and still report no significant effect on business (Glantz and Smith 1997). Areas where restrictions have increased incrementally report no significant effect on business (Hyland et al 2000). Analysis of restrictions in force in Massachusetts tested the effect of including or excluding areas with less restrictive provisions and found that this had no impact on the results (Bartosch and Pope 1999; 2002). Two areas where hardship exemptions were permitted reported none having been granted in one area and exemptions affecting 3.5% of businesses in another area (Glantz and Charlesworth 1999). It should also be clear that these exemptions were known when industry forecasts of adverse effects on business were being made.
Compliance

7.32 The finding of no effect from restrictions would be unsurprising if the restrictions were not complied with and some studies have been criticised for failing to establish levels of compliance with the restrictions. Wakefield et al (2002) reported high compliance. However, separate studies of compliance have been carried out in some of the areas studied. Hyland et al (1999) report high levels of compliance with the New York City Smoke-Free Air Act, which took effect in 1995. This was based on a survey of restaurateurs, unannounced inspections by Health Department inspectors and official complaints. Over two thirds of restaurants were fully compliant on the first two measures and over 90% of the instances of non-compliance in inspected premises were due to problems with configuration rather than outright smoking in prohibited areas. Recorded complaints about non-compliance were quite high in the initial period after the law took effect but declined over time. Weber et al (2003) found increasing compliance with the California Smoke-Free Workplace Law between 1998 and 2002 but absolute levels of compliance remained lower in freestanding bars (75.8%) than restaurants (98.5%).

Selection bias

7.33 Where some communities have adopted restrictions and others have not, comparisons may be subject to selection bias; i.e. the communities adopting the restrictions differ in some way from communities not adopting restrictions and this difference affects the outcome. This is an unavoidable problem with policy research as random adoption of change is unlikely to occur. Studies have used matching or statistical methods to control for potential confounding factors but the possibility remains that the effects observed in communities that have been studied are not typical. This does not negate the findings of the studies but does potentially limit the transfer of results to other settings (see below).

Transferability of results

7.34 Three main issues affect the transferability of results from research conducted in other countries. One relates to social and cultural attitudes that may influence how the public respond to smoking restrictions and bans. Communities that have adopted legislation to restrict smoking may be those that are most likely to have widespread support for such restrictions and where decisions to visit establishments in the hospitality sector may be least adversely or even positively affected by smoke-free ordinances. A second factor is climate, in that restrictions generally apply only to indoor areas and warmer drier climates offer greater opportunities for outdoor eating and drinking areas. However, the
studies in the literature produce consistent results across a range of locations from California to New York and Massachusetts. The final factor, which has not been explicitly considered in the literature, is local market conditions.
8 CONCLUSIONS AND RECOMMENDATIONS FOR FURTHER RESEARCH

CONCLUSIONS

8.1 There is a substantial literature on the health effects of ETS and strong evidence that exposure to ETS increases the risk of mortality and morbidity from lung cancer and CHD. There is less evidence relating to stroke and respiratory disorders but sufficient to suggest a link. There is also evidence to link exposure to ETS with low birth weight and a number of other health problems.

8.2 Smoking bans and restrictions have been shown to reduce exposure to ETS. Smoking bans produce a greater reduction in exposure to ETS than other types of smoking restrictions.

8.3 There is evidence that smoking bans and restrictions have been associated with reductions in the number of cigarettes smoked by continuing smokers, increased quit attempts and reduced smoking prevalence. However, the results may be subject to selection bias and confounded by other smoking cessation initiatives carried out at the same time. The precise size of the effect on smoking prevalence is therefore unclear. However, even a modest reduction in active smoking would produce benefits at a population level as large as those associated with reducing passive smoking.

8.4 There are costs associated with smoking at work, some of which would be averted if smoking were banned. Costs associated with fire damage caused by smoking materials and costs of cleaning and redecoration would be saved immediately. There may also be productivity gains associated with reduced length of smoking breaks. Reductions in passive smoking will reduce productivity losses associated with absenteeism and further gains will be realised if active smoking is also reduced.

8.5 A relatively small number of published studies have measured the impact of smoking bans and restrictions on the hospitality industry using objective data. These studies consistently find small effects, most of which are positive. These studies demonstrate that large negative effects have not occurred but the effects do not differ significantly from zero and the possibility of small negative impacts cannot be ruled out. The studies have been carried out in the USA and Australia and results may not transfer exactly to other settings.
RECOMMENDATIONS FOR FURTHER RESEARCH

8.6 There are a number of areas where the evidence base could be strengthened. In respect of health gains, the volume of evidence relating to stroke and respiratory disease was less than for lung cancer and CHD. Larger studies or meta-analysis of existing studies would be required to produce more precise estimates of the effect of exposure to ETS.

8.7 The impact of smoking bans on smoking cessation could not be estimated precisely as there was limited information about the impact of confounding factors; in particular, whether or not other smoking cessation interventions were implemented at the same time. There were also concerns about how representative the workplaces were where smoking bans and restrictions had been studied. Examination of the primary literature may enable some resolution of this issue.

8.8 The estimates for the costs of smoking in the workplace were quite limited in their number and methods. Given the relative size of the estimates relating to smoking breaks, better estimates relating to the frequency and timing of smoking breaks under alternative restrictions would improve the precision of the estimates.

8.9 The evidence base for the economic impacts of smoking restrictions on the hospitality sector is not particularly robust. Some of the problems in research design are unavoidable given that the impact of restrictions can only be evaluated where they have been implemented. However, the impacts could be estimated more precisely, and the effect on different types of business or different locations could be examined, if studies were carried out in the future at the level of individual businesses or outlets. Such studies would have to be based on objective data. Consideration should be given to the feasibility of using existing secondary data or to the development of a panel of businesses representative of the whole sector and providing verified data on economic activity.
REFERENCES


California Environmental Protection Agency 1997, Health effects of exposure to environmental tobacco smoke, National Cancer Institute, National Institutes of Health, Sacramento.


Appendix 1 – Search Strategies

The impact of smoking bans

MEDLINE (Ovid) 1966-2004 (August week 2)

1. Tobacco Smoke pollution/lj, pc [Legislation & Jurisprudence, Prevention & Control]
2. SMOKING/lj.pc [Legislation & Jurisprudence, Prevention & Control]
3. (smok$ adj3 ban$).tw
4. (tobacco adj3 ban$).tw
5. (smok$ adj3 control$).tw
6. (tobacco adj3 control$).tw
7. (smok$ adj3 restrict$).tw
8. (tobacco adj3 restrict$).tw
9. (smok$ adj3 ordinance$).tw
10. (tobacco adj3 ordinance$).tw
11. or/1-10
12. exp “Outcome and Process Assessment (Health Care)”/
13. exp Health Status Indicators/
14. Program Evaluation/
15. Health Behavior/
16. Patient Compliance/
17. impact.tw
18. or/12-17
19. 11 and 18

ISI Databases (Web of Science)
TS=((smok* ban* or tobacco ban* or smok* control* or tobacco control* or smok* restrict* or tobacco restrict* or smok* ordinance*) and (morbidity or mortality or impact or outcome or health status or exposure or behavior)) DocType=All document types; Language=All; Database(s)=SCI-EXPANDED, SSCI. Timespan-1981-2004
Passive smoking and Coronary Heart Disease

MEDLINE

1. Cardiovascular Diseases/
2. Cardiovascular System/
3. 1 or 2
4. Tobacco Smoke Pollution/
5. 3 and 4
6. animal/
7. human/
8. 6 not (5 and 6)
9. 5 not 8

EMBASE

1. Tobacco Smoke/
2. Environmental Exposure/
3. 1 and 2
4. Passive Smoking/
5. tobacco smoke pollution.tw.
6. or/3-5
7. cardiovascular system/
8. Cardiovascular Disease/
9. 7 or 8
10. 6 and 9
11. animal/
12. human/
13. 11 not (10 and 11)
14. 10 not 13
15. from 14 keep 1-115
Passive smoking and Lung Cancer

MEDLINE

1. Tobacco Smoke Pollution/
2. exp Lung Neoplasms/
3. exp Lung Neoplasms/
4. 1 and 2
5. Animals/
6. Human/
7. 5 not (5 and 6)
8. 4 not 7
9. 4 not 7

EMBASE

1. Tobacco Smoke/
2. Environmental Exposure/
3. 1 and 2
4. Passive Smoking/
5. Indoor Air Pollution/
7. tobacco smoke pollution.tw.
8. or/4-7
9. exp Lung Cancer/
10. 8 and 9
11. animal/
12. human/
13. 11 not (11 and 12)
14. 10 not 13
SCIENCE CITATION INDEX AND SOCIAL SCIENCE CITATION INDEX

TS=((passive smoking or environmental tobacco smoke or tobacco smoke pollution) AND (lung cancer* or lung neoplasm*))
Passive smoking and Stroke

MEDLINE

1. Tobacco Smoke Pollution/
2. Cerebrovascular Disorders/
3. stroke/
4. 2 or 3
5. 1 and 4

Passive smoking and Respiratory Disorders

MEDLINE

1. Tobacco Smoke Pollution/
2. respiratory system/ or respiratory tract diseases/
3. 1 and 2
4. 1 and 2
5. 3 not child$.mp. [mp=title, original title, abstract, name of substance, mesh subject heading]
### APPENDIX 2  HEALTH EFFECTS OF ETS

#### A2.1: Lung cancer

Table A2.1a – Exposure to ETS and Lung Cancer – Meta Analyses

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>No. of Studies</th>
<th>Inclusion Criteria</th>
<th>Pooled RR (A)</th>
<th>Pooled RR (B)</th>
<th>Pooled RR (C)</th>
<th>Pooled RR (D)</th>
<th>Pooled RR (E)</th>
<th>Adjustments common to all included studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blot and Fraumeni (1986)</td>
<td>12</td>
<td>Home/Female</td>
<td>1.3 95%CI = 1.1–1.5</td>
<td></td>
<td></td>
<td>1.7 [N=7] 95%CI = 1.4–2.1 (≥15 cigarettes/day)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>USNRC (1986)</td>
<td>13</td>
<td>Home/Female</td>
<td>1.32 95%CI = 1.16–1.52</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Wald et al. 1986</td>
<td>13</td>
<td>Home</td>
<td>1.35 95%CI = 1.19–1.54 (both sexes)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wells (1988)</td>
<td>17</td>
<td>Home/Female</td>
<td>1.44 95%CI = 1.26–1.66</td>
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</tr>
<tr>
<td>Saracci and Riboli (1989)</td>
<td>14</td>
<td>Home</td>
<td>1.35 95%CI = 1.20–1.53 (both sexes)</td>
<td></td>
<td></td>
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<tr>
<td>Lee (1992)</td>
<td>28</td>
<td>Home/Female</td>
<td>1.18 95%CI = 1.07–1.30</td>
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<tr>
<td>Tweedie and Mengersen (1992)</td>
<td>26</td>
<td>Home/Female</td>
<td>1.17 95%CI = 1.06–1.28 1.05*, 1.076**</td>
<td></td>
<td></td>
<td>1.10 95%CI = 0.95–1.27</td>
<td></td>
<td>*Smoker misclassification ** Smoker misclassification and exposure in ‘unexposed’ group</td>
</tr>
<tr>
<td>Study</td>
<td>N</td>
<td>Gender</td>
<td>Risk Ratio (95% CI)</td>
<td><em>95% CI</em></td>
<td>Smoker misclassification</td>
<td></td>
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<td>-------------------------------------------</td>
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<tr>
<td><strong>US EPA (1992)</strong></td>
<td>31</td>
<td>Home/Female</td>
<td>1.19 [N=11] 90%CI = 1.04–1.35</td>
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<td></td>
<td>1.81 [N=17] 90%CI = 1.60–2.05</td>
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<td></td>
<td></td>
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<td>≥16cigs/day</td>
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<tr>
<td><strong>Gross (1995)</strong></td>
<td>32</td>
<td>Home</td>
<td>1.18 [N=31] 95%CI = 1.06–1.28</td>
<td></td>
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<tr>
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<td></td>
<td></td>
<td>1.12 [N=13] 95%CI = 0.99–1.26</td>
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<td></td>
<td></td>
<td></td>
<td>1.00* 95%CI = 0.88–1.14</td>
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<td>90%CI = 1.04–1.35</td>
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<td>95%CI = 1.06–1.28</td>
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<td></td>
<td>95%CI = 0.99–1.26</td>
<td></td>
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</tr>
<tr>
<td><strong>Pershagen (1994)</strong></td>
<td>25</td>
<td>Home/Female</td>
<td>1.23 95%CI = 1.11-1.36</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>1.18 95%CI = 1.08-1.41</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>1.12* 95%CI = 0.99–1.26</td>
<td></td>
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<tr>
<td><strong>Mengersen et al. 1995</strong></td>
<td>34</td>
<td>Home/Female</td>
<td>1.23 95%CI = 1.08-1.41</td>
<td></td>
<td></td>
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<tr>
<td><strong>Dockery and Trichopoul os (1996)</strong></td>
<td>33</td>
<td>Home/Female</td>
<td>1.27 95%CI = 1.18-1.38</td>
<td></td>
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</tr>
<tr>
<td><strong>Law and Hackshaw (1996)</strong></td>
<td>34</td>
<td>Home</td>
<td>1.24 95%CI = 1.11 -1.38</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>(both sexes)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td><strong>Wang and Zhou (1997)</strong></td>
<td>5</td>
<td>Home/Female</td>
<td>0.91 95%CI = 0.75-1.10</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(China only)</td>
<td>1.17 95%CI = 1.05-1.31</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.58 [N=10] 95%CI = 1.14–2.19</td>
<td></td>
<td></td>
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</tr>
<tr>
<td><strong>Hackshaw et al. 1997</strong></td>
<td>39</td>
<td>Home</td>
<td>1.24 [N=37] 95%CI = 1.13-1.36</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.17 [N=14] 95%CI = 1.05-1.31</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.58 [N=10] 95%CI = 1.14–2.19</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Merletti et al. 1998</strong></td>
<td>39</td>
<td>Home</td>
<td>1.24 95%CI = 1.15-1.34</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Taylor et al. 2001</strong></td>
<td>43</td>
<td>Home</td>
<td>1.29 95%CI = 1.17-1.43</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.22 [N=19] 95%CI = 1.10-1.35 (Western countries)</td>
<td></td>
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</tr>
</tbody>
</table>

*Misclassification at 2%
<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>Setting</th>
<th>RR</th>
<th>95% CI</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wells (1998) (b)</td>
<td>5</td>
<td>Work</td>
<td>1.39</td>
<td>1.15-1.68</td>
<td>*misclassification (on RE model of unadjusted RR = 1.20)</td>
</tr>
<tr>
<td>Levois and Layard (1994)</td>
<td></td>
<td>Home and Work</td>
<td>1.39</td>
<td>1.15-1.78</td>
<td>(US only)</td>
</tr>
<tr>
<td>Tweedie, et al. 1996</td>
<td>40</td>
<td>Home and Work</td>
<td>1.39</td>
<td>1.02-1.26</td>
<td>*publication bias</td>
</tr>
<tr>
<td>Zhong et al. 2000</td>
<td>40</td>
<td>Home and Work</td>
<td>1.39</td>
<td>1.00-1.22</td>
<td>**misclassification (on RE model of unadjusted RR = 1.20)</td>
</tr>
<tr>
<td>Boffeta (2002)</td>
<td>51</td>
<td>Home and Work</td>
<td>1.39</td>
<td>1.04-1.32</td>
<td>*Age and demographic characteristics</td>
</tr>
</tbody>
</table>
Key to Table A2.1a

RR = Relative Risk  
NA = Not available  
N = Number of studies reviewed  
CI = Confidence Interval

A = never smoking women ever exposed to ETS by spouses who smoke  
B = never smoking women ever exposed to ETS by spouses who smoke (US only)  
C = never smoking women ever exposed to ETS by spouses who smoke and risk of squamous and small cell carcinoma  
D = never smoking women exposed to ETS by spouses who smoke more than 20 cigarettes/day  
E = never smoking women exposed to ETS by spouses who have smoked for more than 20 years  
F = ever exposure to ETS in the workplace (both sexes)

Table A2.1b – Exposure to ETS and Lung Cancer – Epidemiological studies published 2000 – 2004

<table>
<thead>
<tr>
<th>Author (s)</th>
<th>Study</th>
<th>Inclusion criteria</th>
<th>No. of LC cases (amongst never smokers)</th>
<th>RR (A)</th>
<th>RR (B)</th>
<th>RR (C)</th>
<th>RR (D)</th>
<th>RR (E)</th>
<th>RR (F)</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Enstrom and Kabat (2003) | CH    | Participants in American Cancer Society prevention study (CPS 1) -- Californian | 75           | **0.93** (95%CI=0.65-1.33) | *0.88* (95%CI=0.60-1.28) |          |        |        |        | Adjusted for age. Followed 1960 – 1998  
|                      |       |                    |                                         |             |        |        |        |        | *Age, race, education level, exercise, BMI, urbanisation, |
| Brennan et al. 2004 | CC (pooled analysis from 2 studies) | Lung cancer patients in 8 countries | 1,263 | **1.18** (95%CI=1.01-1.37) for both sexes | **1.22** (95%CI=0.80-1.85) for exposure to spouse (both sexes) who smokes 16 cigs/day or less. N=457 cases | **1.05** (95%CI=0.86-1.29) for exposure of 16 years or more in both sexes. N=480 cases | **1.13** (95%CI=0.97-1.31) N= 729 cases | Adjusted for age, centre and gender | 

Results restricted to women only are ‘practically identical’ |

---

**Key to Table A2.1b**

RR = Relative Risk

N = Number of studies reviewed

CI = Confidence Interval

NA = Not available

A = never smoking women ever exposed to ETS by spouses who smoke

B = never smoking women ever exposed to ETS by spouses who smoke (US only)

C = never smoking women ever exposed to ETS by spouses who smoke and risk of squamous and small cell carcinoma

D = never smoking women exposed to ETS by spouses who smoke more than 20 cigarettes/day

E = never smoking women exposed to ETS by spouses who have smoked for more than 20 years

F = ever exposure to ETS in the workplace (both sexes)
A2.2: Coronary heart Disease

Table A2.2a – Exposure to ETS and Coronary Heart Disease – Meta Analyses

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>No. of Studies</th>
<th>Inclusion Criteria</th>
<th>Pooled RR (A)</th>
<th>Pooled RR (B)</th>
<th>Pooled RR (C)</th>
<th>Pooled RR (D)</th>
<th>Pooled RR (E)</th>
<th>Adjustments common to all included studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glantz and Parmley</td>
<td>10</td>
<td>Home</td>
<td>1.3</td>
<td>95%CI=1.2-1.4</td>
<td></td>
<td></td>
<td></td>
<td>Age</td>
</tr>
<tr>
<td>(1991)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wells</td>
<td>10</td>
<td>Home</td>
<td>1.42</td>
<td>95%CI=1.15-1.75</td>
<td>1.37*</td>
<td></td>
<td></td>
<td>Age, *smoker misclassification</td>
</tr>
<tr>
<td>(1994)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Law et al. 1997</td>
<td>19</td>
<td>Home</td>
<td>1.30</td>
<td>95%CI=1.22-1.38</td>
<td>1.23*</td>
<td></td>
<td></td>
<td>Age, *diet</td>
</tr>
<tr>
<td>Thun et al. 1999</td>
<td>17</td>
<td>Home</td>
<td>1.25</td>
<td>95%CI=1.17-1.33</td>
<td>95%CI=1.14-1.30</td>
<td>1.22</td>
<td>N = 8</td>
<td>Age</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.32</td>
<td>95%CI=1.04-1.67</td>
<td>95%CI=1.13-1.32</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Author(s)</td>
<td>No. of Studies</td>
<td>Inclusion Criteria</td>
<td>Pooled RR (A)</td>
<td>Pooled RR (B)</td>
<td>Pooled RR (C)</td>
<td>Pooled RR (D)</td>
<td>Pooled RR (E)</td>
<td>Adjustments common to all included studies</td>
</tr>
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<td>------------------------------------------</td>
</tr>
<tr>
<td>Wells (1998) (a)</td>
<td>18</td>
<td>Home and work</td>
<td>1.28 (N = 6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.18 (N = 8)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(95%CI = 1.02-1.61) for morbidity only</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(95%CI = 1.04-1.34)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.21 (N = 6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.34</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(95%CI for mortality only)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.14 (N = 4) (95%CI = 0.99-1.32)</td>
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<td></td>
<td></td>
<td></td>
<td>1.49 (N = 18)</td>
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<td>Mortality only</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>(95%CI=1.29-1.78) for all home exposures</td>
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<td></td>
<td></td>
<td></td>
<td>1.32 (N = 6) (95%CI = 1.01-1.72)</td>
</tr>
<tr>
<td>He et al. 1999</td>
<td>18</td>
<td>Home and work</td>
<td>1.25 95%CI = 1.17–1.32</td>
<td>1.31 (N = 7)</td>
<td>1.29 (N=6)</td>
<td>1.11 (N=8)</td>
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<tr>
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<td></td>
<td>1.26* (N=10)</td>
<td>(95%CI = 1.16–1.38)</td>
<td>(95%CI = 1.16–1.43)</td>
<td>(95%CI = 1.00–1.23)</td>
<td></td>
<td>Age</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.24 (N = 14)</td>
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<td></td>
<td></td>
<td></td>
<td>*Risk factors for CHD (i.e. blood pressure, body weight, serum cholesterol)</td>
</tr>
<tr>
<td>Steenland (1999)</td>
<td>5</td>
<td>Work</td>
<td></td>
<td></td>
<td></td>
<td>1.21 (95%CI = 1.04-1.41)</td>
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</tbody>
</table>

**Key to Table A2.2a**

RR = Relative Risk  
A = never smokers ever exposed to ETS by spouses who smoke
NA = Not available
N = Number of studies reviewed
CI = Confidence Interval
B = never smokers ever exposed to ETS by spouses who smoke (US only)
C = never smokers exposed to ETS by spouses who smoke more than 20 cigarettes/day
D = never smokers exposed to ETS by spouses who have smoked for more than 20 years
E = ever exposure to ETS in the workplace (both sexes)

Note: Both sexes are combined in the relative risk estimates. All authors of meta-analyses have presented these figures and this allows for comparisons to be made. Where separate risks have been estimated, there are very small differences between sexes.
<table>
<thead>
<tr>
<th>Author (s)</th>
<th>Study</th>
<th>Inclusion criteria</th>
<th>No. of CHD cases (amongst never smokers)</th>
<th>RR (A)</th>
<th>RR (B)</th>
<th>RR (C)</th>
<th>RR (D)</th>
<th>RR (E)</th>
<th>Adjustments (or matching)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rosenlund et al. 2001</td>
<td>CC</td>
<td>Patients at all emergency hospitals in Stockholm, Sweden or deaths registered at Statistics Sweden. First event of MI</td>
<td>334</td>
<td>1.14</td>
<td>1.58</td>
<td>1.25</td>
<td></td>
<td>1.07</td>
<td>Age, hospital/catchment area. BMI, socio-economic status, job strain, hypertension, diet and diabetes mellitus</td>
</tr>
<tr>
<td>Pitsavos et al. 2002</td>
<td>CC</td>
<td>Patients at participating Greek hospitals, first event of acute MI or unstable angina</td>
<td>297</td>
<td>1.33 (95%CI = 1.21 – 2.99)</td>
<td></td>
<td></td>
<td></td>
<td>1.97 (95%CI = 1.41 – 3.87)</td>
<td>with regular exposure</td>
</tr>
<tr>
<td>Enstrom and Kabat (2003)</td>
<td>CH</td>
<td>Participants in American Cancer Society prevention study (CPS 1) - Californian cohort only</td>
<td>1,533 females</td>
<td>1.01 (95%CI = 0.93 – 1.09)</td>
<td>0.95 (95%CI = 0.80 – 1.12)</td>
<td></td>
<td></td>
<td>Age *</td>
<td>Age, race, education level, exercise, BMI, urbanisation, fruit/fruit juice intake, health status</td>
</tr>
</tbody>
</table>

**Table A2.2b - Exposure to ETS and Coronary Heart Disease Epidemiological studies published 2000 – 2004**
Key to Table A2.2b

RR = Relative Risk  A = never smokers ever exposed to ETS by spouses who smoke
NA = Not available  B = never smokers ever exposed to ETS by spouses who smoke (US only)
N = Number of studies reviewed  C = never smokers exposed to ETS by spouses who smoke more than 20 cigarettes/day
CI = Confidence Interval  D = never smokers exposed to ETS by spouses who have smoked for more than 20 years
      E = ever exposure to ETS in the workplace (both sexes)
### A2.3: STROKE – SUMMARY OF PRIMARY STUDIES

#### Study 1

<table>
<thead>
<tr>
<th>Author (s)</th>
<th>Lee et al.</th>
</tr>
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<tbody>
<tr>
<td>Year of Publication</td>
<td>1986</td>
</tr>
<tr>
<td>End-point</td>
<td>Stroke</td>
</tr>
<tr>
<td>Location</td>
<td>England</td>
</tr>
<tr>
<td>Participants</td>
<td>N = 55, lifelong non-smokers ever married to spouse who smokes/smoked in whole of marriage</td>
</tr>
<tr>
<td>Controls</td>
<td>N = 269, hospital patients with other diagnosis, matched on sex, age, hospital region</td>
</tr>
<tr>
<td>Results (95%CI)</td>
<td><strong>0.90</strong> (0.53 – 1.52)</td>
</tr>
<tr>
<td>Adjustments</td>
<td>Not reported</td>
</tr>
<tr>
<td>Comments</td>
<td>No surrogate responses? (all non fatal cases). Spousal smoking habits confirmed by spouse. Response rates not given.</td>
</tr>
</tbody>
</table>

#### Study 2

<table>
<thead>
<tr>
<th>Author (s)</th>
<th>Donnan et al.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Year of Publication</td>
<td>1989</td>
</tr>
<tr>
<td>End-point</td>
<td>First episode of fatal or non-fatal cerebral ischaemia</td>
</tr>
<tr>
<td>Location</td>
<td>Melbourne, Australia</td>
</tr>
<tr>
<td>Participants</td>
<td>N = 142, never smokers</td>
</tr>
<tr>
<td>Controls</td>
<td>N = 207, matched on age, sex and residential street</td>
</tr>
<tr>
<td>Results (95%CI)</td>
<td><strong>1.6</strong> (0.6 – 3.9) = risk associated with smoking spouse</td>
</tr>
<tr>
<td>Adjustments</td>
<td>Hypertension, age</td>
</tr>
<tr>
<td>Comments</td>
<td>99% participation rate amongst cases</td>
</tr>
</tbody>
</table>

#### Study 3

<table>
<thead>
<tr>
<th>Author (s)</th>
<th>You et al.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Year of Publication</td>
<td>1999</td>
</tr>
<tr>
<td>End-point</td>
<td>First episode of fatal or non-fatal ischemic stroke</td>
</tr>
<tr>
<td>Location</td>
<td>Victoria, Australia</td>
</tr>
<tr>
<td>Participants</td>
<td>N = 154, never smokers</td>
</tr>
<tr>
<td>Controls</td>
<td>N = 213, matched on age, sex and residential street</td>
</tr>
</tbody>
</table>
Results (95%CI) | 1.70 (0.98 – 2.92) = risk associated with smoking spouse  
| 1.55 (0.83 – 2.35) = risk associated with spouse smoking 1-20 cigarettes/day  
| 1.91 (0.94 – 3.79) = risk associated with spouse smoking 20+ cigarettes/day

Adjustments | Age, sex, hypertension, heart disease, diabetes and education level

Comments | Cases from Donnan, et al are included in analysis,

<table>
<thead>
<tr>
<th>Study 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author (s)</td>
</tr>
<tr>
<td>Year of Publication</td>
</tr>
<tr>
<td>End-point</td>
</tr>
<tr>
<td>Location</td>
</tr>
<tr>
<td>Participants</td>
</tr>
<tr>
<td>Controls</td>
</tr>
<tr>
<td>Results (95%CI)</td>
</tr>
<tr>
<td>Adjustments</td>
</tr>
<tr>
<td>Comments</td>
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</tbody>
</table>

<table>
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<tr>
<th>Study 5</th>
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<tr>
<td>Year of Publication</td>
</tr>
<tr>
<td>End-point</td>
</tr>
<tr>
<td>Location</td>
</tr>
<tr>
<td>Participants</td>
</tr>
<tr>
<td>Length of follow-up</td>
</tr>
<tr>
<td>Results (95%CI)</td>
</tr>
<tr>
<td>Adjustments</td>
</tr>
<tr>
<td>Comments</td>
</tr>
</tbody>
</table>

<table>
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<th>Study 6</th>
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<tr>
<td>Author (s)</td>
</tr>
<tr>
<td>Year of Publication</td>
</tr>
<tr>
<td>End-point</td>
</tr>
<tr>
<td>-----------------------------------</td>
</tr>
<tr>
<td>Location</td>
</tr>
<tr>
<td>Participants</td>
</tr>
<tr>
<td>Length of follow-up</td>
</tr>
<tr>
<td>Results (95%CI)</td>
</tr>
<tr>
<td>Adjustments</td>
</tr>
<tr>
<td>Comments</td>
</tr>
</tbody>
</table>

**Study 7**

<table>
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<tr>
<th>Author(s)</th>
<th>Whincup et al.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Year of Publication</td>
<td>2004</td>
</tr>
<tr>
<td>End-point</td>
<td>Non-fatal stroke and deaths from cerebrovascular disease</td>
</tr>
<tr>
<td>Location</td>
<td>United Kingdom</td>
</tr>
<tr>
<td>Participants</td>
<td>Never smoking men</td>
</tr>
<tr>
<td>Length of follow-up</td>
<td>20 years</td>
</tr>
<tr>
<td>Results (95%CI)</td>
<td>Serum cotinine concentrations:</td>
</tr>
<tr>
<td></td>
<td>0.8 – 1.4 ng/ml = 1.34 (95%CI = 0.53 – 3.40)</td>
</tr>
<tr>
<td></td>
<td>1.5 – 2.7 ng/ml = 1.39 (95%CI = 0.48 – 4.04)</td>
</tr>
<tr>
<td></td>
<td>2.8 – 14.0 ng/ml = 2.16 (95%CI = 0.80 – 5.80)</td>
</tr>
<tr>
<td>Adjustments</td>
<td>age, blood pressure, total cholesterol, FEV1, height, pre-existing CHD, BMI, triglyserides, white cell count, diabetes, physical activity, alcohol intake and social class</td>
</tr>
<tr>
<td>Comments</td>
<td></td>
</tr>
</tbody>
</table>
### A2.4: RESPIRATORY DISEASES

**Key to abbreviations:**

- **AOD** = Airway obstructive disease
- **BMI** = Body Mass Index
- **Ca** = Cases
- **CC** = Case control study
- **CH** = Cohort study
- **CI** = Confidence Interval
- **Co** = Cohort
- **CS** = Cross Sectional study
- **ETS** = Environmental Tobacco Smoke
- **FEF** = Maximal Midexpiratory Flow Rate
- **FEV** = Forced Expiratory Volume
- **FVC** = Forced Vital Capacity
- **OR** = Odds Ratio
- **N** = Number of participants
- **RR** = Relative Risk
- **SES** = Socio-economic status

### Table A2.4a - Mixed respiratory cases

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Country</th>
<th>Type of study</th>
<th>N</th>
<th>ETS exposure</th>
<th>Results (95%CI)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kalandidi et al. 1987</td>
<td>Greece</td>
<td>CC</td>
<td>103</td>
<td>Home</td>
<td>1.9 (1.0 – 4.0)</td>
<td>Obstructive lung disease</td>
</tr>
<tr>
<td>Dayal et al. 1994</td>
<td>Philadelphia, United States</td>
<td>CC</td>
<td>Ca = 219&lt;br&gt;Co = 657&lt;br&gt;Matching = age, gender and neighbourhood</td>
<td>Home</td>
<td>1.16 (0.78 – 1.7) light exposure*&lt;br&gt;1.86 (1.2 – 2.9) heavy exposure**</td>
<td>Obstructive airway disease (including asthma, chronic bronchitis and emphysema).&lt;br&gt;**less than one pack a day&lt;br&gt;**more than one pack a day&lt;br&gt;Controlled for type of heating and presence of gas stove</td>
</tr>
<tr>
<td>Mannino et al. 1997</td>
<td>United States</td>
<td>CS</td>
<td>43,732</td>
<td>Home and/or work</td>
<td>1.44* (1.07 – 1.95)</td>
<td>Chronic respiratory disease exacerbation = limiting activity or visiting physician due to asthma, chronic bronchitis, emphysema or chronic sinusitis&lt;br&gt;*controlled for age, SES, gender, race,</td>
</tr>
</tbody>
</table>
Asthma is a condition which is usually diagnosed in childhood. The studies presented here have primarily looked at self-reported adult onset asthma which has been doctor diagnosed. Due to confusion over the clinical definition of asthma and potential bias from participants who are unaware of diagnosis in childhood, some authors have chosen to look at passive smoking and other symptoms which are ‘asthma like’. These include wheezing, coughing, chronic cough and dyspnea.

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Country</th>
<th>Type of study</th>
<th>N</th>
<th>ETS exposure</th>
<th>Results (95%CI)</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Greer et al. 1993 | California | CH | 3914 | Work | 1.5 (1.12 – 2.01) men  
1.5 (1.17 – 1.92) women  
1.45* (1.2 – 1.8) per 10 year increment | After 10 years follow up  
*Controlled for age, education, gender, childhood history of AOD, ambient ozone concentration |
| Ng et al. 1993    | Singapore | CS women only | 1,438 | Home (live with heavy* smoker) | 1.6 (0.69 – 3.70) women | *More than 20 cigarettes/day                  |
| Robbins et al. 1993 | California | CH | 3917 (as Greer) | Home and Work | 1.57 (0.81 – 2.97) | May have included ex-smokers (15%)            |
| Leuenberger et al. 1994 | Switzerland | CS | 4197 | Home and/or Work | 1.39 (1.04 – 1.86) | Dose-response relationship with intensity and duration.  
Adjusted for age, BMI, parental and sibling history of asthma, atopy, passive smoking exposure in childhood, occupation, gender and city |
| McDonnell et al. 1999 | California | CH | 3091 | Work | 1.21 (1.04 – 1.39) for seven year increments - women | After 15 years follow up |
| Jaakkola et al. 2003 | Finland | CC | 521 | Home and Work | 1.66* (0.99 – 2.78)  
1.71** (1.11 – 2.64) | *ETS exposure in previous 12 months  
**ETS exposure of 150 cigarette years or more  
Adjusted for gender, age, parental |
Table A2.4c - Exacerbation of Pre-Existing Adult Asthma

<table>
<thead>
<tr>
<th>Author (s)</th>
<th>Country</th>
<th>Type of study</th>
<th>N</th>
<th>ETS exposure</th>
<th>Results (95%CI)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jindal et al. 1994</td>
<td>India</td>
<td></td>
<td>200</td>
<td>Exposed to ETS one hour/day or seven hours/week for one year min.</td>
<td>Significantly lower FEV₁, FVC and FEF₂₅₋₇₅ compared to non exposed asthmatics</td>
<td></td>
</tr>
<tr>
<td>Ostro et al. 1994</td>
<td>Denver</td>
<td>CH</td>
<td>164</td>
<td>Home</td>
<td><strong>2.08</strong> (1.63 – 2.64) Restricted activity</td>
<td>Participants with asthma</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><strong>1.21</strong> (1.01 – 1.46) for cough</td>
<td><strong>1.85</strong> (1.57 – 2.18) for dyspnea</td>
<td>Follow-up = 3 months with daily diaries</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><strong>1.24</strong> (1.00 – 1.53) for nocturnal respiratory symptoms</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Eisner (2002)

| Risks may not be based entirely on never smokers |

Table A2.4c - Chronic Bronchitis

<table>
<thead>
<tr>
<th>Author (s)</th>
<th>Country</th>
<th>Type of study</th>
<th>N</th>
<th>ETS exposure</th>
<th>Results (95%CI)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leuenberger, et al. 1994</td>
<td>Switzerland</td>
<td>CS</td>
<td>4197</td>
<td>Home and/or work</td>
<td>1.65 (1.28 – 2.16) 1.66* (1.18 – 2.33) work only</td>
<td>Adjusted for age, BMI, parental and sibling history of asthma, atopy, passive smoking exposure in childhood, occupation, gender and city</td>
</tr>
<tr>
<td>Author(s)</td>
<td>Country</td>
<td>Type of study</td>
<td>N</td>
<td>ETS exposure</td>
<td>Results</td>
<td>Comments</td>
</tr>
<tr>
<td>------------------------</td>
<td>----------</td>
<td>---------------</td>
<td>--------</td>
<td>--------------</td>
<td>------------------------------------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>White and Froeb (1980)</td>
<td>California</td>
<td>CH</td>
<td>2100</td>
<td>Workplace</td>
<td>Exposure associated with reduced FEF&lt;sub&gt;25-75&lt;/sub&gt; and FEF&lt;sub&gt;75-85&lt;/sub&gt;</td>
<td></td>
</tr>
<tr>
<td>Comstock et al. 1981</td>
<td>United States</td>
<td>CS</td>
<td>418</td>
<td>Home</td>
<td>Exposure associated with reduced FEV&lt;sub&gt;1&lt;/sub&gt;</td>
<td></td>
</tr>
<tr>
<td>Kauffmann et al. 1983</td>
<td>France</td>
<td>CS</td>
<td>3855 (women only)</td>
<td>Home</td>
<td>0.55% deficit in FEV&lt;sub&gt;1&lt;/sub&gt;</td>
<td>Women over 40 years old</td>
</tr>
<tr>
<td>Breunkeef et al. 1985</td>
<td>Holland</td>
<td>CS</td>
<td>97 (women only)</td>
<td>Home</td>
<td>Exposure throughout study = significantly lower FEF&lt;sub&gt;75&lt;/sub&gt;</td>
<td></td>
</tr>
<tr>
<td>Svendsen et al. 1987</td>
<td>United States</td>
<td>CS</td>
<td>1245 (men only)</td>
<td>Home</td>
<td>-2.8% deficit in FEV&lt;sub&gt;1&lt;/sub&gt; - men</td>
<td></td>
</tr>
<tr>
<td>Masi et al. 1988</td>
<td>Canada</td>
<td>CS</td>
<td>293</td>
<td>Cumulative exposure</td>
<td>Exposure associated with reduced FEV&lt;sub&gt;1&lt;/sub&gt;, FEF&lt;sub&gt;25-75&lt;/sub&gt; and FEF&lt;sub&gt;50&lt;/sub&gt; amongst males only and at home only.</td>
<td></td>
</tr>
<tr>
<td>Hole et al. 1989</td>
<td>Scotland</td>
<td>CS</td>
<td>671</td>
<td>Home</td>
<td>FEV&lt;sub&gt;1&lt;/sub&gt; significantly lower</td>
<td>Adjusted for age, height and sex</td>
</tr>
<tr>
<td>Kauffmann et al. 1989</td>
<td>United States</td>
<td>CS</td>
<td>1211</td>
<td>Home</td>
<td>Exposure associated with reduced FEV&lt;sub&gt;1&lt;/sub&gt; - women</td>
<td>Adjusted for age, city of origin, educational levels and occupational exposure</td>
</tr>
<tr>
<td>Masjedi et al. 1990</td>
<td>Iran</td>
<td>CS</td>
<td>288</td>
<td>Home or Work</td>
<td>-2% deficit in FEV&lt;sub&gt;1&lt;/sub&gt; significantly lower values for FVC and FEF&lt;sub&gt;25-75&lt;/sub&gt; but only amongst men and only at work</td>
<td></td>
</tr>
<tr>
<td>Ng et al. 1993</td>
<td>Singapore</td>
<td>CS</td>
<td>1008 women</td>
<td>Home</td>
<td>-3.8% deficit in FEV&lt;sub&gt;1&lt;/sub&gt;</td>
<td>Adjusted for age, race, height, area, size of residence and employment status</td>
</tr>
<tr>
<td>Study</td>
<td>Country</td>
<td>Exposure</td>
<td>N</td>
<td>Setting</td>
<td>FEV&lt;sub&gt;1&lt;/sub&gt; and FEC = Decreases associated with exposure. Statistically significant in men (not women)</td>
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</tr>
<tr>
<td>----------------------------</td>
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<tr>
<td>Xu and Li (1995)</td>
<td>China</td>
<td>CH</td>
<td>502</td>
<td>Home, Work</td>
<td>Decreases associated with exposure. Statistically significant in men (not women)</td>
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<tr>
<td>Frette et al. 1996</td>
<td>United States</td>
<td>CS</td>
<td>651</td>
<td>Home</td>
<td>-2.5% deficit in FEV&lt;sub&gt;1&lt;/sub&gt;</td>
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<tr>
<td>Jaakkola et al. 1995</td>
<td>Canada</td>
<td>CH</td>
<td>117</td>
<td></td>
<td>No statistically significant relationship with FEV&lt;sub&gt;1&lt;/sub&gt; or FEF 25–75</td>
<td></td>
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<tr>
<td>Carey and Cook (1999)</td>
<td>United Kingdom</td>
<td>CH</td>
<td>1623</td>
<td>Home</td>
<td>Very small changes in FEV&lt;sub&gt;1&lt;/sub&gt; after 7 years follow up. BUT Higher cotinine levels were associated with larger differences in FEV&lt;sub&gt;1&lt;/sub&gt; over follow up.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2.5 – 14.7 ng/ml = mean adjusted difference = -76 ml</td>
<td></td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td>0.4 – 0.6 ng/ml = mean adjusted difference = -4 ml</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Adjusted for sex, age and height</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>A meta-analysis using 21 studies looked at the association between lung function and passive smoking and estimated a pooled random effects deficit in FEV&lt;sub&gt;1&lt;/sub&gt; of -1.7% (-2.8% - 0.6%)</td>
<td></td>
</tr>
<tr>
<td>Author (s)</td>
<td>Country</td>
<td>Type of study</td>
<td>N</td>
<td>ETS exposure</td>
<td>Results (95%CI)</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------</td>
<td>-----------------------------</td>
<td>---------------</td>
<td>-----</td>
<td>--------------</td>
<td>-----------------</td>
<td>---------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Comstock et al.</td>
<td>Maryland</td>
<td>CS</td>
<td>1,802</td>
<td>Home</td>
<td>1.04 men 1.45 women</td>
<td></td>
</tr>
<tr>
<td>Kauffmann et al.</td>
<td>United States and France</td>
<td>CS women only</td>
<td>6,075</td>
<td>Home</td>
<td>US = 1.35 (0.97 – 1.87) women</td>
<td></td>
</tr>
<tr>
<td>Ng et al. 1993</td>
<td>Singapore</td>
<td>CS women only</td>
<td>1,438</td>
<td>Home (live with heavy* smoker)</td>
<td>2.69 (1.23 – 5.88) women</td>
<td>*More than 20 cigarettes/day Adjusted for age, race, area, size of residence, chronic rhinitis, eczema and employment status</td>
</tr>
<tr>
<td>Leuenberger et al. 1994</td>
<td>Switzerland</td>
<td>CS</td>
<td>4197</td>
<td>Home and/or work</td>
<td>1.94 (1.39 – 1.86) 2.05*(1.46 – 2.92) work only</td>
<td>*Adjusted for age, BMI, parental and sibling history of asthma, atopy, passive smoking exposure in childhood, occupation, gender and city</td>
</tr>
</tbody>
</table>
## Table A2.4f - Sputum Production

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Country</th>
<th>Type of study</th>
<th>N</th>
<th>ETS exposure</th>
<th>Results (95%CI)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hole et al. 1989</td>
<td>Scotland</td>
<td>CS</td>
<td>671</td>
<td>Home</td>
<td><strong>1.19</strong> (0.85 – 1.67)</td>
<td></td>
</tr>
<tr>
<td>Kauffmann et al. 1989</td>
<td>United States and France</td>
<td>CS women only</td>
<td>6,075</td>
<td>Home</td>
<td>US = <strong>1.65</strong> (0.72 – 3.78) women</td>
<td></td>
</tr>
<tr>
<td>Ng et al. 1993</td>
<td>Singapore</td>
<td>CS women only</td>
<td>1,438</td>
<td>Home (live with heavy* smoker)</td>
<td><strong>2.29</strong> (0.94 – 5.59) women</td>
<td>*more than 20 cigarettes/day</td>
</tr>
</tbody>
</table>

## Table A2.4g - Dyspnea

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Country</th>
<th>Type of study</th>
<th>N</th>
<th>ETS exposure</th>
<th>Results (95%CI)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comstock et al. 1981</td>
<td>Maryland</td>
<td>CS</td>
<td>1,802</td>
<td>Home</td>
<td><strong>1.08</strong> men 1.79 women</td>
<td></td>
</tr>
<tr>
<td>Hole et al. 1989</td>
<td>Scotland</td>
<td>CS</td>
<td>7,997</td>
<td>Home</td>
<td><strong>1.09</strong> (0.82 – 1.45)</td>
<td></td>
</tr>
<tr>
<td>Kauffmann et al. 1989</td>
<td>United States and France</td>
<td>CS women only</td>
<td>6,075</td>
<td>Home</td>
<td>US = <strong>1.35</strong> (0.68 – 2.61) women</td>
<td></td>
</tr>
<tr>
<td>Ng et al. 1993</td>
<td>Singapore</td>
<td>CS women only</td>
<td>1,438</td>
<td>Home (live with heavy* smoker)</td>
<td><strong>1.83</strong> (1.30 – 2.58) women</td>
<td>*more than 20 cigarettes/day</td>
</tr>
<tr>
<td>Leuenberger et al. 1994</td>
<td>Switzerland</td>
<td>CS</td>
<td>4197</td>
<td>Work</td>
<td><strong>1.45</strong>* (1.20 – 1.76) for dyspnea on exertion</td>
<td>*Adjusted for age, BMI, parental and sibling history of asthma, atopy, passive smoking exposure in childhood, occupation, gender and city</td>
</tr>
</tbody>
</table>

## Table A2.4 h - Chronic Cough

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Country</th>
<th>Type of study</th>
<th>N</th>
<th>ETS exposure</th>
<th>Results (95%CI)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comstock et al. 1981</td>
<td>Maryland</td>
<td>CS</td>
<td>1,802</td>
<td>Home</td>
<td><strong>0.96</strong> men 0.17 women</td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Country</td>
<td>Group</td>
<td>N</td>
<td>Setting</td>
<td>OR (CI) for cough 3 months/year</td>
<td></td>
</tr>
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</tr>
<tr>
<td>Kauffmann et al. 1989</td>
<td>United States and France</td>
<td>CS women only</td>
<td>6,075</td>
<td>Home</td>
<td><strong>US = 1.14 (0.62 – 2.09) women</strong></td>
<td></td>
</tr>
<tr>
<td>Ng et al. 1993</td>
<td>Singapore</td>
<td>CS women only</td>
<td>1,438</td>
<td>Home (live with heavy* smoker)</td>
<td><strong>3.01 (1.13 – 8.03) for cough 3 months/year</strong></td>
<td></td>
</tr>
</tbody>
</table>

*more than 20 cigarettes/day Adjusted for age, race, area, size of residence, and employment status