

RESPONDEE INFORMATION FORM

Please complete the details below and attach it with your response. This will help ensure we handle your response appropriately:

1

Name: The Tobacco Manufacturers' Association

Address: Burwood House, 5th Floor, 14/16 Caxton Street, London, SW1H 0ZB

Consultation Title: Smoking in Public Places

2a

IF YOU ARE RESPONDING AS AN INDIVIDUAL:

Do you agree to your response being made available to the public (in SE library and/or on SE website)?

Yes (go to 2b below)

No, not at all

2b

Where confidentiality is not requested, we will make your response available to the public on the following basis. (**please tick one** of the following boxes)

Yes, make my response, name and address all available

Yes, make my response available, but not my name or address

Yes, make my response and name available, but not my address

2c

IF YOU ARE RESPONDING ON BEHALF OF A GROUP OR ORGANISATION:

Your name and address as respondents *will* be made available to the public (in the SE library and/or on SE website). Are you content for your response to be made available also?

Yes

No

THANK YOU FOR COMPLETING THIS CONSULTATION DOCUMENT

Smoking in Public Places
RESPONSE FORM

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- 1 **Having considered the health risks associated with passive smoking, do you think that further action needs to be taken to reduce people's exposure to second-hand smoke?** (Please tick one box only)

Yes

No

Don't Know

The body of scientific and epidemiological evidence on ETS does not prove that ETS causes disease in non-smokers and does not provide justification for a public policy prohibiting smoking in work and other places. In those few epidemiological studies that have reported an association that satisfies conventional statistical tests, the level of relative risk is extremely low; could easily be accounted for by inadequate adjustments for errors and flaws arising from study design, methodology, bias or confounding; and, in any event, does not equate to any meaningful increase in risk for any individual person.

Voluntary self-regulation of smoking in work and other places is providing what employers, employees and the public want. Increasingly more provision is being made for non-smoking facilities in the hospitality sector.

- 2 **Would you support a law that would make enclosed public places smoke-free? (Public places include workplaces and public transport).** (Please tick one box only)

Yes

No

Don't Know

Nothing has changed since the Health and Safety Commission and Executive confirmed in 1999/2000 that there is already sufficient health, safety and welfare law that can be applied to the question of passive smoking in the work place. Given the uncertainty surrounding the scientific and epidemiological evidence on ETS, they considered that smoking in public places was a matter better dealt with in ways other than by legislation.

Legislation would be socially divisive and needlessly replace successful, voluntarily adopted policies with oppressive and costly bureaucracy.

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- 3 **If a law was introduced, do you think there should be exceptions to it?** (i.e. any enclosed public places where smoking should be allowed). (Please tick one box only)

Yes

No

Don't Know

If, nonetheless, there were to be legislation prohibiting smoking, that legislation should make provision for places where smoking is permitted. Those provisions should not impose unwarranted and unjustified further costs on employers and businesses, many of which already have formal smoking policies that have the full support of their employees and, where appropriate, their customers and visitors.

- 4 **If we decide not to introduce a law, what more could be done to encourage individual businesses to take voluntary action to become smoke-free or to provide more smoke-free provision?**

The Scottish Executive, Scottish local authorities and NHS Scotland are in a position both to set an example of best practice and to play a key role in encouraging businesses to adopt appropriate smoking policies.

In the hospitality sector it is clear that there is significant scope for the further development of the Public Places Charter in Scotland, the targets of which, set in 1998, were met and exceeded.

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- 5 **What else could we do to reduce people's exposure to second-hand smoke?**

For the purposes of public policy, there needs to be clarity as to precisely why there is perceived to be a need to go further than is stated in answer to the preceding questions to reduce people's exposure to environmental tobacco smoke. That is currently far from being the case.

Smoking is not illegal and there are no reasons to make smoking, or the permitting of smoking, criminal offences., However, smokers do not have a right to smoke anywhere they please, and the vast majority of smokers are respectful of other people's preferences and views, and comply with smoking rules. There is no need for legislation, or the introduction of any other form of formal regulation, when voluntary self-regulation, tolerance and common sense can provide the solutions.

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- 6 **Please let us know about any other views you have about smoking in public places.**

We set out our detailed evidence on the questions and issues raised by the Executive in the following document.

Smoking in Public Places

The response of the
Tobacco Manufacturers' Association
to the Scottish Executive's consultation paper

September 2004

Introduction

The public has repeatedly been given the impression that there is conclusive proof that environmental tobacco smoke (ETS, or 'second-hand smoke', 'passive smoking', or 'involuntary smoking', as it is sometimes called) causes serious disease in non-smokers, and that the risk is great. The more there has been repetition of that claim, the more the public has come to believe it to be incontrovertible fact. This belief also appears to serve as the starting point of the Executive's consultation on reducing exposure to ETS: the first question posed in the consultation asks about further action on reducing exposure to ETS, "*having considered the health risks*"; the supporting introductory document, *Some Key Facts*, states that "*The health risks of exposure ... are clear*", drawing on conclusions principally of the UK Scientific Committee on Tobacco and Health.

Not surprisingly given this presupposition, it appears that the consequent public debate that has taken place in Scotland has failed to examine the evidence on ETS and health with an inquisitive eye, and has been led more by misleading propaganda about what the public wants, and an agenda of coercing people into quitting smoking.

We believe that it is crucial that there should first be an objective analysis and assessment of the published evidence on ETS and the risks to the health of non-smokers that have been claimed. Only then is it possible to weigh this evidence with other considerations – including political aspects – to determine the appropriate public policy response. This we undertake first, in appropriate detail, under the heading **The evidence on ETS** (page 4).

We then consider the smoking policies that currently apply in work and other places and the alternative public policy options that have been suggested, including legislation that could be applied across the UK, in Scotland only, or be implemented at the discretion of individual local authorities – **Smoking in work and other places** (page 33).

We consider and compare the numerous polls and public attitude surveys that have been undertaken over the past two years and the extent to which they can be regarded as truly representing the opinion of the public as a whole – **What the public thinks and wants** (page 42). A more detailed analysis of public attitude surveys undertaken between March 2003 and July 2004 is provided in a separate document.

These three main chapters are preceded by a brief **Statement of general conclusions**.

The Tobacco Manufacturers' Association (TMA) is the body that represents the interests of UK tobacco manufacturers and this response is made on behalf of its principal members – British American Tobacco, Gallaher and Imperial Tobacco.

Statement of general conclusions

The most striking and regrettable feature of the public debate about smoking in work and public places is the misinformed basis on which it has been taking place.

The public has been given to believe that the evidence is clear and incontrovertible: that environmental tobacco smoke is the cause of serious diseases in non-smokers and that prohibition of smoking in work and public places is what the public wants.

However, the facts speak otherwise.

Scientific studies on the nature of ETS and its components, and the effect on non-smokers of ETS exposure, do not prove that ETS causes serious diseases.

ETS epidemiological studies and reviews also fail to support the hypothesis. The findings of individual studies are inconsistent and inconclusive. Even when a positive effect has been found, the relative risk has been so low that it is beyond the limits of reliable epidemiological inference; in any event, the estimated excess risk could be accounted for by the failure to make any, or any adequate adjustments for study design and data flaws, bias and confounding. Few of the studies have produced results that fulfil the conventional statistical tests for significance.

Reviews using meta-analysis, a technique that seeks to make a quantitative synthesis of the results of separate ETS studies and provides a summary of the pooled results, are likewise unreliable. A statistically significant result from a meta-analysis of epidemiological data, does not necessarily establish that any underlying effect exists. The interpretation of systematic reviews is as prone to errors as is the interpretation of data in individual studies. In both cases, interpretations are subjective, not objective judgments. The acceptance by an authority of a particular meta-analysis does not make that meta-analysis any more true. The superficial appearance of the infallibility of meta-analyses should be firmly rejected.

It is important that statistics that are used to inform decision-making are based on rigorous data and assumptions, and sound methodology. However, many widely varying figures have been bandied around regarding the number of deaths claimed to be 'caused by ETS' that 'would be saved' by prohibiting smoking in work and public places, and the NHS treatment costs that would as a result be avoided. Such calculations are mere arithmetical exercises that have no veracity, founded as they are on disregard for fundamental principles of epidemiological and statistical methods.

Likewise, the results of opinion polls and surveys have been misrepresented. Surveys that use sampling techniques that seek to reflect the population as a whole and which offer a choice of response options generally produce reliable results. Those that allow the sample of the population to choose itself do not. People who volunteer to participate in surveys or who respond to general invitations are more passionate in their opinions than most, and what is true of them is most unlikely to be true of the wider population. That flaw is greatly compounded when they are simply asked for a yes or no response to a loaded question.

This has all been borne out by opinion surveys on ETS. Those employing nationally representative survey and standard sampling techniques have produced quite different results from those based on self-selected samples. The former have shown that people do not consider legislation prohibiting smoking to be appropriate or necessary. Rather, people have expressed a strong preference for a free choice of smoking and non-smoking facilities and, reflecting the prevalence of smoking in the population generally, greater provision of non-smoking facilities. In that regard, they believe that more progress is required and that it should be the responsibility of business to satisfy the demand, not for government to impose prohibition or regulations.

88% of employees work in places where smoking is already either banned or permitted only in designated areas. The widespread adoption of self-regulation of smoking in the workplace has been a great success, not least because it has operated through a process of consultation with and involvement of employees and their representatives in both the private and public sectors. More can be achieved by following this same direction.

In the hospitality sector there have been significant advances in providing more non-smoking facilities and improved air quality and ventilation. Many thousands of owners and operators are committed to achieving further improvements to meet public demand.

There is no need or justification for legislation regulating smoking in work or other places. The authorities at a national and local level, working with other public sector bodies and the private sector, can achieve much more by continuing the voluntary approach, without the creation of unwarranted criminal offences.

The evidence on ETS

The scientific evidence

The popular hypothesis

There is a popular hypothesis that lies at the heart of the claims that are made as to the risks of ETS to the health of the non-smoker. According to that hypothesis, ETS contains many of the substances and chemicals inhaled by the smoker which are carcinogens or genotoxins that have no threshold for harm; these are not simply present in ETS but are found, metabolised or otherwise, in non-smokers exposed to ETS; and therefore a non-smoker's exposure to ETS is equivalent to a 'dose' of active smoking.

Ambient air and ETS

In all normal circumstances, indoor ambient air contains a large number of substances. Such substances can include dust, pollen, bacteria, fungi, trace chemicals from industrial plants, vehicle emissions, household furnishings and cleaning products and many other sources of pollutants, as well as, in certain circumstances, emissions from cooking and heating appliances. Research suggests that the types of substances found in indoor air are generally similar, irrespective of the presence of tobacco smoke¹.

ETS is a mixture of the smoke released from the burning end of a cigarette (termed "sidestream" smoke) and the smoke exhaled by the smoker between puffs². This smoke very quickly mixes into the ambient air and is thereby much diluted. Its constituents change over time and according to environmental conditions. Consequently there are significant and important differences between the level and chemical and physical composition of the "mainstream" smoke inhaled by the smoker and the ETS contribution to ambient air that may be inhaled by the non-smoker.

Measuring ETS and its constituents

Identifying and measuring the components of ETS, and assessing the exposure of non-smokers to them in real-life situations, presents very great difficulties. The various substances that make up ETS are generally only present in extremely low concentrations, some below any meaningful measurement. They are substances likely to be present in the air anyway, emanating from other sources and inseparable from the ETS contribution.

For all these reasons, compositional physical studies of ETS, both in laboratories and in real-life conditions, have been very limited:

*"Studies on the complex composition of secondhand tobacco smoke in 'real world' conditions have been limited partly because of the presence of additional sources of secondhand smoke constituents. Therefore compositional and physical studies of secondhand tobacco smoke have often been performed in environmental chambers (also known as a 'controlled experimental atmosphere'). The disadvantage of the controlled experimental atmosphere is that it does not reflect real life situations. The studies of the chemical composition of secondhand smoke, either in a controlled experimental atmosphere or in the field, have been limited. This is mainly because there are still no standardized criteria for the development of experimental atmospheres that represent secondhand tobacco smoke (Jenkins et al, 2000³)."*⁴

¹ Guerin M et al, The chemistry of environmental tobacco smoke: composition and monitoring. *Chelsea, Michigan, Lewis Publishers*, 1992

² Baker R and Proctor C, The origins and properties of environmental tobacco smoke. *Environmental International* 1990;16: 231-245

³ Jenkins RA, Guerin MR & Tomkins BA. Properties and measures of environmental tobacco smoke. In: *The Chemistry of ETS: Composition and Measurement*, 2nd ed. , Boca Raton, FL, Lewis Publishers, 2000

⁴ WHO, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol 83, Tobacco Smoke and Involuntary Smoking, p.1192, *WHO/IARC*, Lyon, France, 2004

The measurement of real-life exposure to ETS is likewise highly problematic and the recent publication of the results⁵ of a crude experiment, comparing ETS with the emissions from a Ford Mondeo diesel car in a private garage in a northern Italian mountain town, adds little, if anything, to the available body of knowledge which shows “considerable variety in the measured levels of constituents of secondhand tobacco smoke”⁶.

Indicators of ETS exposure

Given the very nature of ETS and the infinite variety of real-world situations in which it may be present, the means of assessing ETS exposure that are available can only provide indicators of exposure, not reliable quantitative measurements which can then be taken to apply either in other particular or all situations. The following table⁷ lists available indicators:

Measure	Indicator
Surrogate measures	Prevalence of smoking in men and women
Indirect measures	Report of secondhand tobacco smoke exposure in the home and in the workplace
	Smoking in the household
	Number of smokers
	Smoking by parent(s)
	Number of cigarettes smoked
	Smoking in the workplace
	Presence of secondhand smoke
	Number of smokers
Direct measures components	Concentration of secondhand tobacco smoke
	Nicotine
	Respirable particles
	Other markers
	Biomarker concentrations
	Cotinine
	Carboxyhaemoglobin

The **surrogate measure** of smoking prevalence in the population is a very inadequate measure, providing only a suggestive indicator of the likelihood of exposure to ETS. However, there are many countries where it is the only available indicator.

Indirect measures generally comprise pro-forma self-reported exposure to ETS and descriptions of the sources of smoking in microenvironments, most often in the home, sometimes in the workplace. However, “questionnaire-based reports of intensity of exposure are of uncertain validity”⁸. Also there is little consistency between studies as to the definition of exposure to ETS. For example, in its recent monograph, IARC stated that the WHO definition was “exposure for at least 15 minutes per day on more than one

⁵ Invernizzi G, Ruprecht A, Mazza R, Rossetti E, Sasco A, Nardini S & Boffi R. Particulate matter from tobacco versus diesel car exhaust: an educational perspective. *Tobacco Control* 2004; **13**:219-221

⁶ WHO/IARC Monographs on the evaluation of carcinogenic risks to Humans, Vol 83, Tobacco Smoke and Involuntary Smoking, p.1193, *WHO/IARC*, Lyon, France, 2004

⁷ Reproduced from WHO, IARC Monographs on the evaluation of carcinogenic risks to humans, Vol.83, Tobacco Smoke and Involuntary Smoking p.1205, WHO/IARC, originally Samet J & Yang G, Passive smoking, women and children, in: Samet J & Yoon S-Y eds., *Women and the Tobacco Epidemic: Challenges for the 21st century*, WHO, Geneva, 2001

⁸ IARC Monograph Vol. 83, 2004, p 1206

day per week.⁹ Few studies have adopted such a definition and ETS epidemiological studies generally rely on widely varying, indirect, questionnaire-based measures. These are commented upon more appropriately in the following section, *The epidemiological evidence*.

Direct indicators of exposure include the measurement of the concentration of ETS components in the air and of levels of ETS biomarkers in the body. However, “no compound has a consistent ratio with other components. Therefore, the choice of marker can affect the estimate of exposure. The selection of a particular secondhand smoke component for monitoring is largely based on technological feasibility.”¹⁰

The respirable particles in indoor air have sources other than active smoking and are non-specific indicators of tobacco smoke; nicotine is present in certain foods, such as potatoes, tomatoes, aubergines and tea, but airborne nicotine is specific to smoking. Hence, some studies have measured nicotine, not because it is widely thought to cause lung cancer or any other serious diseases, but because airborne it is unique to tobacco smoke and can be measured even at low concentrations.

For example, one study¹¹ reported that, on average, in the course of a year, non-smokers had an exposure to airborne nicotine that was less than the amount delivered to a smoker by just five cigarettes with a nicotine yield of 1mg per cigarette. Another study¹² of British women exposed to ETS in various settings reported that on average a non-smoker would only be exposed to the equivalent of the direct smoking of just a single cigarette over a period in excess of two years.

Cotinine is one of the many metabolites of nicotine and levels of cotinine can be measured in body fluids. Nicotine is present predominantly in the particulate phase of mainstream smoke and also in the vapour phase of ETS. Thus, generally one would expect levels of cotinine in smokers largely to reflect mainstream smoke, and levels in non-smokers to reflect either exposure to ETS or other sources of nicotine, or a combination of both.

Where cotinine levels differ by a very substantial margin between individuals, this may serve to differentiate between smokers and non-smokers and, in particular, individuals who claim to be non-smokers but who are probably smokers. Smaller differences in cotinine do not, however, provide a reliable quantitative measure of, or serve as a marker of, exposure to ETS: individuals vary in the relative extent to which nicotine is distributed amongst body tissues¹³; metabolism into cotinine varies from person to person, as does the rate of elimination of cotinine¹⁴, by virtue of physiological and genetic factors such as gender, age and race¹⁵; there may also be other, food sources of nicotine. A single positive cotinine determination therefore only indicates that recent exposure to nicotine has taken place (cotinine has a half life of around 20 hours), by an unknown route and unknown origins.

A variety of studies that have measured the biological metabolites of nicotine have suggested ETS exposures of well below 1% of active smoking. Among a sample of 10,000 US citizens, it was reported that typical serum cotinine levels in non-smokers exposed to ETS was the equivalent of no more than 0.2% of the nicotine taken up by a

⁹ Ibid, p.1206

¹⁰ Ibid, p.1206

¹¹ Jenkins R A *et al.* Determination of personal exposure of non-smokers to environmental tobacco smoke in the United States. *Lung Cancer* 1996;14.1: Supplement, p.195

¹² Proctor C *et al.* A comparison of methods of assessing exposure to environmental tobacco smoke in non-smoking British women. *Environmental International* 1991;17.4: 287-297

¹³ Cholerton S, McCracken NW & Idle JR. Sources of individual variability in nicotine pharmacokinetics, in *Nicotine and Related Alkaloids*, pp 219-245, Chapman & Hall, 1993

¹⁴ Benowitz NL & Jacob P. Metabolism of nicotine to cotinine studied by a dual stable isotope method, *Clinical Pharmacology and Therapeutics* 1994; 56, 483-493

¹⁵ Knight JM, Eliopoulos C, Klein J, Greenwald M & Kopren G. Passive smoking in children: Racial differences in systemic exposure to cotinine by hair and urine analysis. *Chest*, 1996; 109: 446-450.

typical smoker¹⁶, Results from a 1994 Health Survey indicated that household ETS exposure was only 0.4% of the level associated with active smoking.

A more recently published study¹⁷ examined levels of cotinine with the risk of coronary heart disease and stroke. This study took data from the British regional heart study, which is a prospective study of cardiovascular disease in men aged 40-59 years that began in 1978-80. In 1978-80, research nurses administered a questionnaire on present and previous smoking habits – but not asking about ETS exposure - and blood samples were taken and frozen. In 2001-02, those samples were thawed and cotinine concentrations were measured. The cotinine values for each person were then compared with heart disease events over the period 1980 to 2000.

The study found no increase in risk of stroke associated with ETS exposure as measured by cotinine, a finding which contradicts results from an earlier case-control study¹⁸. It found no increase in risks for coronary heart disease when measured after 15 to 20 years. For life-time non-smokers, the study reported increases in risk that were not 'statistically significant' (meaning that they could be the result of pure chance and not related to ETS) for all adjustments apart from one.

Contrary to the sensationalist headlines reporting the study in the popular press, the researchers stated that their study was "*modest in size with limited precision*". The researchers also expressed concern as to possible misclassification arising from men in the higher cotinine groups claiming to be non-smokers actually being smokers, if only on an intermittent basis. Such misclassification could account for the otherwise greatly puzzling finding, inconsistent with virtually all other research, of a relative risk for non-smokers exposed to ETS being almost the same as that for active smokers of 1 to 9 cigarettes a day.

Estimates of particulate exposure have produced very low estimates: the results of 24 hour personal monitoring studies in Europe and Asia estimated that living with a smoker is associated with a median increase in exposure to particles that is equivalent to smoking less than 10 cigarettes a year - of the order of 0.05 to 0.1% of that of active smoking.

Extrapolations from active smoking

The significant and important differences in the chemical and physical composition of mainstream and sidestream smoke mean that ETS cannot be regarded simply as a 'dose' of mainstream smoke¹⁹. Also, non-smokers do not inhale sidestream smoke directly, as smokers do of mainstream smoke, and only breathe in air after the sidestream smoke has been greatly diluted in ambient air and after reactive components have been eliminated during ageing.

The facts are as stated by IARC – no one ETS component has a consistent ratio with all other components and the choice of a marker of ETS therefore affects the estimate of exposure. Cotinine "*should not be used to extrapolate from active to passive smoking exposures*"²⁰, nor should any other component. The effects of exposure to ETS cannot be estimated from any individual constituents.

¹⁶ Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA & Maurer K. Exposure of the US population to ETS: the Third National Health and Nutrition Survey, 1988 to 1991, *Journal of the American Medical Association* 1996; 275: 1233-1240

¹⁷ Whincup, P H *et al*, Passive smoking and risk of coronary heart disease and stroke: prospective study with cotinine measurement. *BMJ Online First*, 30 June 2004

¹⁸ Bonita, R *et al*, Passive smoking as well as active smoking increases the risk of acute stroke. *Tobacco Control* 1999;8:156-160

¹⁹ Geurin M, Jenkins RA, Tomkins BA. The chemistry of environmental tobacco smoke: composition and measurement. *Chelsea, MI, Lewis Publishers, 1992*

²⁰ Bayard S, Jinot J, Flatman G: Environmental tobacco smoke and lung cancer: uncertainties in the population estimates but not in the causal association – A rejoinder to Gross. *Environmetrics* 1995; 6: 413-418

Carcinogens and genotoxins

Much is often made of the fact that “among the 4,000 and more chemicals in tobacco smoke there are around 60 known or suspected carcinogens”²¹. However, an analysis of air without the presence of ETS produces much the same result – thousands of chemical constituents, many of them known or suspected carcinogens²². In urban areas where in particular there is much road traffic, the concentrations of those constituents in the air are likely to be a great deal higher²³.

Many of the known or suspected carcinogens or genotoxins present in ETS are also associated, for example, with certain products, industrial processes and working environments. That is precisely why in occupational health and environmental regulations, the authorities lay down permissible limits. The concentrations of the relevant chemicals in ETS are typically very many times lower than those permissible exposure limits^{24 25}. Also it is now widely believed that there are thresholds for carcinogenesis and other disease processes²⁶. Where a genotoxic mechanism may be relevant, it is not possible to infer the absence of a threshold with any confidence.

Conclusions on the scientific evidence and where they lead

The conclusion to be drawn from all this is that the scientific studies into the nature of ETS and its components, and the exposure to and uptake of ETS components by non-smokers, do not provide any firm or reliable conclusions on the possible health effects of ETS. This is also clearly demonstrated by the body of IARC’s most recent report, yet IARC made the overall evaluation that “ETS is carcinogenic to humans (Group 1).”²⁷

A Group 1 classification is nominated by IARC when the evidence of carcinogenicity is sufficient or, exceptionally when the evidence for humans is less than sufficient but that for animals is sufficient and there is strong evidence in exposed humans that the agent itself acts through a relevant mechanism of carcinogenicity. Sufficient evidence of carcinogenicity in humans is satisfied under IARC’s classification regime where a positive relationship has been observed in which chance, bias and confounding (explained at pages 10 to 12) can be ruled out with reasonable certainty.

In an earlier monograph²⁸, IARC briefly considered the evidence relating cancer to ETS exposure and made it quite clear that the evidence available at that time was inadequate to incriminate ETS as a cause. Whilst the volume of evidence available now is greater, the findings overall do not differ in any significant respect from the evidence IARC considered in 1986. Yet IARC’s evaluation is different.

It is evident that IARC’s classification of ETS as a Group 1 carcinogen (announced in 2002, although the report was not published until 2004) depends crucially on IARC’s evaluation that there is sufficient evidence that ETS causes lung cancer in humans, since it clearly considers the evidence as regards other cancers in humans (and animals) to be inconclusive and insufficient. IARC does consider that there is ‘sufficient’

²¹ NHS Scotland and ASH Scotland. Reducing smoking and tobacco-related harm: a key to transforming Scotland’s health, p.11, *Health Scotland*, 2003

²² see footnote 19, and Hoskins JA. Health effects due to Indoor Air Pollution. *Indoor Built Environment* 2003; **12**:427-433

²³ *Scotland on Sunday* on 29th August 2004 reported a study undertaken by Calor Gas that measured levels of nitrogen oxides in 30 locations, of which Glasgow was one. The report was headed “Why a day in Glasgow is worse for you than smoking 40 cigarettes”.

²⁴ Redhead CS, Rowberg RE: Environmental Tobacco Smoke and Lung Cancer Risk. *US Congressional Research Service*, 1995

²⁵ Gori GB, Mantel N: Mainstream and environmental tobacco smoke. *Regulatory Toxicology & Pharmacology* 1991; **14**: 88-105

²⁶ Waddell WJ. Analysis of thresholds for carcinogenicity. *Toxicology Letters*, 2004; **149**:415-419; Thresholds in chemical carcinogenesis: what are animal experiments telling us?. *Toxicological Pathology*, 2003; **31.3** : 260-262; Thresholds of Carcinogenicity of Flavours.

Toxicological Science 2002; **68**: 275-279. Also Hengstler JG, Bogdabffy MS, Bolt HM & Oesch F. Challenging dogma: thresholds for genotoxic carcinogens? The case of vinyl acetate. *Annual Review Pharmacological Toxicology*, 2003; **43**: 485-520; Lovell DP. Dose-response and threshold-mediated mechanisms in mutagenesis: statistical models and study design. *Mutation Research*, 2000; **464.1**: 87-95; Maynard. Setting air quality standards for carcinogens: an alternative to mathematical quantitative risk assessment – discussion paper. *Human Toxicology* 1995; **14**: 174-186; Hrudey & Krewsky. Is there a safe level of exposure to a carcinogen. *Environmental Science & Technology* 1995; **29.8**: 370-375; and Kraus N, Malmfors T and Slovic P. Intuitive toxicology: expert and lay judgements of chemical risks. *Risk Analysis* 1992; **12**: 215-232

²⁷ WHO/IARC Monograph Vol.83, p 1413

²⁸ WHO/IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol.38, *WHO/IARC* 1986

evidence of the carcinogenicity of sidestream smoke condensates, but this finding on its own could not justify a Group 1 classification. For the evidence that ETS causes lung cancer in humans to be considered sufficient, IARC requires that a positive association is observed for which “a causal interpretation is considered to be credible” and for which “chance, bias and confounding can be ruled out with reasonable confidence.” This leads us into the subject of the epidemiological evidence upon ETS on which the claims and arguments of IARC and others rely.

The epidemiological evidence

Epidemiology

Epidemiology is concerned with the investigation of the pattern of disease and ill-health in the population. It seeks to investigate the incidence or prevalence of disease in the population and whether there are particular features about those people contracting or dying of a disease and/or their lives that may be risk factors.

The label “science”, which has a cachet of authority and prestige, is often of critical importance to the credibility of research and study. Whether that label is appropriate to epidemiology is a topic much debated in various circles. Nonetheless, if science it is, it is one that is: concerned with population, not individuals; is distinct from clinical research and other essentially medical sciences; and is greatly reliant on statistical methods and techniques, particularly in the handling, manipulation and analysis of data.

In reality ETS epidemiological studies are in part health studies and in part statistical exercises. Their measurements have limited credibility in terms of accuracy. That is not to say that they are irrelevant, but they need to be viewed in a proper perspective. Epidemiology is “a crude and inexact science”²⁹.

Yet, in the case of IARC’s overall evaluation of ETS³⁰, reliance is put on epidemiological studies to provide convincing evidence to bolster the weak and inconclusive scientific evidence on ETS.

Types of studies

There are a number of types of epidemiological studies but, in the context of ETS, most are case-control studies, a few, cohort studies.

A **cohort study** follows a population group (the ‘cohort’) over a lengthy time period, either prospectively or retrospectively. It tracks the disease incidence in the cohort, assessing whether there are particular features about those people and their lives which may be risk factors, calculating their relationship to the incidence of the disease.

As cohort studies are generally much larger and require many years coverage to deliver data, they are significantly more costly. Few ETS cohort studies have therefore been undertaken over the past twenty years, even though they are the strongest form of epidemiological evidence³¹, especially when multiple risk factors are involved.

A **case-control** study compares the prevalence of a disease in one group of people with that in another group. In the case of ETS studies, the comparison that has generally been made is of non-smokers living with smoking spouses (the cases) and non-smokers living with non-smoking spouses (the controls). Relatively few studies have attempted to assess the exposure of non-smokers to ETS in the workplace.

In case-control studies, great importance attaches to there being close matching between the two groups - ‘cases’ and ‘controls’ – so that, in as many respects as

²⁹ Dr Charles Hennekens, Harvard School of Public Health, New York Times, 1995

³⁰ IARC Monograph Vol 83, 2004, p. 1413

³¹ Adam H-O, Hunter D, Trichopoulos D. Textbook of Cancer Epidemiology. Oxford University Press, 2002

possible, the people are alike other than in respect of the disease; in other words, they share the same characteristics of age, sex, ethnic origin, economic and social status, behaviour patterns (such as exercise, diet, alcohol consumption) etc. The fact is that such a high degree of matching is rarely possible or achieved. However, the more characteristics that are not matched, the more the adjustments that might need to be made to the data by statistical techniques that may not still produce a true picture.

Case-control studies are often used to test a hypothesis. However, many ETS studies have been based on acceptance of the hypothesis that ETS causes certain diseases, and they have not fully explored and investigated associations between the disease and a wide range of factors. In other words, they have not followed the textbook definition of studies investigating the characteristics that are potential explanations for the occurrence of the disease.

Also, whatever the type of study, there are not any common study designs and “*few epidemiological studies satisfy the stringent methodological criteria that should ideally be applied.*”³² Indeed, it is well recognised and accepted that **all** epidemiological studies are flawed in some way³³. Flaws and errors may be inherent in the overall study design or its detail, the selection of the population groups, the research question, the hypothesis, or the quality of the information. The scope for errors and flaws is immense.

Bias is the term in statistics and epidemiology that is used to describe an error which affects comparison groups unequally, or which leads to inappropriate inferences about one group compared with another. It can arise by way of flaws in study design, data collection or analysis, and ETS studies are particularly susceptible to many forms of bias.

For example, **recall bias** refers to the comparative unreliability of individuals’ memories. Questionnaires are frequently used in case-control studies, administered either face-to-face or over the telephone, or the recipient is sent the questionnaire through the post. In terms of ETS exposure, questions may be asked concerning the frequency and intensity of exposure over very many years – perhaps as many as 30 or more - not least because the diseases which have been associated with tobacco develop over or after many years. In the case of a deceased person, the questions may be asked not of an actual member of the population being studied, but say of a surviving partner, spouse or other family member. This substantially increases recall bias and introduces or aggravates other possible sources of bias.

Misclassification bias is a well recognised trait of ETS studies. Uniform definitions of smoking and non-smoking status are generally absent between ETS studies. This becomes a particularly significant form of bias when comparisons are made between studies, or when results of differently based studies are pooled together.

However, perhaps the most significant and frequently recognised form of misclassification bias arises when people claim to be non-smokers (according to the study definition) but are in fact smokers. For example, it is a fact that smokers tend to marry smokers and a proportion of people are known not to tell researchers the full facts about their present or past smoking habits, and perhaps do not even tell their partners or spouses the truth. In Western populations, misclassification of smokers as non-smokers has variously been estimated to be around 2.5%. In Asian populations, which form a significant proportion of the compendium of epidemiological evidence on ETS, it has been estimated that the misclassification rate is as high as 20%³⁴. The principal reason for this elevated level would appear to be that, while in some societies smoking is

³² Peto, J. Meta-analysis of epidemiological studies of carcinogenesis. In: Mechanisms of Carcinogenesis in Risk Identification, p572, IARC 1992

³³ Bhopal RS. Concepts of Epidemiology: an integrated introduction to the ideas, theories, principles and methods of epidemiology. Oxford University Press 2002

³⁴ Lee PN, Forey BA & Fry SS. Revisiting the association between environmental tobacco smoke and lung cancer risk. III: Adjustment for the biasing effect of misclassification of smoking habits. *Indoor Built Environment* 2001; **10**: 384-398

traditionally acceptable in men, it is perceived as being less so in women who may therefore seek to hide their smoking status from their partner.

On its own, misclassification bias can substantially affect the veracity of crude data and of probability and risk calculations based on the data.

Other bias in data collection can also occur. For example, there cannot be certainty about the precise cause of death, given both the difficulty of establishing that fact and also that *“inaccuracies in the registered cause of death are recognised, especially with multiple causes”*³⁵. Whilst UK death certificates, upon which a study may rely, state what was thought to be the disease or condition directly leading to cause of death, and may also provide now for the mention of other diseases or medical conditions that may have contributed to that cause, they do not record ETS exposure.

Publication bias is also a much debated, significant factor in epidemiology.

*“Publication bias occurs in two quite separate forms. Studies with positive results are more likely to be submitted for publication and more likely to be accepted; and significant findings, such as an association with a particular occupation or exposure, are often given prominence by the authors, particularly in case-control studies, while other exposures that were analysed but were not significant may not be mentioned at all. Both types of bias tend systematically to exaggerate associations in the published literature.”*³⁶

*“Quite different conclusions might be drawn from a review of all published and unpublished studies.”*³⁷

*“The presence of even a modest degree of publication bias can lead to a substantial increase in the estimated risk.”*³⁸

*“The result is a biased understanding of the differences and similarities in the disease patterns of populations and an exaggerated view of the importance of associations between risk factors and disease outcomes.”*³⁹

Publication bias is well recognised as existing particularly when a consensus develops among the ‘experts’ themselves – albeit that consensus opinion may not be correct. Once a large number of people believe something, it can be difficult and costly to argue to the contrary. Academics and researchers who go against the grain can find it difficult to achieve publication of their opinions and research, or struggle to find posts or research funds.

An illustration of the reception that can be given to the publication of views which do not conform to the accepted wisdom – and which thereby demonstrates the strong force that publication bias represents - was provided by the reaction to the publication by the British Medical Journal in May 2003 of a major new ETS study⁴⁰, in respect of which the BMJ carried the front-page headline, *“Passive smoking may not kill”*. This prospective cohort study spanning 39 years measured the relationship between ETS, as estimated by smoking in spouses, and long-term mortality from tobacco related diseases and was conducted on over 100,000 Californian adults between 1960 and 1998. The conclusions of the study stated:

³⁵ Derek Wanless, Securing Good Health for the Whole Population, Final Report, para. 5.49, *HM Treasury*, February 2004

³⁶ Peto, J, Meta-analysis of epidemiological studies of carcinogenesis!;. In: Mechanisms of Carcinogenesis in Risk Identification, ed Vainio H *et al*, *IARC*, 1992

³⁷ *The Lancet*, April 23, 2004 on the research commissioned by the National Institute for Clinical Excellence into the prescribing of anti-depressants drugs to children; and *The Independent*, April 23 2004

³⁸ Copas J & Shi J, *BMJ* 2000;**320**: 417-418

³⁹ Bhopal, R.S, Concepts of Epidemiology: an integrated introduction to the ideas, theories, principles and methods of epidemiology, p 91, *Oxford University Press*, 2002

⁴⁰ Enstrom J E & Kabat G C, Environmental tobacco smoke and tobacco related mortality in a prospective study of Californians 1960-1998, *BMJ* 2003;**326**: 1057-1061

“The results do not support a causal relation between environmental tobacco smoke and tobacco related mortality although they do not rule out a small effect. The association between exposure to environmental tobacco smoke and coronary heart disease and lung cancer may be considerably weaker than generally believed.”

The publication of the study by the BMJ gave rise to a violent storm of criticism from the medical community. In responding, the editor of the BMJ was minded to comment –

“Of course the study we published has flaws – all papers do – but it also has considerable strengths: long follow-up, large sample size, and more complete follow up than many such studies. It’s too easy to dismiss studies like this as fatally flawed with the implication that the study means nothing . . . I found it disturbing that so many people and organisations referred to flaws in the study without specifying what they were. Indeed, this debate was much more remarkable for its passion than its precision.”

“We must be interested in whether passive smoking kills, and the question has not been definitively answered. It’s a hard question, and our methods are inadequate.”⁴¹

ETS epidemiological studies are also particularly prone to a form of bias called **confounding** – distortion because there may be an association of disease with factors other than ETS, such as diet, alcohol consumption, socio-economic circumstances, the level of exercise, the history of disease in the family etc., that happens to correlate with living in a household with a smoker. For example: family history of disease is recognised as being a potentially important factor; lung cancer and heart disease have also been shown in epidemiological studies to be associated with dietary factors, such as low consumption of fruit and vegetables and high consumption of saturated fats, and also alcohol consumption. If the appropriate data on these risk factors are not collected and taken into account, an untrue upward bias in the correlation between ETS and disease may result.

While some ETS studies have attempted to collect information on some confounding factors, there has generally been an inconsistency and inadequacy in approach. Thus the potential of confounding generally remains a most important consideration in study design, data analysis and interpretation. This is particularly so as the diseases in smokers that have been associated with smoking are well recognised to be multi-factorial. For example, cardiovascular disease has been associated with over 300 different factors.

Adjusted data

There are methodological and statistical techniques to adjust for likely confounding and other biases. They are complex and essentially comprise mathematical adjustments to the data collected. As speculative as they often are, they are also not applied uniformly as between individual studies. Nor are they in any event anything other than devices that may not reflect the true situation, and are themselves subject to limitations. They are not an adequate substitute for dealing at the outset with possible sources of bias and confounding in a study.

Presentation and interpretation

Basic epidemiological data on disease occurrence and population structure can also be manipulated and presented in many other ways. Different ways of presenting the same data can have a major impact on the perception of risk.

The findings of epidemiological studies are extracted from an analysis of the data. That data will inevitably be flawed. Interpretation is influenced by the philosophy and theories that researcher holds and the judgements that are made are subjective, not incontrovertible, objective conclusions. Not surprisingly, therefore, epidemiologists are well known for their capacity for criticism.

⁴¹ Richard Smith, editor BMJ, *BMJ* 2003;327:505

Often most contentious of all are judgements made, on the basis of epidemiological studies, of cause and effect. All textbooks and guides on epidemiology caution, in fact strongly argue against such judgements being made, other than very tentatively - particularly when the level of risk is found to be very low - unless the judgement is strongly corroborated by other reliable evidence. In the case of ETS, *“the judgement as to whether the links observed are causal or not remains difficult.”*⁴²

Risk

In everyday language, risk is the possibility that something unpleasant or unwelcome will happen – harm, loss or damage etc. In epidemiology, the prime interest is in the interaction between the probability of a disease or risk and those factors that influence risk. Thus in an ETS case-control study, the incidence of disease in one population exposed to ETS is compared with that in another population not so exposed to ETS.

There are two popular ways of expressing this comparison of measures of association in ETS epidemiological studies – by way of an Odds Ratio or Relative Risk.

An **Odds Ratio** (OR) is calculated simply by dividing one set of odds by another. For example, the odds of disease in those exposed to ETS is divided by the odds for those not exposed⁴³

The epidemiological idea is simple. If the odds of disease in the exposed group is higher than the corresponding odds in the unexposed group (in other words, that it is greater than 1), it provides a measure of the association between the disease and the exposure. If the OR is 1, no association is indicated. If the OR is less than 1, it indicates that the exposure protects against the disease.

A **Relative Risk** (RR) is the ratio of two incidence rates – the incidence rate of the disease in the population exposed to the risk, divided by the rate in the unexposed population.⁴⁴

The same guide applies to the interpretation of a RR as to an OR. In other words, a RR greater than 1 indicates a measure of an association with the disease; 1 indicates no association; and less than 1, a protective effect. Generally, in ETS studies, an OR provides an acceptable estimate of the RR.

Significance testing

Faced with a set of results from a study, a crucial question is how to decide when the difference between the two groups is ‘significant’. In epidemiology and statistics, the description significant does not signify magnitude. Significance is a statistical test intended to determine whether the result is one of pure chance or one indicating an association.

⁴² Report on the health effects of environmental tobacco smoke in the workplace. *Commissioned by the Health and Safety Authority of Ireland and the Office of Tobacco Control from an independent scientific group, January 2003.*

⁴³ For example, take the following table:

ETS	Outcome		Total
	Diseased	Not diseased	
Exposed	a	b	a + b
Not exposed	c	d	c + d
TOTAL	a + c	b + d	a+b+c+d

The disease OR = a/b divided by c/d. Using the arithmetical rule that division by a fraction is equivalent to multiplication by the inverse of the fraction, this is simplified to: $\frac{a \times d}{b \times c}$

⁴⁴ Based on the same table as above, the RR = $\frac{\text{incidence in those exposed to ETS}}{\text{incidence in those not exposed}}$

That is: $\frac{a}{a+b}$ divided by $\frac{c}{c+d}$, in other words $\frac{a(c+d)}{c(a+b)}$.

One of the tests generates what is termed a P-value. On the assumption that the observed difference arises by chance only (in other words that there is no association between the disease and the risk factor) this aims to indicate the frequency with which the result, at least of the order found in the study, would be found if the same experiment were to be repeated many times. Conventionally, but not with any objective good reason other than convenience, 0.05 is regarded as the level below which a P-value is deemed significant. Values of 0.01 are generally regarded as being the standard cut-off for highly significant P-values and 0.001 for very highly statistically significant results.

Alternatively, and now more frequently the practice, statistical significance is indicated by what is termed a Confidence Interval (CI). Rather than stating a RR simply as a central, single figure, a CI which indicates a range of values is calculated. The confidence interval is normally stated at the level of 95% - again not with any objective good reason other than convenience - and is shown in parentheses after the RR, say as 1.2 (95% CI 0.8 - 2.2). A CI at 95% does not mean that there is 95% certainty that the RR is correct. The CI refers to the frequency with which the statistical test used will generate boundaries capturing the true figure. In other words, it indicates that if the test were to be repeated 100 times, on 95 occasions the result would lie within the bounds of the CI. Thus the CI relates to the reliability of the test, not to the parameter.

Again the same guide applies to the interpretation of confidence intervals. If the confidence interval range includes 1.0, as in the sample RR in the preceding paragraph, the reported RR is not regarded as being statistically significant.

Interpreting and misinterpreting Relative Risk

In interpreting what a RR figure means in terms of the population, it is necessary to know what the ratio or incidence of the disease is in the population not exposed to the risk.

Taking the example of lung cancer, as explained in a 1988 UK report⁴⁵, in the UK population, the rate of death or disease amongst non-smokers living with non-smokers (the 'background rate') is generally considered to be 10 per 100,000 person years of the population. This is not a definitive figure, but is one which has come to be accepted as providing a reasonable indication of the true figure.

This 'background' rate – 10 per 100,000 person years – is thus the rate that corresponds to the RR of 1.0. A finding of an OR or RR of 2.0 for non-smokers exposed to ETS would represent a risk of 20 per 100,000, 10 more cases than would normally be anticipated; a RR of 1.20, would indicate 2 more cases a year than would normally be expected in a group of 100,000 people not exposed to ETS. Results from epidemiological studies are generally reported to be at this very low end of excess risk – 1.20 to 1.30. In terms of a percentage increase in risk, an OR or RR of 1.20 amounts to an increase from 0.010% to 0.012%, and one of 1.30 an increase from 0.010% to 0.013%.

However, increases in risk relating to ETS are often stated and promoted in a very different fashion, principally it would seem, so as to gain maximum interest and media coverage for the researchers and the publication of their research. For example, a very small increment in risk – of 0.002% - would not make news that demands loud, clear and unequivocal headlines and sound-bites. So an increase in risk – from 0.010% to 0.012% (from an RR of 1.00 to 1.20 per 100,000) - is frequently stated to be 20%, being the increase in percentage terms that a RR of 1.20 represents of 1.00. To the ordinary person, who does not generally fully comprehend mathematical representations of risk and relative risk, it quite wrongly conveys that 20% of non-smokers will contract lung cancer as a result of exposure to ETS – and that is how such claims have been reported

⁴⁵ 4th Report of the Independent Scientific Committee on Smoking and Health, 1988, para 69, citing IARC Monograph on the evaluation of the carcinogenic risk of chemicals to humans: Tobacco Smoking, Vol 38, 1986 pp 214, 230-232.

by the press and the media. That has the effect of grossly misrepresenting the evidence and the true meaning of relative risk, for the sake of news headlines.

How the magnitude of a Relative Risk finding should be interpreted

As has already been explained, in epidemiology and statistics, the words 'significant' or 'statistically significant', have nothing to do with the magnitude of a measured difference. Statistical significance does not imply real-life significance. It is a probability statement of the likelihood that the results did not occur by luck or chance if the groups were really alike; about how certain it is that the results are not pure chance.

It has also already been explained how, traditionally, conventionally and historically, a RR is considered to be statistically significant – not a fluke - when at a 95% CI it does not include 1.0, albeit that the choice of the value of 95% CI is arbitrary.

There are conventional guides for the interpretations of RR figures, particularly in relation to tentative cause and effect associations.

A RR finding of around 3.0 is generally considered necessary as one of the criteria to support conclusions of a causal association. For example:

"The association between cancer occurrence and exposure to either extremely low frequency (ELF) or radiofrequency (RF) fields is not strong enough to constitute proven causal relationship, largely because the relative risks in the published reports have seldom exceeded 3.0..."⁴⁶

A RR of 2.0 or less is generally regarded as being weak and not indicative of a causal association.. The nearer a RR is to 1.0, the more likely this is to be the case:

"...relative risks of less than 2.0 are considered small and are usually difficult to interpret ... Such increases may be due to chance, statistical bias, or effects of confounding factors that are sometimes not evident."⁴⁷

"...when the relative risk lies between 1 and 2 . . . problems of interpretation may become acute, and it may be extremely difficult to disentangle the various contributions of biased information, confounding of two or more factors, and cause and effect."⁴⁸

"...when the relative risk is higher than 1 but less than 2, the individual who has been exposed and has developed the disease is more likely than not to have developed the disease for reasons not entirely due to the exposure."⁴⁹

"Until the 1980s, epidemiologists were concerned mainly with relative risks that exceeded about 1.5 and were often much higher. Many controversies now centre on much lower risks, a notable example being the effect of 'passive smoking' on lung cancer risk. The pooled data show a statistically significant effect, and all studies are consistent with a relative risk of about 1.3 (US National Research Council, 1986). In view of the many difficulties discussed above, however, it can plausibly be argued that such small effects are beyond the limits of reliable epidemiological inference (particularly for lung cancer, in which the major cause produces large relative risks), as smoking habits may be inaccurately recorded and are correlated with many other social and occupational factors, including the smoking habits of spouses. A number of spurious associations with relative risks for lung cancer of this order might thus be found in a large enough sample. The observations that short-service workers in various industries suffer

⁴⁶ Evaluation of the potential carcinogenicity of electromagnetic fields, *US EPA Review*, October 1990

⁴⁷ National Cancer Institute, USA, *Press Release*, 26 October 1994

⁴⁸ Doll, R and Peto, R, *The causes of cancer*, p. 1219, *OUP 1981*

⁴⁹ Adam H-O, Hunter D, Trichopoulos D. *Textbook of Cancer Epidemiology*, *Oxford University Press* 2002

*elevated risks for lung cancer, which seem unlikely to be caused by their recorded occupational exposure, further illustrates the problem.*⁵⁰

In 1998, in answer to a Parliamentary Question, the Minister provided the Government's guidance on the interpretation of relative risks, albeit incorporating an incorrect explanation of a CI:

“Relative risk provides a measure of the strength of association between a factor and an illness. It is an important way of measuring increases or decreases of risk over time or between different groups by comparing the incidence of an illness or hazard within a population to some baseline (for example, if drinkers are twice as likely to suffer from a particular disease as compared with the general population, a factor of 2 may be cited). A stronger association of greater than 2 is more likely to reflect causation than is a weaker association of less than 2 as this is more likely to result from methodological biases or to reflect indirect associations which are not causal. The significance of any such number does though need to be considered in context and from a number of viewpoints.

First, there is a statistical significance: in other words, what confidence is there in the number itself. This will depend on the quality and extent of the available data. Scientists usually express these by giving a confidence interval: rather than by saying that the relative risk factor is 2, they will say that (for example) one can be 95 per cent certain that it lies between 1.6 and 2.4.

Even when the strength of an association is precisely determined, it is insufficient in itself to confirm a direct causal link between possible cause and illness. The strength of an association is only one of several criteria which must be considered in the assessment of causation. Other criteria include:

the cause must precede the effect;

the biological plausibility of the association - is the association consistent with other knowledge e.g. experimental evidence?

the consistency of the finding – is the same result obtained from different studies using different methodologies elsewhere?

the presence of a “dose-response” relationship – an increased response to the possible cause being associated with an increased risk of developing the illness.

All these factors would be taken into account in trying to pinpoint cause.

The practical significance of risk factors, also needs to be considered and depends on how great is the underlying risk. Doubling a very small probability (risk), say 1 in 10,000,000, still results in only a very small risk of illness. Doubling a risk of, say, 1 in 100 could, depending on its nature, be more serious.

In practice, scientific judgments will be made and debated on a case-by-case basis. The Government can draw on the expertise of independent scientific advisory committees which are constituted to provide balanced judgment on the questions covered above⁵¹.

Meta-analysis

Before considering the actual results of individual ETS epidemiological studies, it is essential to explain meta-analysis, a weight-of-evidence technique that involves the

⁵⁰ Peto, J. Meta-analysis of epidemiological studies of carcinogenesis. In: Mechanisms of Carcinogenesis in Risk Identification, ed Vainio H, p573, IARC 1992

⁵¹ Baroness Jay of Paddington, Minister for Public Health, House of Lords, Written Answer, *House of Lords, Official Report*, 31 March 1998, Cols. 31-32.

quantitative synthesis of the results of separate studies, to provide a summary of the pooled results.

The objective of meta-analysis is, by combining studies, to increase power to detect significant results. However, for meta-analysis to be a valid and reliable approach, studies need to be similar and comparable in design and many other respects, otherwise the result is no better than mixing apples with oranges. Such inappropriate mixes may result from pooling studies of widely varying design and methodology; individual studies may be distorted by confounding and other types of bias which may not be common to all studies; there may be studies from different countries and populations in respect of which there may be significant and varying confounding variables; studies will have been undertaken in significantly different time frames that may affect results; there will be selectivity by virtue of the inclusion of studies based on the researcher's subjective impressions of study quality; and there will be publication bias.

Individual ETS studies exhibit wide variations in design, methodology, data collection, country, population and study size. The findings show little consistency and are generally weak and well below the levels of RR that are considered to be indicative of a causal association. Indeed, even those few studies that have found an association that is 'statistically significant', have reported a level of RR which could well be accounted for by bias or confounding. All these factors make interpretations and comparisons, and pooling for the purposes of meta-analysis, both extremely difficult and highly contentious.

Quite apart from virtually all ETS studies having been of individual design and methodology, the vast majority have been of populations in the USA and Asia, which are populations different in important respects from the UK. The studies have also been undertaken over a time period since 1981 and there is a marked difference in the findings between those studies conducted before and after 1989.

The difficulties of extrapolating data on one population and applying it to another on the basis that one group of people is broadly equivalent to another has been vividly illustrated by the extrapolation of risk scoring methods for coronary heart disease derived from the US Framingham heart study⁵² and its application to the UK. The Framingham study played a key role in quantifying risks such as smoking and high cholesterol. The UK researchers compared the Framingham results with the British regional heart study⁵³. They found that using Framingham, there was an over-estimation of the risk of non-fatal coronary events of 57%, and also that 84% of British heart deaths occurred in the 93% of men classified as low risk by Framingham criteria. The fact is that substantial variations in coronary heart disease are found between different regions and different ethnic groups, and according to socio-economic status and family history of coronary heart disease.

Meta-analysis does not solve the problem of the differences that exist between individual studies, the results of which are being combined. It does not resolve distortions in those studies arising, for example, out of varying study quality, the accuracy or otherwise of measurements of exposure and disease, the nature of individual populations, the times of the studies, distortions in them by confounding and other forms of bias, or the consequences of publication bias. Indeed, meta-analysis can introduce its own form of bias by virtue of the criteria used to select studies for inclusion in the meta-analysis, and by any overall adjustments to the results that the meta-analysis technique may make.

Thus, a statistically significant result from a meta-analysis of epidemiological data does not necessarily establish that any underlying effect exists.⁵⁴ Meta-analysis can result in

⁵² Dawber T R *et al.* An approach to longitudinal studies in a community: The Framingham Study, *Annals of the New York Academy of Science*, 1996; 107:539-556

⁵³ Brindle P *et al.* Predictive accuracy of the Framingham coronary risk score in British men: prospective cohort study, *BMJ* 2003; **327**:1267-1270

⁵⁴ Shapiro S. Meta-analysis/Shmeta-analysis. *American Journal of Epidemiology*, 1994; **140**: 771-778 and Petitti DB. Of babies and bathwater. *American Journal of Epidemiology*, 1994; **140**: 779-782

a combined relative risk estimate that has narrow confidence limits, and may appear to be precise, but this can in fact be an inaccurate estimate of the true association, if any.

“The interpretation of systematic reviews is as prone to errors as is the interpretation of any data. Thus the reader should ask: Do they reflect the weight of evidence? Was due allowance made for the strengths of the research methods? Were defects in the studies taken into account? Review articles may appear unchallengeable, particularly those which have involved extensive searches and have combined findings using meta-analysis techniques. This semblance of infallibility should be rejected; review articles are prepared by people, and people make mistakes.”⁵⁵

A misuse of relative risk findings

In a recent peer reviewed article⁵⁶, researchers at the US National Center for Health Statistics urged great caution in the use of current estimates of the number of deaths attributable to obesity in the United States and urged researchers to devote greater efforts to improve data and methods. They found that the estimates were reliant on epidemiological studies that were not consistent; the RR findings also fell in the range 1.0 to 2.0; they did not adequately allow for variation with age; and they were not necessarily appropriate for the total US population. They found that, instead of the claim frequently stated in scientific and lay literature that obesity causes about 300,000 deaths per year in the USA, the true figure could be almost 200,000 less. In other words, they argue that the claimed 300,000 deaths are more likely to be generated by statistics than by obesity.

A similar, serious misuse of relative risk findings occurs in the case of ETS. Various figures are bandied about as to the number of deaths caused in the UK population by ETS. These figures variously cover a wide range, from 600 lung cancer deaths per year up to 13,620 deaths by coronary heart disease⁵⁷. Often their precision conveys accuracy and reliability. That is far from being the true case.

Indeed, such attempts to calculate for ETS what in epidemiology is called a ‘population attributable fraction’ - which endeavours to answer the question what proportion of the incidence of the disease in the population as a whole is attributable to ETS (put alternatively, how many lives could be saved by removing ETS?) - are not valid or reliable.

Two fundamental, necessary assumptions underlying valid estimation of a population attributable fraction are a clear, unequivocal causal relationship between the risk factor and the disease, and the absence of confounding. Those assumptions cannot be made in the case of ETS. Thus, the calculations that are made in respect of ETS can only be regarded as mere arithmetical exercises that create data with no veracity.

Yet, despite the important conceptual and computational problems⁵⁸ that surround population attributable fractions, figures for ETS continue to be promoted and publicised, again giving the public an wholly false impression of the true facts, just as is the case with the relative risk estimates on which they are based.

THE FINDINGS OF ETS EPIDEMIOLOGICAL STUDIES

In the following series of tables, the numerous individual ETS studies and reviews which have been undertaken and their findings are listed by the TMA⁵⁹. The lists exclude studies that have been superseded by later results of the same researchers, where data

⁵⁵ Crombie IK. The Pocket Guide to Critical Appraisal, p.60. *BMJ Publishing*, 1996

⁵⁶ Flegal KM, Williamson DF, Pamuk ER & Rosenberg HM. Estimating deaths attributable to obesity in the United States, *American Journal of Public Health*, September 2004; **94** (9); 1486-1489

⁵⁷ Peterson S & Peto V. Smoking Statistics 2004, *British Heart Foundation Health Promotion Research Group*, 2004

⁵⁸ For an explanation of these, see Rockhill B, Newman B and Weinberg C. Use and Misuse of Population Attributable Fractions. *American Journal of Public Health*, January 1998; **88**(1);15-19

⁵⁹ Based on papers on epidemiological evidence on ETS and heart disease by PN Lee, available at www.pnlee.co.uk

has been included in other studies, or where the data or size of study is virtually universally regarded as being inadequate. The reference numbers for each study in Table 1 are carried through in Tables 2 to 4 and the reference sources are listed in Annex 1 to this document. The reference sources for Table 8 are listed in Annex 2.

In order that there can be confidence in these lists, the comparable listing of studies and findings as published by IARC in its recent monograph⁶⁰ are also shown. IARC included studies published only up until 2001.

The RRs listed for the IARC publication are the reported crude relative risks, or the adjusted estimates where the crude ones were not reported. Those in the TMA listing are adjusted for covariates, if adjusted data was reported; otherwise the crude, unadjusted figures are stated. Throughout, therefore, differences will be found between the TMA and IARC listings, accounted for by the fact that the TMA has adopted the adjusted RRs as reported, whereas IARC takes the crude RR and only adopts the adjusted RR when the crude figure is not available. However, there are no significant differences between the two lists when reported adjusted estimates (as stated in the TMA listing) are substituted for the crude estimates in the IARC listing.

The only other differences arise by virtue of the facts that: IARC, unconventionally, chooses to state RRs 'rounded' to one decimal point; in the TMA listing, where necessary, relative risks and 95% CIs were estimated from data presented and, where studies reported only by the level of exposure, the adjusted risks and intervals were used to estimate effective numbers of cases and controls, which could then be combined to allow estimation of risks for overall exposure.

Table 1 lists the reported relative risks for lung cancer among lifelong non-smoking women in relation to smoking by the husband. Of the 46 studies listed by IARC (one of which is of an unpublished analysis of data provided to IARC in a personal communication by Kreuzer), IARC states that only 7 report a RR estimate that is statistically significant (study nos. 4, 9, 17, 20, 35a, 48 and 54). These studies were of populations in Greece (1), Hong Kong (2), China (1), Taiwan (1), USA (1) and Russia (1).

However, crucially, of these 7 studies, 4 are wholly unadjusted for age (a most serious shortcoming of an epidemiological study) or for other factors.

In 4 of the 7, the lower bound of the CI is extremely close to 1.00, at which level the estimate would not be statistically significant (in study nos. 9, 20, 35a and 48. the lower bound, as stated conventionally at two decimal points, is 1.09, 1.08, 1.04 and 1.06).

The TMA list of studies is more up to date than the list published by IARC and also includes several earlier studies not included by IARC. Of the 62 studies listed by the TMA, 4 additional to those listed by IARC report statistically significant estimates (study nos. 6, 27, 41 and 44). These 4 studies were of populations in Japan, Greece and China (2); in 3 of them the lower bound of the CI is very close to 1.00. In any event, 2 of the 4 studies were included in IARC's listing but no. 6 was not stated to be statistically significant by virtue of the 'rounding' down of the CI lower bound from 1.02 to 1.0 and, on the crude, unadjusted data (which was taken by IARC to determine the significance test) no. 27 was not statistically significant at 0.9.

Quite apart from the fact that around 82% of the studies show no statistically significant association between smoking by the husband and the development of lung cancer in the non-smoking wife, and that even where there is a statistically significant association, it is generally extremely weak, there are other important observations to be made.

⁶⁰ WHO/IARC Monograph, Vol 83, 2004

Some of the very largest studies show no association, including 4 of the 5 studies involving over 400 lung cancer cases: One (study no. 31) reported no statistically significant association between lung cancer and any index of ETS exposure; another (no.29) even reported a significantly reduced risk of lung cancer for non-smoking women married to smokers.

About 20% of the studies did not adjust for age in the analysis, a standard procedure in epidemiology to avoid bias; those studies reported much stronger associations with spousal exposure than those that did age-adjust.

ETS spousal studies are also particularly susceptible to other, various biasing factors, including failure to consider diet, lifestyle, family medical history, education, socio-economic status and other factors recognised as being different between smoking and non-smoking households. The studies are also well recognised as being particularly susceptible to misclassification, especially in the case of Asian populations. The studies also rely on reported, rather than objectively measured ETS exposure data; and publication bias needs to be taken into account.

TABLE 1
Relative risk of lung cancer among lifelong non-smoking women in relation to smoking by the husband

Ref	Author	Year	Location	No. of lung Cancers	Relative Risk	Confidence Interval at 95%	IARC 2004 RR **
1	Garfinkel 1	1981	USA	153*	1.17	0.85 – 1.61	1.2 (0.9 - 1.4)
2	Chan	1982	Hong Kong	84	0.75	0.43 – 1.30	0.8 (0.4 - 1.3)
3	Correa	1983	USA	25	2.07	0.81 – 5.25	2.1 (0.8 - 5.3)
4	Trichopoulos	1983	Greece	77	2.08	1.20 – 3.59	2.1 (1.2 - 3.8)
5	Buffer	1984	USA	41	0.80	0.34 – 1.90	0.8 (0.3 - 1.9)
6	Hirayama	1984	Japan	200*	1.45	1.02 – 2.08	1.5 (1.0 - 2.1)
7	Kabat 1	1984	USA	53	0.79	0.25 – 2.45	0.8 (0.3 - 2.5)
8	Garfinkel 2	1985	USA	134	1.23	0.81 – 1.87	1.2 (0.8 - 1.9)
9	Lam W	1985	Hong Kong	75	2.01	1.09 – 3.72	2.0 (1.1 - 3.7)
10	Wu	1985	USA	31	1.20	0.50 – 3.30	1.2 (0.5 - 3.3)
11	Akiba	1986	Japan	94	1.50	0.93 – 2.76	1.5 (0.9 - 2.6)
12	Lee	1986	UK	32	1.00	0.37 – 2.71	1.0 (0.4 - 2.6)
13	Brownson 1	1987	USA	19	1.68	0.39 – 6.90	1.5 (0.4 - 6.0)
14	Gao	1987	China	246	1.30	0.89 – 1.91	1.2 (0.8 - 1.7)
15	Humble	1987	USA	20	2.20	0.76 – 6.56	2.3 (0.7 - 6.8)
16a	Koo	1987	Hong Kong	88	1.64	0.87 – 3.09	1.6 (0.9 - 2.7)
17	Lam T	1987	Hong Kong	202	1.65	1.16 – 2.35	1.7 (1.2 - 2.4)
18	Pershagen	1987	Sweden	83	1.20	0.70 – 2.10	1.0 (0.6 - 1.7)
19	Butler	1988	USA	8*	2.02	0.48 – 8.56	2.0 (0.5 - 8.6)
20	Geng	1988	China	54	2.16	1.08 – 4.29	2.2 (1.1 - 4.3)
21	Inoue	1988	Japan	28	2.25	0.77 – 8.85	2.6 (0.7 - 8.8)
22	Shimizu	1988	Japan	90	1.08	0.64 – 1.82	1.1 (0.6 - 1.8)
23	Choi	1989	Korea	75	1.63	0.92 – 2.87	1.6 (0.9 - 2.9)
24	Hole	1989	Scotland	6*	1.89	0.22 – 16.12	
25	Svensson	1989	Sweden	38	1.36	0.53 – 3.49	
26	Janerich	1990	USA	146	0.75	0.47 – 1.20	
27	Kalandidi	1990	Greece	91	2.11	1.09 – 4.08	1.6 (0.9 - 2.9)
28	Sobue	1990	Japan	144	1.13	0.78 – 1.63	1.1 (0.7 - 1.5)
29	Wu-Williams	1990	China	417	0.70	0.60 – 0.90	0.8 (0.6 - 1.0)
30	Liu Z	1991	China	54	0.77	0.30 – 1.96	0.7 (0.3 - 1.7)
31	Brownson 2	1992	USA	432	1.00	0.80 – 1.20	1.0 (0.8 - 1.2)
32	Stockwell	1992	USA	210	1.60	0.80 – 3.00	1.6 (0.8 - 3.0)
33	Du	1993	China	75	1.09	0.64 – 1.85	1.2 (0.7 - 2.1)
34	Liu Q	1993	China	38	1.72	0.77 – 3.87	1.7 (0.7 - 3.8)
35a	Fontham	1994	USA	653	1.29	1.04 – 1.60	1.3 (1.0 - 1.5)

Ref	Author	Year	Location	No. of lung Cancers	Relative Risk	Confidence Interval at 95%	IARC 2004 RR **
36	Layard	1994	USA	39	0.58	0.30 – 1.13	
37	DeWaard	1995	Netherlands	23	2.57	0.84 – 7.85	
38	Kabat 2	1995	USA	69	1.08	0.60 – 1.94	1.1 (0.6 - 2.0)
39	Schwartz	1996	USA	185	1.10	0.72 – 1.68	
40	Sun	1996	China	230	1.16	0.80 – 1.69	1.2 (0.8 - 1.7)
41	Wang S-Y	1996	China	82	2.53	1.26 – 5.10	
42	Wang T-J	1996	China	135	1.11	0.67 – 1.84	1.1 (0.7 - 1.8)
43a	Cardenas	1997	USA	246*	1.20	0.80 – 1.60	1.2 (0.8 - 1.6)
44	Zheng	1997	China	69	2.52	1.09 – 5.85	
46	Boffetta 1	1998	W. Europe	509	1.11	0.88 – 1.39	1.0 (0.8 - 1.3)
47	Shen	1998	China	70	0.75	0.31 – 1.78	1.5 (0.7 - 3.3)
48	Zaridze	1998	Russia	189	1.53	1.06 – 2.21	1.6 (1.1 - 2.3)
49	Boffetta 2	1999	Europe	66	1.00	0.50 – 1.90	
50	Jee	1999	Korea	79*	1.72	0.93 – 3.18	1.9 (1.0 - 3.5)
51	Rapiti	1999	India	41	1.20	0.50 – 2.90	1.0 (0.4 - 2.4)
52	Speizer	1999	USA	35*	1.50	0.30 – 6.30	
53	Zhong	1999	China	504	1.10	0.80 – 1.50	1.2 (0.8 - 1.6)
54	Lee C-H	2000	Taiwan	268	1.87	1.29 – 2.71	1.7 (1.3 - 2.4)
55	Malats	2000	Europe/Brazil	105	1.50	0.77 – 2.91	
56	Wang L	2000	China	200	1.03	0.60 – 1.70	
56a	Kreuzer***	2000	Germany	100			0.9 (0.6 - 1.4)
57	Johnson	2001	Canada	71	1.20	0.62 – 2.30	1.2 (0.6 - 4.0)
58	Lagarde	2001	Sweden	242	1.15	0.84 – 1.58	
59	Nishino	2001	Japan	24*	1.80	0.67 – 4.60	1.9 (0.8 - 4.4)
60	Ohno	2002	Japan	191	1.00	0.67 – 1.49	
62	Seow	2002	Singapore	176	1.29	0.93 – 1.80	
63	Enstrom	2003	USA	177*	0.94	0.66 – 1.33	
64	Zatloukal	2003	Czech Republic	84	0.48	0.21 – 1.09	

Notes

* indicates a prospective study, all others being case-control studies.

** IARC RRs are the crude RRs, or the adjusted estimates when the crude ones were not available.

*** This reference is to a personal communication from Kreuzer to IARC and is reported to be the results from analysis excluding cases and controls already included in study no.46.

The IARC Monograph excludes studies which gave results for men and women combined and does not separate the two. These studies are 24, 26, 39 & 49

A variety of indices of ETS exposure were used in these studies. Nearly all considered smoking by the spouse (or partner) as a measure of exposure, with a number of studies considering ETS exposure by other household members, in the workplace, in childhood or in social situations.

Table 2 lists the reported relative risks for lung cancer among lifelong non-smoking men in relation to smoking by the wife. The IARC report lists only one study (a cohort study by *Hirayama, 1984*) out of a total of 11 that reported a statistically significant finding. That remains the case including the further 11 studies listed by the TMA. In other words, only 1 out of 22 studies reported a finding that was statistically significant.

TABLE 2
Relative risk of lung cancer among lifelong non-smoking men in relation to smoking by the wife

Ref.	Author	Year	Location	No. of Lung cancers	Relative Risk	Confidence Interval at 95%	IARC 2004
3	Correa	1983	USA	10	1.97	0.38 – 10.32	2.0 (0.2 - 11.8)
5	Buffler	1984	USA	11	0.52	0.14 - 1.79	0.5 (0.1 – 2.2)
6	Hirayama	1984	Japan	64*	2.25	1.05 – 4.76	2.3 (1.1 - 4.8)
7	Kabat 1	1984	USA	25	1.00	0.20 – 5.07	1.0 (0.2 - 6.7)
11	Akiba	1986	Japan	19	1.80	0.39 – 6.96	2.1 (0.5 – 8.6)
12	Lee	1986	UK	15	1.30	0.38 – 4.39	1.3 (0.3 – 5.4)
15	Humble	1987	USA	8	4.08	0.70 – 23.91	
23	Choi	1989	Korea	13	2.73	0.49 – 15.21	2.7 (0.5 - 15.2)
24	Hole	1989	Scotland	3*	3.52	0.32 – 38.65	
26	Janerich	1990	USA	45	0.75	0.31 – 1.78	
36	Layard	1994	USA	21	1.47	0.55 – 3.94	
38	Kabat 2	1995	USA	41	1.60	0.67 – 3.82	1.6 (0.7 - 3.9)
39	Schwartz	1996	USA	72	1.10	0.60 – 2.03	
43a	Cardenas	1997	USA	116*	1.10	0.60 – 1.80	1.1 (0.6 - 1.8)
44	Zheng	1997	China	25	0.67	0.22 – 2.04	
45	Auvinen	1998	Finland	44	0.69	0.28 – 1.74	
46	Boffetta	1998	Western Europe	141	1.47	0.81 – 2.66	1.3 (0.8 - 2.2)
55	Malats	2000	Europe/Brazil	17	1.50	0.41 – 5.43	
56	Wang L	2000	China	33	0.56	0.20 – 1.40	
56a	Kreuzer**	2000	Germany	23			0.4 (0.1 - 3.0)
58	Lagarde	2001	Sweden	191	1.15	0.81 – 1.63	
63	Enstrom	2003	USA	79*	0.63	0.33 – 1.22	

Notes

The Notes at the foot of Table 1 are also relevant to this Table.

* indicates a prospective study, all others being case-control studies

** This reference is to a personal communication from Kreuzer to IARC and is reported to be the results from analysis excluding cases and controls already included in study no.46.

In these studies, the index of exposure is based on smoking by the spouse or, if not available, the nearest equivalent: otherwise exposed to ETS at home.

Table 3 lists the reported relative risks for lung cancer among non-smokers exposed to ETS during childhood. IARC reports that study results are contradictory – 3 reporting a statistically significant increased risk from smoking by the mother (this is not borne out by IARC’s report of the data, which indicates only 2 studies), 2 of those same studies reporting a statistically significant increase in RR related to exposure from the father or either parent, and 1 study (no.46) finding a significant inverse association (a protective one) with exposure from the father or either parent. IARC states: *“Overall, the findings from studies of childhood cancer and exposure to parental smoking are inconsistent and are likely to be affected by bias.”*

TABLE 3

Relative risk of lung cancer among lifelong non-smokers in relation to ETS exposure in childhood

Ref	Author	Location	Sex	Relative Risk	Confidence Interval at 95%
8	Garfinkel 2	USA	F	0.91	0.58 – 1.42
10	Wu	USA	F	0.60	0.20 – 1.70
14	Gao	China	F	1.10	0.70 – 1.70
16a	Koo	Hong Kong	F	0.56	0.21 – 1.50
18	Pershagen	Sweden	F	1.00	0.40 – 2.30
25	Svensson	Sweden	F	3.30	0.50 – 18.80
26	Janerich	USA	Combined	1.33	0.86 – 2.06
28	Sobue	Japan	F	1.28	0.71 – 2.31
31	Brownson 2	USA	F	0.80	0.60 – 1.10
32	Stockwell	USA	F	1.66	0.80 – 3.44
35	Fontham	USA	F	0.89	0.72 – 1.10
38	Kabat 2	USA	M	0.90	0.43 – 1.89
			F	1.63	0.91 – 2.92
40	Sun	China	F	2.29	1.56 – 3.37
42	Wang T-J	China	F	0.91	0.56 – 1.48
46	Boffetta 1	West Europe	M	0.79	0.52 – 1.21
			F	0.77	0.61 – 0.98
48	Zaridze	Russia	F	0.92	0.64 – 1.32
49	Boffetta 2	Europe	Combined	0.60	0.30 – 1.20
51	Rapiti	India	M	1.09	0.38 – 3.18
			F	12.0	4.30 – 32.0
53	Zhong	China	F	0.93	0.72 – 1.20
54	Lee C-H	Taiwan	F	2.10	1.40 – 3.14
56	Wang L	China	M	1.46	0.60 – 3.70
			F	1.51	1.00 – 2.20
57	Johnson	Canada	F	1.38	0.81 – 2.34
60	Ohno	Japan	F	1.00	0.51 – 1.98
61	Rachtan	Poland	F	3.31	1.26 – 8.69
64	Zatlopukal	Czech Republic	F	1.61	1.01 – 2.57

Note

This table is compiled by the TMA and it does not differ in any significant respect from the studies listed by IARC.

Two other studies – Nos 3 and 11, reported finding no association but gave no detailed results.

Table 4 lists for lung cancer the reported relative risks among life-long non-smokers reportedly exposed to ETS in the workplace. IARC states the following⁶¹: *“In total, 23 studies have been published on exposure to secondhand smoke at the workplace. The results from these studies are mixed with some showing a positive association and others not. Only one study reported a statistically significant association between exposure to secondhand smoke at the workplace and risk for lung cancer (Reynolds et al, 1996).”*

This reference to Reynolds *et al*, 1996 is to a letter⁶² in which the correspondents ‘modestly enhanced’, as they described, the estimates of risk reported in their original article that reported study no. 35b. This enhanced estimate is shown in the table.

If regard is had for the adjusted RR reported, a second study (no. 53) also finds a statistically significant association. However, IARC’s significance test relates to the crude analysis that does not show a statistically significant result.

⁶¹ WHO/IARC Monographs Vol 83, p. 1257

⁶² Reynolds P, Fontham ETH, Wu A, Buffler PA, Greenberg RS Letters to the Editor, *Journal of the American Medical Association*, 1996; 275(6):441-2

**TABLE 4 –
Relative risk of lung cancer among life-long non-smokers reportedly exposed to ETS exposure
in the workplace.**

Ref	Author	Publication	Location	Sex	Relative Risk	Confidence Interval at 95%	IARC 2004
7	Kabat	1984	USA	M	3.27	1.01 – 10.62	3.3 (1.0 - 10.6)
				F	0.68	0.32 – 1.47	0.7 (0.3 - 1.5)
8	Garfinkel	1985	USA	F	0.93	0.55 – 1.55	0.9 (0.7 - 1.2)
10	Wu	1985	USA	F	1.30	0.50 – 3.30	1.3 (0.5 - 3.3)
12	Lee	1986	UK	M	1.61	0.39 0 6.60	1.6 (0.4 - 6.6)
				F	0.63	0.17 – 2.33	0.6 (0.2 - 2.3)
16	Koo	1987	Hong Kong	F	1.19	0.48 – 2.95	1.2 (0.5 - 3.0)
22	Shimzu	1988	Japan	F	1.18	0.70 – 2.01	1.2 (0.6 - 2.6)
	Butler	1988		M			1.0 (0.2 - 5.4)
				F			0.98 (0.2 - 5.4)
26	Janerich	1990	USA	C	0.91	0.61 – 1.35	
27	Kalandidi	1990	Greece	F	1.70	0.69 – 4.18	1.4 (0.8 - 2.5)
29	Wu-Williams	1990	China	F	1.06	0.80 – 1 40	1.2 (1.0 - 1.6)
31	Brownson	1992	USA	F	0.98	0.74 – 1.31	
35b	Fontham	1994	USA	F	1.56	1.21 – 2.02	1.4 (1.1 - 1.7)
38	Kabat 2	1995	USA	M	1.02	0.50 – 2.09	1.0 (0.5 - 2.1)
				F	1.15	0.62 – 2.13	1.2 (0.6 - 2.1)
39	Schwartz	1996	USA	C	1.50	1.00 – 2.00	1.5 (1.0 - 2.2)
40	Sun	1996	China	F	1.38	0.94 – 2.04	1.4 (0.9 - 2.0)
42	Wang T-J	1996	China	F	0.89	0.46 – 1.73	0.9 (0.5 - 1.8)
43b	Cardenas	1997	USA	M	1.09	0.62 – 1.91	
				F	1.00	0.65 – 1.54	
46	Boffetta 1	1998	Western Europe	M	1.3	0.68 – 1.86	1.2 (0.8 - 1.8)
				F	1.19	0.94 – 1.51	1.3 (0.9-1.5)
48	Zaridze	1998	Russia	F	0.88	0.55 – 1.41	0.9 (0.6 - 1.4)
49	Boffetta 2	1999	Europe	C	1.50	0.80 – 3.00	1.0 (0.5 - 1.8)
51	Rapiti	1999	India	C	1.10	0.30 – 4.10	1.1 (0.3 - 4.1)
53	Zhong	1999	China	F	1.70	1.30 – 2.30	1.4 (1.0 - 1.8)
54	Lee C-H	2000	Taiwan	F	0.91	0.52 – 1.62	1.2 (0.7 - 1.9)
56	Wang L	2000	China	C	1.56	0.70 – 3.30	1.6 (0.7 - 3.3)
56a	Kreuzer*	2000	Germany	M			0.5 (0.2 - 1.3)
				F			1.1 (0.7 - 1.7)
57	Johnson	2001	Canada	F	1.36	0.80 – 2.31	1.2 (0.7 - 2.0)
60	Ohno	2002	Japan	F	1.38	0.92 – 2.05	

Notes

* This reference is to a personal communication from Kreuzer to IARC and is reported to be the results from analysis excluding cases and controls already included in study no.46.

Study No 32 also reported finding no association but gave no detailed results.

Lung cancer – meta-analysis

In IARC's Monograph Vol. 83, Table 2.7 (p.1265) lists the results of selected meta-analyses of risk for lung cancer in never smokers exposed to secondhand smoke from the spouse. The pooled relative risk (95% CI) in the selected studies ranges from 1.12 to 1.39. Adjusted pooled relative risks are provided but without any statement of the confidence intervals. Furthermore, in only 1 review out of the 10 quoted is adjustment made for misclassification, dietary confounding and exposure to ETS other than from the spouse, and in that case the adjustments made have been questioned. No adjustments are made for other biases.

Although at Table 2.10 in its 2004 Monograph IARC presents its own updated meta-analyses of the evidence relating ETS exposure to lung cancer risk in non-smokers, these analyses are not adjusted for bias or confounding. Instead IARC relies heavily on previously published meta-analyses – the majority of which are old and based on limited data – to support its claim that, after controlling for "*some potential sources of bias and confounding*", the excess risk remains. That is an highly contentious and extremely fragile claim, given the very low level of the excess risk before adjustments and the evidence that is available with regard to the possible extent of biases and confounding.

Here at table 5, a shortened version of IARC's updated meta-analyses are shown. They are based on crude, unadjusted estimates of relative risks and only when these are not available on reported adjusted estimates

Table 5
Summary of IARC up-dated meta-analyses of the relative risk for lung cancer in never-smokers exposed to specified sources of secondhand smoke

Source of exposure	No of studies	Sex of Subject	Pooled RR (95% CI)
Spouse	46	Women	1.24 (1.14 - 1.34)
	11	Men	1.37 (1.02 - 1.83)
Childhood by either parent	6	Men & Women combined	1.14 (0.77 - 1.70)
Workplace	7	Men & Women combined	1.03 (0.86 – 1.23)

Here, at Table 6, the results of combining the overall incidence for each of the four most commonly used indices of ETS are shown, as calculated from the studies listed here in Table 1⁶³:

TABLE 6
Meta-analysis : Lung Cancer

Index of ETS Exposure	Estimates Combined	Fixed effects		Random Effects	
		RR	95% CI	RR	95% CI
Smoking by husband	62	1.17	1.11 - 1.24	1.22	1.13 – 1.33
Smoking by wife	21	1.13	0.95 - 1.35	1.13	0.95 – 1.35
Workplace exposure	30	1.21	1.11 -1.31	1.21	1.11 – 1.31
Childhood exposure from any co-habitant	29	1.07	0.99 – 1.16	1.18	1.00 --1.40*
Childhood exposure from mother specifically	9	0.96	0.77 – 1.20	0.98	0.77 – 1.25
Social exposure	12	1.04	0.92 - 1.17	1.02	0.80 – 1.28

⁶³ From Lee PN, available at www.pnlee.co.uk

Notes

*This estimate is inflated by one study (No 14, Gao – China) reporting an extremely high estimate of 12.0 (4.30 – 32.0)

In this table, fixed effects meta-analysis assumes all the individual study estimates derive from a common mean, with their contribution to the overall estimate depending only on within-study variability, with large studies carrying more weight than small ones. Random effects meta-analysis assumes that the individual study estimates derive from a distribution of effects, with the weighting of the individual estimates depending both on the within-study and between-study variability.

Here at table 7, subdivision of the 62 studies of smoking by the husband, produces the following:

TABLE 7
Meta-analysis: Lung Cancer.
Of studies of smoking by the husband, by publication date

Studies published	Estimates Combined	Fixed Effects		Random Effects	
		RR	95% CI	RR	95% CI
1981 – 1989	25	1.38	1.23 – 1.55	1.38	1.23 – 1.55
1990 – 2003	37	1.11	1.04 – 1.18	1.16	1.04 – 1.28

In other words, the combined results of the spousal studies vary over time, with the association being significantly weaker in studies published since 1989 than in those published in the 1980s; they also vary by region, study size, study quality and by the type of control group used (with no significant association evident in those studies using healthy population controls).

Heart disease

Table 8 lists for heart disease the studies that have been undertaken among lifelong non-smokers reportedly exposed to ETS in the workplace. There have been around 30 studies of heart disease and ETS among life-long non-smokers. Given the wide variety of study designs and data, and the fact that only a limited number provide data relating to exposure to ETS in the workplace, the listing here is confined to those that cover ETS exposure in the workplace. However, some general comments are appropriate:

Most estimates for spousal smoking are not statistically significant and there are a number of important reasons why the findings should not be interpreted as indicating a causal effect of ETS exposure.

For example, the reported results vary markedly with study size. Many of the studies fail to consider possible lifestyle confounding factors, important because there are over 300 different risk factors reported for heart disease⁶⁴ and several studies have shown differences in many life-style factors between smoking and non-smoking households⁶⁵. The studies generally rely on reported, rather than objectively measured ETS exposure data. The only two studies to use serum cotinine as a marker of ETS exposure found no significant relationship between this marker and the risk of heart disease. Some studies

⁶⁴ Hopkins PN & Williams RR. Identification and relative weight of cardiovascular risk factors. *Cardiological Clinology* 1986; **4**: 3-31

⁶⁵ Sidney S, Caan BJ, Friedman GD. Dietary intake of carotene in non-smokers with and without passive smoking at home. *American Journal of Epidemiology* 1989; **129**: 1305-9; Thompson DH, Warburton DM. Life-style differences between smokers, ex-smokers and non-smokers, and implications for their health. *Psychological Health* 1992; **7**: 311-321; Thornton A, Lee P & Fry. Differences between smokers, ex-smokers, passive smokers and non-smokers. *Journal of Clinical Epidemiology* 1994; **47**: 1143-1162; Cress HD, Holly EA, Ahn DK, Kristiansen JJ & Aston DA. Contraceptive use among women smokers and non-smokers in the San Francisco Bay area. *Preventive Medicine* 1994; **23**: 181-189; Subar AF, Harlan LC & Mattson ME. Food and nutrient intake differences between smokers and non-smokers in the US. *American Journal of Public Health* 1990; **80**: 1323-9; Le Marchand L, Wilkens LR, Hankin JH & Haley NJ. Dietary patterns of female non-smokers with and without exposure to environmental tobacco smoke. *Cancer Causes Control* 1991; **2**: 11-6; Matanoski G, Kanchanaraksa S, Lantry D & Chang Y. Characteristics of non-smoking women in NHANES I and II epidemiologic follow-up study with exposure to spouses who smoke. *American Journal of Epidemiology* 1995; **142**: 149-57.

have relied on unvalidated reports by the subject of current or past heart disease, with no confirmation of the diagnosis. In some studies misclassification is also evident.

Two of the most substantial pools of available data are the databases of the American Cancer Society's Cancer Prevention Study and the database of the US National Mortality Follow-back Survey. Analyses of these data sets have reported no overall association between ETS and heart disease⁶⁶. A further large study of ETS and heart disease was published in 2003⁶⁷ and also showed no increase in risk.

A report of the US Surgeon General⁶⁸ noted "because smoking is but one of the many risk factors in the aetiology of heart disease, quantifying the precise relationship between ETS and this disease is difficult".

Table 8 lists the studies that have provided estimates of the relative risk of heart disease among lifelong non-smokers in relation to exposure to ETS in the workplace. Of the 12 studies, only 1 has reported a statistically significant association (study no. 28).

TABLE 8
Relative Risk of heart disease among lifelong non-smokers reportedly exposed to ETS in the workplace

Ref	Author	Publication	Location	Sex	Relative Risk	Confidence Interval at 95%
3	Lee	1986	UK	M	0.66	0.26 - 1.66
				F	0.69	0.26 - 1.87
5	Svendson	1987	USA	M	1.40	0.80 - 2.50
9	Jackson	1989	New Zealand	M	1.80	0.94 - 3.46
				F	1.55	0.48 - 5.03
12	Dobson	1991	Australia	M	0.95	0.51 - 1.78
				F	0.66	0.17 - 2.62
17	Muscat	1995	USA	M	1.20	0.60 - 2.20
				F	1.00	0.40 - 2.50
19	Steenland	1996	USA	M	1.03	0.89 - 1.19
				F	1.06	0.84 - 1.34
21	Kawachi	1997	USA	F	1.68	0.81 - 3.47
24	Spencer	1999	Australia	M	No RR, but no significant association	
25b	He	2000	China	F	1.85	0.86 - 4.00
27	Rosenlund	2001	Sweden	M	1.14	0.78 - 1.67
				F	0.94	0.59 - 1.50
28	Pitsavos	2002	Greece	M+F	1.97	1.16 - 3.34
29	Chen	2003	USA	M+F	1.70	0.90 - 3.20

Note

In study no. 21, the estimates were given by study No 32.

Table 9 shows the results of meta-analyses of the studies on ETS and heart disease, again using the alternative approaches of fixed and random effects analysis. It is notable that the RRs from the two largest US studies published in 1995

⁶⁶ LeVois M and Layard M. Publication bias in the environmental tobacco smoke/coronary heart disease epidemiologic literature, *Regulatory Toxicology and Pharmacology* 1995;**21**:184-191

⁶⁷ Enstrom J & Kabat G. Environmental Tobacco Smoke and tobacco related mortality in a prospective study in California, 1960-1998, *BMJ* 2003;**326**: 1057-1061

⁶⁸ Reducing tobacco use, a report of the US Surgeon General, *US Dept. of Health and Human Services, Public Health Service, Office of Smoking and Health*

and 2003⁶⁹, were very close to 1.00 in each sex, and not statistically significant. These two studies provided data on a total of over 20,000 heart disease cases, greater than the total number of cases in all the other studies combined.

Whilst the overall RR estimates for spousal smoking are statistically significant, in that they are greater than 1.00, they are so by only a very small margin. They are also based on heterogeneous estimates that are substantially higher in small than in large studies. Also many of the studies failed to control adequately for confounding or the various other sources of bias present in such epidemiological studies, with none adjusting for misclassification of smoking habits.

TABLE 9
Meta-analysis: Heart disease

Studies	Estimates Combined	Fixed Effects		Random Effects	
		RR	95% CI	RR	95%CI
Spouse ever smoked	42	1.07	1.04 – 1.09	1.14	1.07 – 1.20
Spouse current smoker	42	1.08	1.05 – 1.11	1.16	1.09 – 1.23
Workplace exposure	17	1.11	1.01 – 1.23	1.13	1.01 – 1.27

Notes

In this table, 'spouse ever smoked' is used where a study also provides data for 'spouse current smoker', and estimates for 'spouse current smoker' are used where a study also provides data for 'spouse ever smoked'.

CONCLUSIONS ON THE EPIDEMIOLOGICAL EVIDENCE

The following conclusions can be drawn from the above analysis of individual studies and reviews by way of meta-analysis.

The reported findings of individual ETS epidemiological studies are inconsistent. Where an elevated level of relative risk has been reported, it is of a very low order, generally well below 2.00, and could be accounted for by bias or confounding for which there has not been any or inadequate statistical adjustment. In any event, however, in the vast majority of studies the findings do not meet the crucial test of statistical significance. The balance of evidence taken as whole does not demonstrate a causal link between ETS and serious diseases.

For meta-analysis to be a valid and reliable approach, studies need to be similar and comparable in design and many other respects. They are generally not and meta-analysis techniques reliant as they are on sophisticated statistical manipulation therefore add considerably to the artificiality of their findings and *“their semblance of infallibility should be rejected”*⁷⁰. A statistically significant result from meta-analysis of epidemiological data does not necessarily establish that any underlying effect exists.

WHY THEN IS IT THAT THE MESSAGE THAT THE PUBLIC HAS BEEN GIVEN BY THE AUTHORITIES DIFFERS FROM THE SCIENCE AND EPIDEMIOLOGY

“Because people do not have time to research complex issues themselves, they rely on those they trust: scientists and doctors. If television or newspapers pick up the experts’ opinions and add colour or drama, they can turn them into full-blown scandals. It is not only that the public assumes the experts must be right. A consensus develops among

⁶⁹ LeVois ME, Layard MW. Publication bias in the environmental tobacco smoke/coronary heart disease epidemiologic literature. *Regulatory Toxicology & Pharmacology* 1995; **21**: 184-191 and Enstrom JE, Kabat GC. Environmental tobacco smoke and tobacco related mortality in a prospective study of Californians, 1960-98 [abridged version]. *BMJ* 2003; **326**:1057-1061. Full version available at <http://bmj.com/cgi/content/full/326/7398/1057>

⁷⁰ Crombie IK. The Pocket Guide to Critical Appraisal, *BMJ Publishing*, 1996

the experts themselves. Once a large number believe something, it can be costly to argue the contrary. Academics who go against the grain can struggle to find posts or research funds. Europe's activists are particularly powerful. Many European campaigning organisations enjoy more credibility than scientists, and certainly more than governments. They can often win the public over even when science is not on their side...⁷¹

"Critics complain that policy is too often driven by public opinion that has been inflamed by sensationalist news headlines that distort science and exaggerate risk. Scientists themselves are sometimes assailed for manipulating science and the media to promote their own personal political agendas, and interest groups on all sides of these issues increasingly roll out battling experts who appear to agree on little, other than that their opponent's science is fundamentally flawed, misleading, biased or worse. These allegations raise serious questions about the role of science in informing public policy and the role of the media in covering scientific issues, particularly in contentious public policy areas. How the public learns about scientific news through the media, how public opinion is reflected and shaped by media coverage, and how pressure groups influence media coverage of science for their own political ends, are all important aspects of the policy-making process."⁷²

There could not be more apt explanations than those which the above quotations provide that so well describes how the public have come to believe that ETS is a serious risk to the health of the non-smoker. They raise extremely important questions about the role that science and the authorities play in informing public policy, and the role of the media in covering scientific issues.

On ETS, the root of the problem goes back some 30 years or so. In the US Surgeon General's reports of 1972 and 1975, initial speculations were raised about the possible consequences of exposure to ETS. The US Surgeon General's 1979 report noted several adverse outcomes that appeared to have an association with ETS; but also that there was only a limited amount of systematic information available regarding the health effects of ETS. The Surgeon General's 1982 report raised the concern that ETS might cause lung cancer. Following that report a number of epidemiological investigations were published which claimed to show a relationship between ETS and lung cancer.

Then in 1986, the US Surgeon General's report, as well as reviews by the National Research Council and National Academy of Science (sponsored by the US Environmental Protection Agency (EPA)), concluded that ETS caused lung cancer and claimed an increase in risk of 30%, with the latter two reviews also associating ETS exposure with adverse respiratory outcomes in young children.

However, a review published in 1986 by the International Agency for Research on Cancer (IARC) of the World Health Organisation came to different conclusions. It did not produce estimates of risk but concluded that available studies:

"had to contend with substantial difficulties in determination of passive exposure to tobacco smoke and to other possible risk factors. The resulting errors could arguably have artefactually depressed or raised estimates of risk, and, as a consequence, each is compatible either with an increase or with an absence of risk."⁷³

Nonetheless, in June 1989, the US EPA issued a public notice that stated categorically that ETS "is a known cause of lung cancer". However, the EPA did not provide an analysis of the data on which it had based its conclusion. It was pressed to do so but did

⁷¹ Michael Skapinker, management editor, Strategies to avoid being ambushed by public opinion., reporting on Corporate Political Strategies for Widely Salient Issues, Profs. Bonardi & Keim, Academy of Management Review 2004, *The Financial Times*, March 10 2004

⁷² When media, science and public policy collide, Proceedings of a workshop of the Kennedy School of Government at Harvard University, November 2002 (Words written in the context of genetically modified foods), *Harvard University*

⁷³ IARC, 1986: p.308

not produce its analysis and risk assessment until 1992⁷⁴. This took the form of a review of selected published studies. It was subjected to devastating criticism, not least by members of the US Congressional Research Service appearing before a Committee of the US Senate, who said:

“The EPA study analysed and summarised 30 studies of passive smoking lung cancer effects. Critics have questioned how a passive smoking effect can be discerned from a group of 30 studies of which 6 found a statistically significant (but small) effect, 24 found no statistically significant effect, and 6 of the 24 found a passive smoking effect opposite to the expected relationship.”

“... our evaluation was that the statistical evidence does not appear to support a conclusion that there are substantial health effects of passive smoking.”⁷⁵

The report was later also challenged in the courts⁷⁶ where the EPA was found to have knowingly, wilfully and aggressively disseminated false information with far reaching regulatory implications in the US and worldwide. Judge Osteen found that the EPA had :

“changed its methodology to find a statistically significant association . . . In conducting the ETS Risk Assessment, EPA disregarded information and made findings on selective information; did not disseminate significant epidemiologic information; deviated from its Risk Assessment Guidelines; failed to disclose important findings and reasoning; and left significant questions without answers . . . Gathering all relevant information, researching, and disseminating findings were subordinate to EPA’s demonstrating ETS a Group A carcinogen.”

Yet to this day, despite that judgement which vacated (annulled) the report after ‘forensic’ investigation of the EPA’s review and process, the report is used as a ‘gold standard’ by the authorities. It is the ultimate foundation of the estimates made by UK authorities of UK deaths resulting from exposure to ETS. The report and its methods have subsequently been used as a model for other reports by the Californian EPA⁷⁷, the National Health & Medical Research Council of Australia⁷⁸, and the UK’s Scientific Committee on Tobacco and Health (SCOTH)⁷⁹. In 1998, the US National Toxicology Program accepted the EPA 1992 report and its twin from California as the basis for listing ETS as a known human carcinogen.

Since then, more ETS epidemiological studies have been published, but they have not substantially added to the body of knowledge on ETS and it remains a fact that it has not been proven that ETS causes disease in non-smokers. The idea that it does is no more than what IARC describes as *“the current concept”*⁸⁰.

That is not, however, what the public has been told by the authorities, nor by the media and press.

Scientific and statistical studies are generally replete with ‘ifs, buts and maybes’, unfamiliar terminology and jargon, complex formulae and calculations. They do not generally make news that demands loud, clear and unequivocal headlines and sound bites. But if that kind of message is not provided by the research itself, by the professional journals publishing their work and wanting to promote their own publications, or by the authorities to whom the public and government looks for advice, the danger is that it can then be generated by the spinning of headlines and reporting

⁷⁴ Respiratory health effects of passive smoking: lung cancer and other disorders, EPA, Washington DC, 1992

⁷⁵ Oral statement of Dr Jane Gravelle & Dr Dennis Zimmerman of the Congressional Research service, *The Library of Congress, WashingtonDC*, May 11 1994

⁷⁶ Flue-cured Tobacco Stabilization Corporation *et al v* United States Environmental Protection Agency and Carol Browner, *District Court for the Middle District of North Carolina before District Judge Osteen, Order and Judgement*, 17 July 1998

⁷⁷ Californian EPA 1997

⁷⁸ NHMRC 1998

⁷⁹ SCOTH 1998

⁸⁰ WHO/IARC Monographs Vol 83, p1383

that lacks thoroughness and concern for detail and accuracy. Over the past 24 months in particular, there have been numerous examples of grossly inaccurate reporting of ETS studies.

The results of other epidemiological studies

Despite all the conventional guides on levels of RR explained earlier, and particularly as they relate to findings of relative risks lower than 3.0, in the case of ETS, quite different interpretations have been promoted.

A 1997 meta-analysis⁸¹ accepted by the UK authorities found a RR of 1.26 (95% CI 1.06 – 1.47), derived amongst non-smokers living with smoking spouses. That was claimed to be a "substantial" excess risk and one warranting bans on smoking in work and public places.

IARC's 1998 report⁸² was a case-control study of lung cancer and exposure to ETS in 12 centres from 7 European countries that the researchers claimed provided "the most precise available estimate of the effect of ETS on lung cancer risk in Western European populations." It reported no overall statistically significant increase in risk of lung cancer from ETS in any of the situations where people were exposed to ETS. The conclusions of the study stated:

"Our results indicate no association between childhood exposure to ETS and lung cancer risk (0.78 (95% CI 0.64-0.96)). We did find weak evidence of a dose-response relationship between risk of lung cancer and exposure to spousal (1.16 (95% CI 0.93-1.44)) and workplace ETS (1.17 (95% CI 0.94-1.45)). There was no detectable risk after cessation of exposure."

In other words, not only were relative risks found to be low, but at the 95% CI they included 1.0, indicating that they were not statistically significant (pure chance was at play). The following observation was also made in the report:

"The available literature on ETS exposure from the spouse and lung cancer is large. However, only six studies are available from Europe; two of them, conducted in Greece, showed a twofold increase in risk for women ever married to a smoker. Of the other studies, one from Scotland provided very unstable risk estimates of the same magnitude as the Greek studies and two – one from the UK and the other from Sweden – provided little evidence of an association."

The results were within the range at which the IARC itself concluded that unequivocal results may be forever unachievable. Yet after negative reporting of the results by the media, IARC insisted that the findings "add substantially" to previous evidence of the risk between ETS and lung cancer. A WHO press release then implied that the results proved a link between ETS and lung cancer, a highly problematic conclusion given their own guidelines of epidemiological best practice⁸³.

It is difficult to see how it could be claimed that the study added substantially to the case against ETS and much less that it proved a link between ETS and lung cancer. The interpretation of such weak evidence is not in line with the official interpretation of very similar findings of other supposed health risks.

It is even more difficult to discern the reasons for IARC in 2002 (report published 2004), without the benefit of any substantial additional evidence, forming the conclusion, largely on the basis of the epidemiological evidence available, that ETS should be declared a Group 1 carcinogen.

⁸¹ Hackshaw A K, Law M R, Wald N J. The accumulated evidence on lung cancer and environmental tobacco smoke. *BMJ* 1997; **315**:980-988

⁸² Boffetta P *et al.* Multi-centre case-control study of exposure to environmental tobacco smoke and lung cancer in Europe, *Journal of the National Cancer Institute* 1998;**90**: 1440-1450

⁸³ Isabel dos Santos Silva. Cancer epidemiology: Principles and methods, *IARC* 1999

What is absent is an explanation as to why the low RRs that have been reported in respect of lung cancer and ETS - with 95% CIs often including 1.0 and any excess risk capable of being accounted for by only modest degrees of bias and confounding, or by inadequate statistical adjustment for such factors - are regarded by some as providing incontrovertible proof of a causal link.

A major study⁸⁴ of the supposed link between electric power lines and childhood leukaemias produced a RR of 1.24, with a non-significant 95% CI of 0.86 - 1.79. The researchers concluded that this provided "little evidence" of a link between power lines and leukaemia. The US National Cancer Institute went further, declaring that the study showed magnetic fields "do not raise children's leukaemia risk".

Another study⁸⁵ of women with breast implants found a RR for hospitalisation for connective tissue disorders of 1.3 with a non-significant 95% CI of (0.7 – 2.2), again close to the IARC passive smoking study. But whereas the IARC findings were claimed to prove a link between ETS and lung cancer, in the breast implant study they were found not to be associated "with a meaningful excess risk of connective tissue disorder"⁸⁶.

⁸⁴ Linet M, *et al.* Residential exposure to magnetic fields and acute lymphoblast leukaemia in children. *New England Journal of Medicine*; 1997

⁸⁵ Nyren O *et al.* Risk of connective tissue disease and related disorders among women with breast implants: a nationwide retrospective cohort study in Sweden. *BMJ* 1998;**316**:417

⁸⁶ Cooper C, Dennison E. Do silicone breast implants cause connective tissue disorder? *BMJ*;1998;**316**:403

Smoking in work and other places

In 1988⁸⁷, the Health & Safety Executive published guidance for employers explaining what they should do to comply with health, safety and welfare law as it applies to passive smoking (ETS). Many employers subsequently introduced smoking policies, after consultation with employees or their representatives.

In 2003⁸⁸, 50% of people reported their workplace as being one in which smoking was banned (10% higher than in 1996); 38% reported that they worked in places where smoking was permitted only in designated places. 4% worked alone. That left only 8% of people, down from 13% in 1996, working in places where there were not formal restrictions on smoking - that represents around 2 million people⁸⁹, down from 3.4 million in 1996.

Those data represent a massive endorsement of the appropriateness of voluntary self-regulation of smoking in the work place, put in place by employers after consultation and agreement with employees and their representatives.

Given that the absence of formal rules on smoking applies now only to 8% of the working population – and that the percentage is falling year by year – it is astounding that there should even be debate, let alone promotion of the idea that there should be legislation banning smoking in the workplace.

In the main, the debate has focused on the hospitality sector – such places as hotels, pubs, bars, clubs, restaurants. They are public places only in the sense that they are places which the public may visit or not, as they wish. If someone does not like the goods or services, or the environment of the place, or its other customers, they may and generally do take their business elsewhere. Essentially, however, the places are privately owned and operated, no different to shops, supermarkets or any other business that is privately owned or operated, in respect of which the owner or operator rightly determines the smoking policy. In doing so, he should, as the law determines, ensure, so far as is reasonably practicable, the health, safety and welfare of his employees. Also, it is in the owner's or operator's best commercial interests to adopt such policies as best suit his customers and clientele.

When, in 1999, the Health & Safety Commission published its consultation paper⁹⁰ on a possible code of conduct on smoking policies, it had much to say that remains relevant today, about workplaces generally and about workplaces that were also public places, particularly such places in the hospitality sector:

On the risk to health of ETS in the work place and public places

"Proving beyond reasonable doubt that passive smoking at a particular workplace was a risk to health is likely to be very difficult, given the state of the scientific evidence:" (para 11)

"[However] we do not know how big the risk is of developing diseases such as cancer or ischaemic heart disease from passive smoking at work." (para.26)

"...information about the magnitude and extent of the risk to the public from passive smoking in public places is even more uncertain . The issue is better dealt with in the other ways discussed in the White Paper⁹¹." (para. 29)

"Employers should not simply ignore their duty to ensure the welfare of employees who smoke ... but... where the welfare needs of employees who smoke and employees who do not smoke come into conflict, priority should be given to the needs of employees who do not smoke." (para. 28)

⁸⁷ Passive smoking at work, *Health & Safety Executive*, November 1988

⁸⁸ Smoking Related Behaviour and Attitudes, 2003, *Office for National Statistics*, July 2004

⁸⁹ Annual Abstract of Statistics, Labour Force Survey 2002, *Office for National Statistics*, 2003

⁹⁰ Proposal for an Approved Code of Practice on passive smoking at work, *Health & Safety Commission, Health Directorate, Division A*, 1999

⁹¹ Smoking Kills: A White Paper on Tobacco, *Department of Health*, December 1998

On the adequacy of existing law

"We believe there is already sufficient health, safety and welfare law that can be applied to the question of passive smoking". (para.15)

"...tobacco smoke causes discomfort and irritation. These are matters of employee welfare, rather than their health. A provision in existing health, safety and welfare law⁹² already requires employers to protect employees who do not smoke from the discomfort caused by tobacco smoke in rest areas and rest rooms." (para.27)

". . . regulation 6 of the Workplace (Health, Safety and Welfare) Regulations 1992 requires employers at enclosed workplaces to which those Regulations apply, to ventilate workplaces with a sufficient quantity of fresh or purified air. Similar provisions apply in other workplaces." (para. 79)

On the inappropriateness of prescriptive regulations

"We cannot be specific about what people should do to control passive smoking in every workplace, so prescriptive health and safety regulations about passive smoking could well be unreasonable and unenforceable. This would especially be the case in certain sectors where the workplace is also a public place (for example pubs and restaurants) or provides accommodation for off-shift workers (for example offshore installations)." (para.14)

Practicable solutions

"In our view, it is not currently reasonably practicable under health, safety and welfare law to ban smoking in all such [restaurants, pubs, residential care homes] workplaces: in some cases, because it would not be commercially viable, and in others because it would interfere with personal freedoms." (para. 36)

"In some ways the charter [Public Places Charter] goes beyond what could be required under health, safety and welfare law for the protection of workers . . . So if an employer follows the charter's principles, they should find it easy to comply with health, safety and welfare law." (para. 25)

In March 2000, the Health and Safety Executive published a revised draft ACoP, which offered the following guidance

"If your business is in the hospitality sector it might not be reasonably practicable to ban smoking completely in areas where employees need to work because you wish to cater for customers who choose to smoke. . . The hospitality industry's Public Places Charter⁹³ aims to encourage better provision for non-smoking customers in places such as pubs, restaurants and hotels." (para.58)

"Decisions on banning smoking by customers or clients will have to be made on a case-by-case basis. However, it will usually be reasonably practicable under health, safety and welfare law to ban completely customers or clients smoking for reasons of employee welfare in places where the public must visit, but where they normally remain only for a short time – for example, in: libraries, bus and railway station ticket offices and buffets, shops, supermarkets, Government (including Local Government) buildings, banks, building societies and post offices. This list is not exhaustive." (para 60)

"Currently, it may not be reasonably practicable to ban completely customers or clients smoking where:

- a) the public must visit, but where they remain for some time – for example, airport departure lounges; or,*
- b) the public visit out of choice – for example, restaurants, cafes, public houses, bars, clubs, hotels, casinos and betting shops; or*
- c) people live – for example, the accommodation on an offshore installation, residential care homes, and prisons." (para. 61)*

⁹² Workplace (Health, Safety and Welfare) Regulations 1992, Regulation 25(3).

⁹³ Smoking Kills: A White Paper on Tobacco, *Department of Health*, December 1998, p.69

Given these facts, in 1999 and 2000, the Health & Safety Commission and Executive suggested that one way of improving the situation might be for an Approved Code of Practice (ACoP) to be adopted as guidance for employers on smoking policies in the workplace. Its consultation paper served to highlight the practical difficulties involved in providing such guidance and the need to make decisions on a case-by-case basis according to the circumstances. It estimated that there were then around 63,000 organisations and 223,000 units in the hospitality sector.

If that was the case in 1999, it is appropriate to consider what has since changed, if anything, that might prompt a change in policy. As far as the Government at Westminster is concerned, it has to date consistently stood firm in its belief in the voluntary partnership approach provided that it makes substantial progress:

“The Government have no plans to ban smoking in public places. We have consistently said that smoke-free public places are the ideal. We do not think that a universal ban on smoking in all public places is justified while we can make fast and substantial progress in partnership with industry...We look forward to faster and more substantial progress from employers and businesses.”⁹⁴

In 1998, the Government agreed with representative bodies within the hospitality sector a Public Places Charter which sought, by encouraging the adoption of formal smoking policies, best practice and the provision of more non-smoking areas, to provide customers with better choice. Virtually all the agreed targets for the five years ahead were met or well exceeded.

The Scottish Executive laid out its current policy on ETS in 2004⁹⁵:

“Current Activity

5.4 *A central plank of [the Executive’s] policy has been an industry initiative, the “Scottish Voluntary Charter on Smoking in Public Places”, to drive improvements in non-smoking facilities in the licenced, tourism and hospitality sector by encouraging venue operators to set a formal smoking policy and to highlight this through external signage.*

5.5 *Supported by the four main industry bodies – the Scottish Beer and Pub Association, the Scottish Licensed Trade Association, the Scottish Tourism Forum and the British Hospitality Association – the Charter was launched in May 2000. Targets were set to achieve - by the end of 2002 – 10% increases in the number of places with smoking policies, written smoking policies, signs indicating smoking policy near entrances, and non-smoking provision.*

5.6 *An independent evaluation, published on 23 September 2003 [Smoking in Public Places: A follow-up survey of the Scottish Leisure Industry: prepared for ASH Scotland/Health Scotland by MVA] of smoking policies in the Leisure Industry would suggest that the industry had met three out of the four targets set under the Charter, including the key target of availability of smoke-free provision. We welcome the progress made under the Charter and believe that it demonstrates the progress which can be made through partnership with the business community in this most challenging of sectors.*

Way Forward

5.9 *...Much progress has been made in smoke-free environments in public places in Scotland through voluntary action...*

5.10 *...in our view statutory controls can only be truly effective – and ultimately enforceable – if they take place in an environment in which the legislation reflects rather than attempts to force public opinion on what remains essentially an issue of personal behaviour...*

⁹⁴ Lord Warner, Written Answer, House of Lords, *Official Report*, Col.WA177, March 10, 2004

⁹⁵ A Breath of Fresh Air for Scotland – Improving Scotland’s Health: The Challenge Tobacco Action Plan, *Scottish Executive*, Edinburgh, 2004

In the Spring of 2004, with a view to achieving more rapid progress, UK Ministers discussed with the hospitality sector the possible directions for the way forward over the next few years. Similar discussions are ongoing with the Scottish Charter members and the Scottish Executive.

Progress has significantly quickened in pace over the past twelve months during which a significant number of businesses have reviewed their smoking policies, some introducing bans, others measures to segregate smokers from non-smokers and/or improve air quality generally by ventilation etc. Such developments have been voluntary and driven as they should be by what customers want, not what an arbitrary law has determined.

However, this progress has been overshadowed by the strength of the voice of anti-smoking activists who, following the ban on tobacco advertising and promotion in the UK and the prohibition of smoking in the workplace in New York and Ireland, have vigorously campaigned for the imposition of the same prohibitions on smoking in the UK.

Campaign themes have changed and a brief examination of some of the remarks made in the introductory statement of the Chief Executive of ASH in the recently published 'toolkit' guide for local decision-makers⁹⁶ gives a flavour of the rhetoric that is now typical:

"Secondhand smoke is a killer.... the science on the point is quite clear."

Not true. The evidence – of science and epidemiology – is not strong, consistent or conclusive... it is far from clear.

"Yet at least three million employees are still regularly exposed to secondhand smoke in the workplace."

Not true. There are about 2 million people (as stated in the ASH accompanying press release but contradicted in the toolkit document that is based on outdated data) that work in places where there are no formal rules on smoking. This does not mean that these people are regularly exposed to ETS.

"Most restaurant chains and almost all pubs continue to permit smoking in all or part of their premises."

Untrue and misleading. Many restaurants and chains of restaurants have strict smoking policies, many prohibiting smoking. Many pubs continue to permit smoking, but many confine smoking to segregated areas. An increasing number of pubs are also becoming no smoking throughout.

"Attempts to encourage voluntary bans have largely failed."

Not true. The independently monitored results of the Public Places Charter confirm that the major targets were achieved. It was not an object or target of the Charter to achieve bans but to introduce formal, clearer smoking policies and to make greater provision for non-smokers. There would appear to be every prospect that the progress achieved to date can continue to be built upon significantly over the next few years.

"Professor Konrad Jamrozik of Imperial College London has recently estimated that one worker in the hospitality industry dies every week from the effects of secondhand smoke, and that 700 employees in all industries die every year."

Professor Jamrozik's estimates are based on the manipulation of epidemiological findings that are seriously flawed and largely not UK-based, and even more significantly on wholly incorrect UK employment data. Even using his own methodology, his estimates are wrong by a very wide margin.

"Stopping smoking in the workplace, as in New York, Ireland, Norway and elsewhere is a successful and popular policy."

The recent bans introduced in New York and Ireland have so far been largely respected by the public and it remains to be seen if this will continue to be the case. More significantly, however, the bans have not been a success in economic terms. The economic data frequently quoted for New York does not include any necessary adjustment to take account of the general New York

⁹⁶ Achieving Smoke Freedom Toolkit: A Guide for Local Decision Makers, ASH/Chartered Institute of Environmental Health, 3 September 2004.

economic upturn since “9/11”. When it is, significantly lower trading and job losses are indicated. The economic, trading and employment data with regard to Ireland is only just emerging but is showing substantial downturns.

“... most smokers want to give up.”

The personal decision to smoke is one that a smoker may change at any time, as a very great many do. In the UK in 2002, 26% of persons over 16 smoked; 54% had never smoked; 21% reported that they were ex-smokers⁹⁷. In Scotland the figures indicated that 28% of adults smoke.⁹⁸

Of those smokers who give answers in surveys to the effect that they would like to give up smoking – 68%⁹⁹ – many will. That millions have done so already is testimony.

It is also the case, however, that an answer given by a smoker to the effect that they would ‘like to quit’ does not convey a commitment to quit. It is more likely to be an answer prompted by the context and nature of the questioning.

The fact is that any smoker who decides and is determined to quit smoking can do so. Some find it to be more difficult than others, but if smokers are constantly given to believe that it will be difficult and that they might not be able to cope without support, it becomes more likely that many will not even make the attempt.

These remarks of the CEO of ASH also confirm what has now openly become the principal ground advanced by anti-smoking activists for the prohibition of smoking in work and other places – reducing the smoker’s opportunity to smoke so as to force the smoker into quitting. If that were to be adopted as a justification for a public policy of prohibition, it would represent an alarming denial of individual freedom, and set a most dangerous precedent.

Smoking is not illegal, but smokers do not have a right to smoke anywhere they please, and they do not do so. Most smokers are respectful of other peoples’ preferences and views, and respect smoking rules. There is no need or justification for intolerant, socially divisive prohibition. No freedoms or rights need be lost in a society that is tolerant and sensible.

Current legislative proposals in the UK and Scottish Parliaments

The Prohibition of Smoking in Regulated Areas (Scotland) Bill, a Member’s Bill¹⁰⁰, is currently being considered in the Scottish Parliament. The Tobacco Smoking (Public Places and Workplaces) Bill [HL], a Private Member’s Bill¹⁰¹, is currently being considered in the House of Lords in relation to England and Wales. In addition, a Private Member’s Bill, currently in the House of Commons, seeks to provide the National Assembly of Wales with powers to regulate smoking in public places.

The main purpose of the Bill introduced in the Scottish Parliament is to prevent people smoking in certain public areas. The Bill prohibits smoking in public places where food is supplied and consumed but with provisions such as would enable extension of the meaning of a regulated place. The Bill does this by: defining areas where smoking is not permitted (regulated areas); making it an offence to smoke in regulated areas; making it an offence for owners, occupiers and the like to knowingly permit smoking in regulated areas; requiring signs to be clearly displayed inside and outside regulated areas; and providing that offences can be prosecuted summarily. The Bill provides that these offences would be subject to a fine not exceeding level 3 on the standard scale (£1,000).

The Bill before the House of Lords imposes a prohibition on smoking in all enclosed work and public places (other than a few exempt places). It enables certain enclosed spaces to be designated smoking spaces (but not the whole of any place), subject to each space

⁹⁷ General Household Survey: Living in Britain, 2002, Tables 8.2 and 8.3, *Office for National Statistics*, 2004

⁹⁸ *Ibid*, Table 8.6

⁹⁹ *Ibid*

¹⁰⁰ Introduced in the Scottish Parliament by Stewart Maxwell on February 3, 2004

¹⁰¹ Introduced in the House of Lords by Lord Faulkner on March 10, 2004

complying with regulations to be made by Ministers. The Bill creates two new criminal offences – smoking in an enclosed public place, other than in a designated smoking area complying with regulations, and failure of an occupier of any such place to take reasonable steps to ensure compliance, each offence being subject to a fine not exceeding level 5 on the standard scale (£2,500), significantly higher than the level applying under the Bill in the Scottish Parliament.

Under the Bill “a suitable and sufficient proportion” of a place could be a “designated smoking area” provided that there was compliance with regulations made by Ministers. Those regulations could determine the maximum size as a proportion of the total capacity; require consultation with employees; set maximum permitted exposure levels to ETS or any of its constituent ingredients, for any employee or member of the public; make provisions for signs; regulate the design of ashtrays; and make regulations governing the provision, design and operation of ventilation and air purification equipment.

Both Bills would render void the current voluntary approaches to the regulation of smoking in workplaces and would replace them with the need to comply with bans and regulations, for no good, sensible or justifiable purpose. Worse still, the provisions of the House of Lords Bill, are similar, yet not directly analogous to the provisions of the Bill introduced before the Scottish Parliament, which illustrates how confused the situation might become if both Bills were to be enacted.

Neither the Bill at Westminster nor that in the Scottish Parliament defines a public place other than by way of listing in its Schedules places which are included within that term. The Schedules to each Bill are similar but not identical and this inconsistency across the UK would lead to uncertainty and confusion especially for the many chains that operate on a UK wide basis.

Most importantly, the Bill in the House of Lords also makes significant provisions with regard to employment law. Amongst other things, it radically changes the law as it currently stands with regard to contracts of employment; extends the concept of “automatic unfair dismissal”; and makes provisions that are out of step with current law in that they extend rights on a quasi-public health basis, but to employees only, not to other workers or independent contractors. The latter provisions would be likely to result in services having to be provided in smoking environments through the use of non-employee workers and other contractors, presumably the opposite of what the Bill intends.

Local decisions

Various city and other councils in Britain have made known their wish for their city, town or region to become smoke-free. The proposition that local authorities should be able to regulate or ban smoking in work and other places, by introducing laws to apply within their area, may have some political appeal at Westminster or Holyrood. It might, even if only in part, remove the issues from the desks of ministers. It would also enable the claim to be made that decisions were being delegated to local councils, communities and democracy. However, such a proposition implies that ETS issues have an overriding local dimension and there are grounds for the costly bureaucracy that would be involved, which is simply not the case. Indeed, sweeping smoking bans are not even what the public wants.

Laws prohibiting and/or regulating smoking, whether on a national or local scale, are not justified on health or other grounds. They would needlessly be socially divisive and unnecessarily create criminal offences that would involve not insignificant costs of enforcement. The voluntary approach is not failing. It is producing what the public wants – both choice and more smoke free areas.

It would be particularly problematic if local authorities were to be enabled to introduce laws, whether by way of bye-laws or other means, prohibiting or regulating smoking in

the workplace. It would certainly not assist the legal framework of health, safety and welfare and employment legislation much of which is not devolved and continues to operate on a UK-wide basis. The probability would then arise of there being different legal provisions applying from one local authority area to another. Businesses with more than one establishment and in different local authority areas could find that there were different provisions affecting their employees, according to where they worked. What would be an offence in one place might not be in another. The costs to local government and the private sector in managing such arrangements would be disproportionate and unnecessary, particularly when perfectly acceptable voluntarily adopted policies are already in place, working without any difficulties, and can be developed further.

If local authorities were to be afforded powers, other than by way of delegated powers to make bye-laws, it would be a major constitutional departure.

Currently local authorities do not have the power to make laws without such power being delegated to them by primary legislation at Westminster. Under s.201(1) of the Local Government (Scotland) Act 1973, local councils may make bye-laws "for the good rule and government of the whole or any part of their area and for the prevention and suppression of nuisances therein." Under s.202(1)(b) of that Act parliamentary legislation may give power or impose a duty to make bye-laws which are effectively delegated legislation. Generally, bye-laws are of limited scope and are of a negative character, in that they prohibit activities, unlike Local Acts that may permit them. Also, in every case a bye-law has to be confirmed or approved by the appropriate Secretary of State (in Scotland by the Scottish Ministers) before it is valid and enforceable, not least because it creates a criminal offence.

In the case of a bye-law on smoking, different local authorities might make different decisions, some wishing to introduce a bye-law, others not. Then different smoking policies would apply in different areas. The local authority areas might adjoin one another, so creating a borderline on one side of which it would be a criminal offence to smoke in defined places, but not on the other.

Bye-laws, as they are generally provided for in legislation, and as they are promoted and brought into force, would also appear to be a most inappropriate means of regulating smoking. Firstly, the general power in s.201(1) of the Local Government (Scotland) Act 1973 arguably would not extend to making bye-laws to control smoking. Secondly, a specific power would need to be introduced by way of primary legislation delegating responsibility to local authorities in this regard. A council that wished to introduce a bye-law would have to draft the bye-law and give notice of its intention to apply for confirmation of it in one or more local newspapers, at least one month before the application for confirmation by the Scottish Ministers is made.

As a general rule-of-thumb, it would appear that the considerations taken into account when examining a bye-law are: is it *intra vires*; have all the requisite procedural actions, such as consultation with named public bodies been taken; does it duplicate or conflict with general, existing bye-laws or any local Act or common law; does the nuisance that it addresses merit criminal sanctions; to a reasonable person, is the penalty available proportionate; does the bye-law address a genuine and specific local problem, or does it attempt to deal with an essentially national issue; and does it conflict with government policy. There would currently appear to be some difficulty in providing positive answers to all these questions.

Not only would it appear that it would first be necessary for primary legislation to be introduced at Westminster or Holyrood to enable local authorities to prohibit or regulate smoking/ETS but that it would also be necessary to provide legislative guidance to local authorities to ensure a lawful and consistent approach.

The Scottish Executive's consultation on Smoking in Public Places puts forward '*giving powers to Local Authorities to regulate smoking in public places in their areas*' as an

option for future action. It does not suggest the means by which any local authority provisions which banned smoking in public places might be enforced.

In the Private Member's Bill that is currently being considered in the House of Lords¹⁰², a provision is included at clause 8 that makes it a duty of a local authority to enforce the Act and regulations made under it in respect of designated smoking areas. It then enables Orders to be made (subject to the negative procedure) "to make provision for powers to be granted to local authorities so as to enforce the Act and the regulations made under section 5 [regulations on designated smoking areas]."

With regard to that provision, the Select Committee on Delegated Powers and Regulatory Reform of the House of Lords has reported that it is "*very open-ended and would cover giving authorised persons rights of entry and seizure for example. In view of the scope of this power, we recommend that its exercise (by the Secretary of State) should be subject to the affirmative procedure.*" That should indeed be the case, but it also prompts the question why the Proposer did not include detailed provisions regarding enforcement in his Bill.

The Prohibition of Smoking in Regulated Areas (Scotland) Bill creates several offences but does not contain any enforcement provisions because the Proposer apparently does not consider them to be necessary. The normal criminal law processes would therefore apply.

More fundamental still is whether local authorities would be in a position of being able to enforce bans on smoking without undue cost to council tax and business rate payers. It would be extremely surprising also if people were to agree with the proposition – which they have not seen in that light – that smoking in the work place or in a so-called 'public place', privately owned and run, should be a criminal offence, with the penalty on conviction perhaps being imprisonment and/or a significant fine.

Alternatives to legislation

Without having resort to legislation, there are a number of ways by which local authorities, towns and cities, working with other public sector bodies and the private sector locally, could help to achieve more smoke-free public places. Legislation is not required, nor is it appropriate.

The widespread adoption of self-regulation of smoking in the workplace has been a great success, not least because it has operated through a process of consultation with and involvement of employees and their representatives in both the private and public sectors. More can be achieved by following this same direction. Two recent documents¹⁰³ indicate both the opportunities and a way forward for local authorities, simply by using their existing powers.

In particular, local councils are in a position of being able to set an example of best practice for the whole community by the way in which they manage smoking policy amongst their own employees and in their own premises, including those that are public places. That means developing an approach that is sustainable and firmly rooted in good practice; reviewing policies and the issues to which they may give rise; developing policy and engaging staff in consultation.

Best practice should also mean everyone being subject to the same policy in the same premises. For example, if the policy is one of smoking only in certain designated rooms, the same policy should apply to everyone, irrespective of their status.

¹⁰² Tobacco Smoking (Public Places and Workplaces) Bill [HL]

¹⁰³ Tobacco at work: guidelines for local authorities, *A joint publication between NHS Scotland, ASH Scotland and Convention of Scottish Local Authorities*, 2004; and Achieving Smoke Freedom Toolkit, *Chartered Institute of Environmental Health and ASH*, September 2004.

Local councils can also play a key role in engaging local businesses. There are numerous ways in which this can be done. It need not be costly. For example, the policy aims of a council can be promoted through environmental health officers who can make suggestions and give advice, particularly to businesses in the hospitality sector, when undertaking their inspections of premises.

Given the loud voice of those calling for legislation to ban smoking, the impression might be created that there are large numbers of people working in local businesses where smoking is not regulated, and that the non-smoker cannot avoid tobacco smoke in restaurants, cafes, pubs, bars and the like. Neither claim would be true. As data show, 88% of employees work in places where smoking is either banned or permitted only in designated areas; and there is increasing provision of non-smoking facilities in the hospitality sector. The public is being provided with more non-smoking areas and with choice, rightly so to reflect the fact that, whilst smokers are in a minority in the population, there are still between 26% and 30% of adults who smoke.

These achievements – from a point where the vast majority of the population smoked, and where very few places were smoke free – have all been by way of voluntarily adopted self-regulation, and gradualism which has reflected changes in society. That, the TMA believes, should also be the approach of local councils and communities for the future.

What the public thinks and wants

There is no shortage of claims as to what the public thinks and wants about smoking in work and other places. Over the last 18 months, 18 major polls have been commissioned and published by a variety of interested parties. The TMA has published an analysis of these polls which is submitted to the Scottish Executive as part of its response to the consultation on smoking in public places.

The purpose of all these public attitude surveys has been to shape public and political debate. They have all been released into and disseminated via the print, broadcast and electronic media. A growing number of media outlets have also produced and published their own polls on the issue.

The quality of the methodologies underpinning the surveys has differed considerably. The amount of information on methodology put into the public domain by commissioning and polling organisations has also been variable, making it difficult to assess the relative validity of claims made in support of different options for change.

Polling that canvasses votes or opinions – say by telephone, the web or adding one's name to a written list – does not provide a snapshot truly representative of public opinion. The people most likely to respond are those who have the strongest opinions on the issue, and their views are not necessarily representative of the population as a whole. Such polls are also readily open to manipulation, by way of the encouragement and canvassing of mass responses of a particular view. They are frequently exploited in that way, albeit not by smokers who are not an organised group.

To be able to make claims about public opinion as a whole on the basis of a restricted sample attitude survey, the sample has to be demographically representative. Omnibus polls are designed to be demographically representative from the outset; other polls have not necessarily reported their precise sampling. To prove the significance of their findings, polls also need to explicitly state confidence intervals, margins of error or statistical thresholds. If they do not, there is no statistical means of showing that the polls are an accurate representation of public opinion.

The offer of choice in questionnaires and the framing of questions are also important considerations. Many polls have asked simple black and white questions of whether smoking should be banned in public places, without allowing respondents to consider alternatives, such as designated smoking and non-smoking areas. The way that questions are asked also sets up a context and an emotional environment which can frame and shape the response given. Polls have variously started by setting the scene with a ban or restrictions.

For example, a BBC poll¹⁰⁴, variously referred to as a consultation or survey, asked people to give a simple Yes/No answer to the question: *“Should the Government ban smoking in all public places, in order to cut down on illnesses associated with smoking and passive smoking?”* Given the loaded framing of the question, it was not surprising that the majority said Yes. More recently a BBC poll¹⁰⁵ stated one of the key findings as *“67% support a smoking ban in all workplaces, including pubs and clubs, versus 32% opposed [see below for more, arguably contradictory, results on this issue]”*; below, it was stated that *“When presented with a choice between which alternative “best reflects your views on smoking in the workplace”, the most popular policy option – 44% - is a requirement to restrict smoking to designated areas. Just under a third – 32% - support a ban. 22% think it should be up to individual employers to decide in consultation with staff. 2% think there should be no restrictions.”* Contradictory? – Yes; a confused sample of people – yes. Yet even so, the headline that the BBC promoted was related to the figure of 67%.

¹⁰⁴ ‘Consultation’ carried out for “Your NHS: For Better or Worse”, *BBC One*, March 24 2004

¹⁰⁵ For the *Today Programme*, 6 September 2004

Amongst all the polls, the sheer scale of the Big “Smoke” debate may be impressive, but unfortunately it suffers most of the shortcomings mentioned above and close inspection of the “Big Smoke” polls that have been undertaken shows that they cannot possibly be regarded as being representative and fair. In effect, in terms of providing a representative picture of public opinion, they are statistically worthless.

Two surveys in particular stand out as providing reliable indications of public opinion. They were sound in representative sampling, satisfied statistical significance testing and coincidentally and without any prior knowledge of the fact, asked virtually the very same questions about the key category of pubs and bars., The surveys were undertaken by the Office for National Statistics¹⁰⁶ and by Populus for FOREST (10 polls in Scotland, NE England and 8 cities). The results were as follows:

In pubs would prefer	ONS	Populus (for Forest)	
		Scotland	Total (10 polls)
No smoking throughout	20%	22%	24%
Mainly non-smoking with smoking areas	50	51	49
Mainly smoking with non-smoking areas	19	21	19
Smoking allowed throughout	8	5	6
Don't know / don't go to pubs etc.	2	1	1
<i>Base=100%</i>	<i>1753</i>	<i>1000</i>	<i>10000</i>

When this data is considered alongside the fact – again ONS data – that only 8% of people work in places where there are not formal restrictions on smoking, and where there might not even be smokers, an explanation needs to be provided as to why legislation/ prohibition/ formal regulations/ the creation of criminal offences etc. are even being considered to be options for the way forward.

¹⁰⁶ Smoking-related Behaviour and Attitudes, 2003. *Office for National Statistics, 2004*

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