prejudice & propaganda

the truth about passive smoking

prejudice & propaganda

the truth about passive smoking

'There is a worrying trend in academic medicine which equates statistics with science, and sophistication in quantitative procedures with research excellence… Epidemiology is a main culprit, because statistical malpractice typically occurs when complex analytical techniques are combined with large data sets. The mystique of mathematics blended with the bewildering intricacies of big numbers makes a potential cocktail… Science is concerned with causes, but statistics is concerned with correlations.'

B G Charlton MD
University of Newcastle upon Tyne
Pages 112-114, Statistical Malpractice,
Journal of the Royal College of Physicians
March-April 1996

Acknowledgements:
Gian Turci

ISBN 1 871833–81–2

FOREST
Sheraton House
Castle Park
Cambridge CB3 0AX

Telephone 01223 370156
Email contact@forestonline.org

www.forestonline.org

CONTENTS

a challenge to the chief medical officer

HARDLY A WEEK is allowed to pass without some new scare story about the perils of 'passive smoking', *writes Ralph Harris [Lord Harris of High Cross]*. One of the latest, based on an experiment in an Italian garage, is that tobacco smoke is more lethal than car exhaust fumes. Another argues that 'passive smoking' is even more dangerous than direct smoking. Meanwhile the number of deaths attributed to environmental tobacco smoke (ETS) has been variously projected from 'about several hundred' – in an adult population of above 40 million! – all the way up to 10,000 or even 12,000 a year.

As a lifelong pipe-man I have increasingly come to mistrust the dogmatic vehemence with which the stop smoking (SS) brigade recycle their denunciations of 'passive smoking'. Certainly, my smoke may be irritating or even upsetting to sensitive bystanders, as are popcorn, perfume and garlic on crowded tube trains. But lethal? Despite a barrage of media publicity, most non-smokers in my experience remain unmoved by dire warnings that tobacco smoke – massively diluted in the atmosphere – could actually kill them. It is this commonsense implausibility that has goaded the tight network of anti-smoking lobbyists – ever more shrilly – to demonise ETS and brandish mounting estimates of its death toll.

But it was when the Chief Medical Officer Sir Liam Donaldson trespassed onto my territory of economics – claiming that the Irish ban on smoking in 'public places' was actually good for business because takings had increased – that I was emboldened to question also his scientific credentials. Now of course if that were true a ban, enforced by 'tobacco control officers', massive fines and, ultimately, imprisonment, would no longer be necessary. Everyday market forces would lead publicans to exclude smokers as a simple way to increase profits.

Instead there are well-vouched stories from the real world that many Irish pubs have suffered a drop in trade of 15/25% in town/country districts. At whatever level the Irish figures eventually settle, this single sample of wishful thinking suggests that zeal in such a noble cause as reducing smoking can lead even our most senior medical man to strain after 'evidence' to bolster his subjective preconceptions.

So my **first question** for the CMO is: *Would he acknowledge that his counter-intuitive judgement on the commercial 'benefits' of the Irish (as of the New York) ban has already proved rather facile?*

Sticking with dubious economics, Sir Liam's 2004 annual report further claims that smoke-free workplaces would bring annual benefits of up to £2,700 million. Although he

dismisses any testimony from the tobacco industry as tainted, he relies on the Department of Health's own staff obligingly to serve up figures *from unrevealed sources* on such 'costs' as smoking-related absenteeism (£70-140 million), smoking breaks (£430 million), and 'health benefits' (precisely £2,171 million).

On top of such wild estimates of benefits, his tame economists conjure up two disbenefits: one hard-looking figure of £1,145 million as lost tax to the Exchequer (presumably from an *assumed* reduction in cigarette sales), and another wholly esoteric invention of 'loss of satisfaction' to smokers (£700 million). I would not embarrass the CMO by asking how seriously he expects us to take such home-produced figures on the benefits of banning smoking!

BLACK ART OF EPIDEMIOLOGY

OF COURSE much of the propaganda endlessly recycled by the anti-smoking lobby looks much more impressive, and even scientific. But all of it turns out on close inspection to be fatally flawed, as judged by conventional standards of scientific validity or statistical significance. Rather than examine such criticisms, the medical mafia close ranks like a religious sect and scornfully dismiss them as coming from the hated tobacco industry. So I'd better come clean at the outset.

Not only have I been contentedly sucking my briars and meerchaums for well over half a century, but I am a former chairman, now president, of FOREST, the leading European defender of smokers' rights. It follows that I would take a good deal of convincing about the dangers of so-called 'passive smoking' – *but then so should politicians and journalists require convincingly hard evidence* before seriously contemplating an unprecedented coercive ban that must destroy the everyday pleasures of millions of ordinary people, as well as disrupting and damaging many tens of thousands of businesses after they have invested heavily in air filtration and separate non-smoking areas to placate their critics.

At least, as chairman and president of FOREST for almost two decades, I have had good reason to look more closely at the black art of epidemiology, of which the CMO was once a practitioner. Its purpose in ordinary language is to seek correlations between diseases and their *possible* cause or causes. Since it is impossible to measure scientifically or to control precisely the *amount* of smoke to which people may be exposed in changing atmospheric conditions, a surrogate measure has to be improvised.

The favoured method has been cunningly christened 'spousal smoking'. The big idea is to estimate the long-term (even lifetime) exposure to ETS of a selected group of *non-smoking* lung cancer 'cases' and compare the result with the exposure of a matched 'control' group of professed non-smokers without lung cancer. Thus the non-smoking wives with cancer are asked to recall how many cigarettes a day were smoked in their homes by their parents and later by their husbands (or, in one survey, by up to half a dozen previous partners!). If the wife had meanwhile died of lung cancer then her next of kin – husband, children or even nephews or nieces – may be interrogated.

We are here in the realms of anecdotage. One questionnaire I have studied actually asked of them: 'How many months of the year did she keep the windows of her house open

during her adult life?' How's that for cod science! However solemnly such data is collected, tabulated, calibrated, manipulated and presented, we must keep constantly in mind that they are all are purely *subjective estimates* without any reliable or consistent relation to objective reality. Let readers – smokers and non-smokers alike – pause and try to remember or guess how many cigarettes were smoked a week, a month or a year *in the house and in their presence* by their parents or close relations!

Anyway, how could you quantify the amount of smoke about the house? One indication of the difficulty is that when a Swedish toxicologist from Stockholm University, Professor Robert Nilsson, tried conscientiously to measure typical indoor exposure to ETS, he could do no better than come up with the equivalent of the effect on individuals of smoking between one cigarette a week and two cigarettes a year.

My **second question** is: *Does the CMO, from his earlier academic study of epidemiology, consider that what I would call 'secondhand recollections' of what he and his friends call 'secondhand smoke' are likely to be accurate within a very wide margin of approximation, say within 50 or even 100% or more?*

SMOKE AND MIRRORS

EVEN THE MOST scrupulous investigators inevitably face the demonstrable impossibility of their subjects being able to achieve accurate recall of exposure decades earlier. And even if such vague gropings were not sufficient to discredit all such ingenious research, there are two further major imponderables.

The first is that some former (and even current) *smokers* among the lung cancer cases will pass themselves off as *non-smokers* in order to shift the blame for their cancer on to smoking by others. In the trade this deception is labelled 'misclassification' and it is not uncommon. It has been found that self-reported 'non-smokers' can include between five and 25% of past or even current smokers.

The second defect is that while *the* cause of cancer eludes discovery, it is well known to be multifactorial – that is, associated with a multitude of possible causes or contributory factors. To isolate the effect of ETS on the lung cancer cases would require scrupulous examination of all possible alternative causes, to check whether the cancer cases differed from the control group, for example in genetics, diet (especially fruit, vegetable and fat intake), alcohol, urban living, lifestyle, stress, exposure to car exhaust, radon, asbestos, etc. (In the trade these alternative explanations of cancer are called 'confounders'.)

Instead of attempting to exclude such influences, much research crudely assumes that 'cases' differ from 'controls' wholly or mainly in estimated exposure to ETS. And researchers who do give a nod in the direction of enquiring about diet content themselves with desultory questions about consumption of fruit, vegetables and fat. Again, let readers ask themselves how many fried breakfasts, cabbages, lettuces, apples, bananas, etc they have consumed at various stages of their lives!

This has nothing to do with science, which requires well-defined, separable, stable elements that can be isolated and tested by repeated laboratory experiments yielding

consistent results. It is pseudo-science, based upon fallible recall, arbitrary opinion and biased human judgement which is then worked up into slippery statistics projected, as I like to emphasise, to two decimal places.

In all these investigations 'proof' of death by 'passive smoking' turns ultimately on comparing two spectacular, speculative, subjective guesstimates of relative exposure to ETS: that of the non-smoking lung cancer cases and that of the healthy control group. To repeat, it is essentially the relationship between these two guesstimates of exposures that is used to calculate the increased risk of lung cancer from ETS.

My **third question** for the CMO is: *Can he doubt that even the most conscientious attempt to measure and calibrate the (assumed) fatal concentration of ETS in varying conditions – from open windows to full air conditioning – presents formidable, even insoluble, problems, rather like nailing jelly to the ceiling?*

FANCY STATISTICAL FOOTWORK

CALCULATING the increased risk involves what one participant in all this nonsense has described as 'a veritable ballet of fancy statistical footwork'. Thus answers to the questionnaires (perhaps including fruit and vegetable consumption!) must first be rendered into malleable statistics before the risk of lung cancer associated with ETS can be calculated by a formula that divides the odds of exposure of the lung cancer cases by those of the (healthy) control group. There emerges a fraction called the 'relative risk' (RR) which is taken to indicate the enhanced risk of lung cancer from 'passive smoking'.

In effect, the exposure of the control group is regarded as a baseline (equal to 1.0) and the 'excess', if any, in the lung cancer cases is used to measure the precise increase in risk from ETS to, remember, *two decimal places*. It is this appearance of precision that assembles a rag-bag of often biased recall, rough estimates, impressions, subjective guesswork and anecdotal gossip, and invests the result with a spurious impression of scientific authority.

I should add that, since the risk of lung cancer among the general population is generally pitched at around 10 per 100,000, a relative risk of 1.1 would imply an increase from 10 to 11 per 100,000! Yet a whole industry has been spawned by epidemiologists straining to pitch the phantom statistical difference in assumed risk higher than before.

The **fourth question** is: *What weight would the CMO allow for such 'confounding factors' as 'misclassification' (of smokers as non-smokers), diet and 'publication bias' when most studies reveal small estimated differences in relative risk commonly around 10 to 30% (RR of 1.1 - 1.3)?*

An awkward fact is that the results of all the studies published in Table 1 (pp 35-39) exhibit little consistency in RR, with a dispersion (in those on spousal smoking, for example) ranging from 0.51 (based on eleven cancer cases) to 4.82 (based on eight). On the most strict reckoning a large majority of results show that the RR *could be negative* (ie below 1.0) which implies a *reduced risk* of lung cancer from higher exposure – that is, a *protective* effect from ETS – just as inoculation protects against subsequent infection.

Of course, since each research study examines only a small sample of the total universe (some fewer than a dozen cases of lung cancer), allowance must be made for a margin of error, which is conventionally pitched at 5%. The resulting 95% probability is shown with a varying 'confidence interval' which conveys the range within which the true relative risk is likely to fall.

For example, the result of the Butler study in 1988, based on a tiny sample of eight lung cancer cases and a probability of only 90% (in place of 95%), yielded a 'best estimate' for the RR of 2.02 (that is, twice the risk of lung cancer from ETS exposure) but the range of the confidence interval is shown as 0.48 - 8.56 at 90%. In layman's language this means we can be 90% certain (not even 95% in this study) that the true relative risk fell *somewhere* between 0.48 – which implies a strong *protective* effect from exposure to ETS – and 8.56, which implies a much enhanced risk of cancer.

To non-statisticians – as to any honest professional – the only safe conclusion from such an ambiguous result must be that the research was *completely inconclusive*. Yet the Butler study was eagerly selected for inclusion in the benchmark report by the US Environmental Protection Agency (EPA). Perhaps that 8.56 possibility looked too good to miss!

GREAT STATISTICAL HOAX

IF LAYMEN dare to question any of these guesstimates and projections the sophisticated statisticians take refuge behind their computers which have been heavily programmed to incorporate a variety of elaborate assumptions and statistical techniques. And since researchers have discovered that the bigger the reported risk the better the chance of attracting funding and getting their results published (known in the trade as 'publication bias'), they have exerted much ingenuity, as we shall see, in what is known as 'data dredging' – that is, torturing the statistics until they confess!

Even so, of 80 recorded case studies of 'spousal smoking', 57 show an increased risk of cancer which is not (definitively) statistically significant (because the confidence intervals range below 1.0) and ten show a *reduced* risk which is likewise equivocal. That leaves twelve studies (that is, one in seven) showing an increased risk which the medical mafia might seize upon as statistically significant – on the basis, remember, of the dubious data fed into their computers.

I have no space to extend my review to cover the proliferating research projects into ETS exposure in the workplace, which was hoped to provide ammunition for smoke-free offices. It will have to suffice to report the verdict of the International Agency for Research on Cancer (IARC) which has become increasingly watchful for evidence against smoking. In a recent monograph it concluded:

> 'In total, 23 studies have been published on [workplace] exposure to secondhand smoke … Only one study reported a statistically significant association between exposure to secondhand smoke at the workplace and risk for lung cancer.'

In 1992 the EPA issued its keynote judgement on 'passive smoking' as a cause of cancer and declared tobacco to be a 'Group A human carcinogen'. It did not conduct any new

research but, from the 40 studies of 'spousal smoking' then available from around the world, chose eleven published results from the USA. These were carefully selected as likely to support its boldly declared *'a priori hypothesis that a positive association exists between exposure to ETS and lung cancer'.*

Combining the results of different studies into a 'meta-analysis' is thought legitimate only if the researches are comparable in design, data collection, statistical procedures, etc. These eleven studies were far from complying with such criteria. Over half the studies embraced fewer than 30 lung cancer cases and the widely varied results were presented by the EPA – as we have seen – on a weaker statistical probability of 90% in place of the conventional 95%. Even so, three of the eleven studies reported a best estimate for a relative risk of below 1.0 which implies a *reduced* chance of cancer for those *most exposed* to ETS (ie a *protective* effect).

On the strictest test (which requires the lowest reading of the confidence interval to be above 1.0) only a single study survives as statistically significant – even, remember, at the weaker 90% probability! Yet it was on the slender basis of this rigged meta-analysis, claiming a weak combined relative risk of 1.19, that the EPA declared ETS a certain cause of lung cancer. The range of the confidence intervals spread from 0.30 to 8.56. That is, as we have seen, from a highly protective effect of ETS against lung cancer to a much higher risk of going down with the disease!

Even if all this statistical manipulation had yielded a consistent and strong association, we would still have to remember the accountants' caution of GIGO: garbage in, garbage out. The basic truth on which we must keep a firm hold is that the raw data from which all these ingenious calculations proceed were nothing more than crude, subjective estimates of varying-term exposure to an imprecise artefact which the medical mafia have shrewdly christened 'environmental tobacco smoke' or 'secondhand smoke' and tendentiously labelled 'passive smoking'.

Little wonder, then, that when the EPA was challenged in a US court the verdict was that it had wilfully disseminated false information, although on appeal the North Carolina Court was ruled as having no jurisdiction over the agency.

My **fifth question** therefore asks itself – indeed screams out for a direct answer: *Has the CMO studied the EPA's 1992 'benchmark' study and, if so, does he seriously consider it acceptable, let alone conclusive, evidence of the dangers of 'passive smoking'?*

QUESTION OF INTEGRITY

I HAVE LEFT to last my most serious charge against the integrity of those (probably a tiny minority) of the stop smoking brigade who have some familiarity with all these statistical pitfalls. Even where the figures show a 20, 30, 50% or higher increase in relative risk of lung cancer from exposure to 'passive smoking' they should know that scrupulous epidemiologists would not take that as *proof* of any significant correlation, let alone as *the cause* of cancer. I rest my case on three magisterial quotations from expert sources which cannot be dismissed as tainted by tobacco interests:

WHO International Agency for Research on Cancer, 1980: 'Relative risks of less than 2.0 may readily reflect some bias or confounding factor, those over 5.0 are unlikely to do so' (WHO/IARC, 1980 Science Publication 32, Lyon, page 36)

Sir Richard Doll, 1981: 'When relative risk lies between 1.0 and 2.0 … problems of interpretation may become acute, and it may be extremely difficult to disentangle various contributions of biased information, confounding of two or more factors, and cause and effect' (The Causes of Cancer, Doll and Peto, OUP, 1981, page 1,219)

National Cancer Institute, 1994: 'In epidemiological research, relative risks of less than 2.0 are considered too small and usually difficult to interpret. Such increases may be due to chance, statistical bias or effects of confounding factors that are sometimes not evident' (NCI, Abortion and Possible Risk for Breast Cancer: Analysis and Inconsistencies, Oct 1994)

So my **sixth and final question** is: *'Does the CMO attach any weight to the cautions expressed by leading epidemiologists against deriving proof of cause from anything less than a doubling of assumed statistical risk – that is, a relative risk above 2.0?'*

The reason anti-smokers throw such scrupulous cautions to the wind is a crude suspicion, even superstition, which might be expressed as: *there's no smoke without death*. In the stilted, giveaway words of the 1998 report of the Scientific Committee on Tobacco and Health (SCOTH): 'The consideration as to whether passive smoking is causally related to lung cancer *starts from the standpoint* [my italics] that active smoking is recognised as a major cause of lung cancer.'

So we see that the whole make-belief, and the accompanying abracadabra, turns on the semantic trick of describing ordinary breathing – in the presence of a smoker – as 'passive smoking'. It is precisely as though the campaigners against obesity chose to prejudge the ordinary process of eating as 'passive weight-enhancing'!

WHO DARES DISSENT?

PRIVATELY I have encountered Very Important Persons in the medical world who, in response to my earnest enquiry about 'passive smoking', have dropped their voices and looked around furtively before assuring me there was 'nothing in it', except for a possibly adverse effect on serious asthmatics. Since my VIPs would prefer not to be quoted I conclude with two prominent authorities who did venture to put their doubts on the record – and brought retribution upon their heads.

Take the plight of a venerable scientist commanding universal respect as the first to establish a link between cigarette smoking and cancer, namely Sir Richard Doll. In 2001, in the relaxed atmosphere of the BBC Radio 4 programme *Desert Island Discs*, this 90-year-old doyen of epidemiologists confided to presenter Sue Lawley: *'The effect of other people smoking in my presence is so small it doesn't worry me.'* When the gist was publicly quoted there was an immediate outcry from the anti-smoking propagandists at ASH, in response to which Sir Richard felt obliged to state he was only 'speaking personally'. But of course, how else would an honest man – even a conscientious anti-smoker – be expected to speak?

Since then I was surprised to hear that Sir Richard seems to have overcome his doubts. Following a 2004 newspaper report in which he appeared to have retreated from that view, I wrote to him seeking clarification. He replied courteously as ever that he had not abandoned his belief that relative risks between 1.0 and 2.0 are 'difficult to establish' but he now added that they were not 'impossible to establish'. He reaffirmed his long-standing view that ETS 'should be accepted as a cause of lung cancer, albeit one which produced only a small risk'. It suggested less than whole-hearted support for the alarmists who use statistics to justify a ban on smoking in all public places.

A more overt example of intimidation followed the publication by the prestigious *British Medical Journal* in 2003 of a study by two American medical scientists, Enstrom and Kabat, who drew on data from the American Cancer Society which tracked 118,000 Californians over 40 years (1959-1998) and concluded: *'The results do not support a causal relationship between ETS and tobacco-related mortality …'*

Publication provoked a fierce barrage of 140 letters, many of which treated the editor to lectures on 'the evils of tobacco' and accused him of being 'naive', 'stupid', 'mad', 'irresponsible' or having 'darker motives'. Every known lobby, including some new ones such as Smoke-Free Educational Services, joined forces in an attempt to bully the editor to withdraw.

Instead of retraction or resignation, which the lobbyists impudently demanded, the then editor, Dr Richard Smith, was stung into a robust rejoinder. He explained that, although 'passionately anti-tobacco', the *BMJ* was not 'anti-science' and *'the question* [of whether passive smoking kills] *has not been definitively answered'.* (Little over a year later, at a fringe meeting organised by the Institute for Public Policy Research during the 2004 Labour party conference in Brighton, he appeared to have retreated from that view, although without revealing his reason.)

SUPPRESSION OF DEBATE

THERE have been further reports on the subject in the past year, including one from SCOTH in April 2004 and another from the International Agency for Research on Cancer in May. Neither however appeared to add anything in the way of fresh evidence but played the old game of regurgitating earlier studies. The summaries I have seen do not even begin to allay my suspicions. Thus the press release on the IARC monograph, running apparently to 1,500 pages, included a completely inconsequential statement that: 'Non-smokers are exposed to the same carcinogens as active smokers', which blithely begs the question of the amount/frequency/extent of exposure.

The brief SCOTH review (nervously marked 'restricted' and 'for members' use only') reported on recent studies which mostly failed to reach statistical significance, but on a 'pooled analysis' showed an excess risk of lung cancer from passive smoking of – surprise, surprise – 24%, which is almost exactly the figure this committee has been pedalling for years! Apart from smacking of 'data-dredging', this pooled analysis defies belief by claiming that almost identical (average) results should emerge from dozens of studies, conducted by different researchers presumably with differing degrees of competence (and hunger for 'significant' results), all purporting to measure exposure to such an elusive will of the wisp as environmental smoke.

We must keep constantly in mind that this assortment of contrived 'evidence' is based, as we have demonstrated, on nothing more substantial than estimates, guesswork, subjective recollections and even gossip by the next of kin of selected wives who claimed never to have smoked but were married to or lived with smokers and may have died of lung cancer.

A conscientious student coming to this important, even life and death, topic for the first time must be struck by the apparent lack of doubts about the crude modus operandi of research into the assumed lethal potential of 'passive smoking'. The uncanny unanimity of the whole tribe of vocal practitioners – who constantly quote one another's findings with unquestioning approval – might arouse suspicions of collusion, especially in the efficacy of peer review.

A lively-minded student new to these mysteries would surely conclude there was ample scope for the keen discussion and disputation about methodology and interpretation common among most other ambitious academics competing for attention and publication. The suppressed truth is that there *are* many highly qualified statisticians and scientists who dissent from the overbearing consensus on 'passive smoking' but who lack any influential outlet for their heretical views.

The lengths to which the anti-smoking establishment is prepared to go to smother rival views was blatantly displayed in October 2004 when the Tobacco Manufacturers Association booked a room at the Royal Institution in London for a seminar on 'The Science of Environmental Tobacco Smoke'. Specialists representing alternative viewpoints were invited to take part in the hope of civilised debate. Here was an opportunity at least to narrow differences of expert opinion and perhaps improve on the ramshackle methodology.

Instead there was an immediate outcry against Baroness Greenfield who as well as being professor of Pharmacology at Oxford University is a director of the RI. The chairman of the Royal College of Physicians protested vociferously against Lady Greenfield's decision to hire out its public rooms to people who would use them to 'cast doubt on whether passive smoking is harmful and to promote the idea that ventilation might be a solution for public places'. (A characteristic of those demanding an outright ban on smoking is that they veto talk about ventilation, which of course blows a large hole in their bogus assumption that ETS is a stable, uniform entity.)

Baroness Greenfield responded patiently by explaining that the RI did not endorse any of the industries that hire its premises, adding robustly: 'If we blocked this in a politically correct way, where would we be with the drinks industry or food companies. We would have Alcoholics Anonymous and the anti-obesity lobby objecting too.'

Here we glimpse the absolutist character of the anti-smoking campaigners. Having boycotted the seminar at the RI they would presumably wish also to suppress this modest essay if only their power extended to control over the printing presses. Instead they will have to content themselves with closing rank either to ignore any public response or to denounce the author as a lackey of the international tobacco cartel! We may, alas, expect that their many allies in the media will hesitate to give my critique publicity, less from honest doubt than from fear of being stigmatised as soft on smoking.

I understand the Chief Medical Officer was invited to participate in the TMA seminar but declined. Such resistance to open public debate may suggest to onlookers a certain lack of confidence in the ability of the anti-tobacco crusaders to carry the day. Why otherwise should these high-minded, would-be saviours of millions of stubborn smokers fail to understand that their participation in such a debate might have impressed sceptics more than all the shrill propaganda, intimidation and ever more gruesome advertising on television?

COLLECTIVE CONVICTION

AFTER MUCH anxious pondering I have come to the settled conclusion that what we are witnessing here is a variant of political correctness which I would call 'collective conviction'. I define this condition as *a dogmatic shared sense of absolute certainty among a mutually supporting intellectual elite*. It is not unique to the smoking debate, or rather non-debate. On other important topics, such as 'global warming', we have seen how a ruling consensus is first established by the conceit of a coterie of prominent, articulate pioneers.

The Big Idea then spreads by the contagion of novelty and fashion until it infects almost the whole intellectual class. Finally, as Hayek showed in his scholarly essay on *The Intellectuals and Socialism*, the pervasive influence of journalists and other 'second-hand dealers in ideas' completes the chain of collective conviction by establishing a new consensus which comes to dominate public discussion, opinion and, ultimately, public policy.

I witnessed this process at close hand in my own subject of economics after the last war when the novel theories of J M (later Lord) Keynes led to a radical school of thought that spawned powerful lobbies among trade unions, industrialists, academics and footloose political activists which came to dominate public thinking and policy on the central questions of unemployment and planning.

As with the issue of 'passive smoking', the broadcasting and print media largely fell in with the new 'spirit of the time' and it took some courage for a comparative handful of independent, non-conforming economists, mostly associated with the Institute of Economic Affairs, to withstand the stampede and keep alive the classical tradition of free markets and monetary policy. The tables were eventually turned on the Keynesians not only by the superior logic of their critics but by the brute force of the resulting inflation and disorder which those critics had long predicted.

If the fashionable claims of those I might call 'passive thinkers on passive smoking' could similarly be put to the test of experience I have not the least doubt they would be equally discredited. As it is so much intellectual capital has been invested in this will of the wisp of ETS that, as we saw with the Royal Institution seminar, its practitioners fiercely oppose even the usual processes of civilised open debate with their equals who dare to disagree. Indeed, dissent is taken to disqualify sceptics from participating in serious public discourse!

THE VERDICT

MEANWHILE most laymen, journalists and politicians who have jumped on the bandwagon of 'passive smoking' have no idea that the evidence turns out to be no more than statistical projections based on varying recollections of the exposure to tobacco smoke by (self-proclaimed) non-smoking wives married to smokers – who may spend more time smoking in their studies or workshops or local pubs than puffing over their partners!

The first question to ask anyone pontificating on the dangers of ETS is not whether they have studied the neat statistical projections concocted from variable answers to questionnaires on 'spousal smoking' – which a minority may well have done. The knock-out question is to ask whether they have actually inspected the original questionnaires about long-term 'exposure to ETS' on which the 'relative risks' are then ingeniously calculated to two decimal places!

But what are we to make of those 'experts' who, with few brave exceptions, have led the bloodcurdling chorus of death from ETS and keep any scientific doubts to themselves? The best that can be said is that, like the members of SCOTH, they are driven on by such a single-minded obsession with cancer that they have allowed themselves to be persuaded that any means of stigmatising and punishing smokers may be justified in the 'good cause' of reducing such a self-evidently risky and 'anti-social' activity. This is the fatal fallacy that the end justifies the means, including exaggeration, spinning, deception and – when that does not work – the outright persecution of dissenters.

The more militant anti-smokers have allowed their hatred of cancer to give way to contempt, if not hatred, for some 12 to 14 million of their fellow men and women who continue to smoke. They think nothing of inventing further statistics to bring smokers into public obloquy. Thus they have dishonestly claimed that smokers cost the NHS £0.8 billion, then £1.5 billion, then £1.7 billion, deliberately ignoring the tax on tobacco which brought into the Exchequer over £10 billion (£10,000,000,000) a year until smokers were driven to buy a quarter of their cigarettes from Europe where taxation is lower.

They have further ignored the visible evidence that the spread of air conditioning and ventilation is transforming – and improving – the atmosphere in more and more pubs and restaurants. They affect not to notice the consideration and courtesy which most smokers increasingly display in the presence of non-smokers. They raise not a murmur of humane protest when some doctors have refused to give urgent medical treatment to smokers. They appear to be unconcerned that a smoking ban would almost certainly lead to the spread of obesity and drug addiction.

Having already escalated the war against smokers – from harsh taxation, gruesome (often false) health warnings on cigarette packets, to vivid TV advertising, all of which would cost millions of pounds at commercial rates, whilst unprecedently deploying legal censorship against rival commercial promotion of tobacco brands – our 'keepers' are understandably frustrated that at least a quarter of the adult population dare to defy them by continuing to smoke. So by natural progression they have recruited children, neighbours and colleagues into the battle.

The campaign was launched in 1975 by Sir George Godber, a former Chief Medical Officer, who addressed the World Health Organisation on the need to move on from frightening smokers about damaging their own health to frightening non-smokers about the lethal effects on the health of innocent bystanders: 'We must foster an atmosphere where *it is perceived* [my italics] that active smokers would injure those around them, especially their family and any infants or young children.'

Since then huge amounts of public and charitable money have been lavished on persistent 'research' which has failed to yield any consistent or statistically significant evidence against the artefact of 'passive smoking'. Instead we have witnessed the prostitution of science as the final solution, short of attempting to extinguish the last cigarette by outright prohibition, backed by whatever police invigilation and ultimately imprisonment may prove necessary.

The American experience with prohibition of alcohol after the First World War suggests that a legal ban on smoking in 'public places' will not succeed in permanently stopping so well-entrenched and time-hallowed, if potentially hazardous, a relaxation as smoking. The evidence against the danger of ETS remains in my view quite unequivocal. The imposition of a ban on smoking in so-called 'public places' would therefore represent a triumph of prejudice and propaganda masquerading as science. It has no place in a free and tolerant society and must in the long run risk bringing science itself into further disrepute.

POSTSCRIPT

HAVING re-read this introduction I am not tempted to censor any of my harsh judgement on the perpetrators of the pseudo-scientific fraud of 'passive smoking'. Hatred of cancer is perfectly understandable, like hatred of road deaths, medical incompetence or child molesting. The dangers of heavy cigarette smoking may justify a steady educational campaign on the possible consequences, especially directed at the young. But hatred of cancer is no excuse for hatred of smokers nor for stirring up the wholly phantom fear of 'passive smoking', especially by cynical politicians, to whip up support for illiberal, intolerant policies of prohibition.

A modest taste of this mania was recently displayed by a dedicated minority of peers who deliberately chose the final, thinly-attended sitting before Christmas 2004 to spring a vote on smoking policy within the House of Lords. After a bad-tempered debate, the few remaining areas where smoking was still permitted (including such convivial places as the Peers Guest Room and the Bishops Bar) were whittled down to a single room less than 30 feet square where neither food nor drink are served.

The speeches from the mostly Labour anti-smokers were full of the familiar unsupported assertions of the harm caused by 'passive smoking', especially to members of staff. (Their claim of unanimous support from unions – here as elsewhere – is of course not borne out by direct enquiry among employees.)

From the Cross-benches I could detect not the least whiff of interest in the contrary case which was enthusiastically voted down by 88 votes to 53. To judge by the senior heavyweights who voted with me for preserving choice, including Geoffrey Howe, David

Steel, Jean Trumpington, Jack Weatherill, Betty Boothroyd, David Stoddart, Patrick Jenkin and Gerry Fitt – I could console myself only with the reflection that the vote may have gone the other way in a better-attended House!

The very latest outrage comes, no longer surprisingly, from Scotland, once the home of steady reason, moderation and commonsense but now a cockpit for radical pressure groups. Following a patently bogus period of 'public consultation', first minister Jack McConnell came up with the predictable announcement of a comprehensive smoking ban in all indoor 'public' places, including private clubs and privately-owned bars and restaurants. To justify this draconian measure, to be implemented in 2006, he claimed that 'passive smoking' is associated with 1,000 deaths a year in Scotland alone.

I do not expect the media to call him to task for such a reckless exaggeration that puts even SCOTH in the shade with its cagey estimate of 'about several hundred extra lung cancer deaths a year' for the whole of the UK. However it bears constant repetition that this flimsy supposition relates to an adult population above 40 million and is based upon a trivial elevation of a statistical fiction called 'relative risk' – a 'risk', as we have seen, of nothing more than exposure to an arcane, esoteric, semantic artefact which anti-smokers delight in bandying around as 'environmental tobacco smoke'.

But good may yet come out of so much mischief. Might not all these brazen inventions provoke an overdue public response to such a plainly irresponsible political game as 'Think of a number – and make it larger than the last time!'. Might such blatantly obvious puffing up yet stir a growing number of respected voices to join me in publicly calling the bluff on the entire 'passive smoking' industry?

As I have hinted, what finally inspired me to probe more deeply into the mysteries of 'passive smoking' was partly the memory of those Very Important Persons who, looking around and lowering their voices, whispered in my ear: 'There's nothing in it.' Now, having confirmed they were right, I have attempted to set forth the case for the prosecution against the anti-smoking brigade. My real challenge, however, is over the heads of this claque band, led by a plainly partisan Chief Medical Officer, to the many independent champions of open, straightforward, fearless public debate. What is now most urgently required is a lot more active scrutiny of the myth of 'passive smoking'.

Ralph Harris
February 2005

PART ONE

questions and answers about ETS

In Part One we will address some common questions about environmental tobacco smoke (ETS) aka 'secondhand smoke' (SHS) or 'passive smoking' (PS). In Parts Two and Three we will provide a more detailed primer on the 'science' behind the alarmist claims that are encouraging some governments to ban smoking in all enclosed public places.

NOTHING WORKS better than a slick slogan, as the advertising industry has always known. That's why, when antismoking campaigners began claiming that 'passive smoking kills', they found a winning formula. Some of them no doubt were sure that epidemiological studies would eventually bear out their claims. Others were quite content to push the idea of secondhand smoke as a danger to non-smokers because that, they reasoned, would put pressure on smokers to quit or be marginalised.

The only problem with the claim that ETS is a serious danger to health is this: there is no conclusive evidence that it's true. At the time of writing research into the health effects of ETS have been going on for 25 years. And despite the spin put on the results by antismoking campaigners, the best that can be said is, 'case *still* not proved'.

Q *If it's not true that ETS is dangerous, why do so many people say it is? Why do we hear about it every time a national body or local authority proposes a smoking ban?*

A Antismoking campaigners, including many politicians, are determined to stop people smoking. The suggestion that smokers are harming non-smokers is a carefully orchestrated means to an end. They believe that banning smoking in all public places will encourage some smokers to quit while marginalising the rest. Even when they know that the 'mountain of evidence' about passive smoking is a mountain of something quite different, they believe it doesn't matter. ETS a very effective weapon with which to demonise smokers and justify their calls for a blanket ban on public smoking and they are making the most of it.

Q *But doctors and health authorities support the claims. Why would they if it wasn't true?*

A In a word, politics. There is a 'party line' on passive smoking and dissent is neither encouraged nor welcome. We also live in an age of specialisation. Very few people in the health community are 'experts' on ETS. Many doctors and health officials simply accept the 'consensus' view. Some understand that the claims aren't sound but they conclude that obscuring the truth about passive smoking is acceptable if it helps people to quit smoking. Others fail to speak out for fear of professional retaliation. 'Going against the grain' has its costs in career terms.

Q *Why are there so few public debates about ETS?*

A Quite simply, the antismokers refuse to take part. Their approach is summed up by one leading campaigner who argued that 'There is no debate' about passive smoking. It's easier for the medical profession (and politicians) if the public accepts what they have to say about ETS as an act of faith. Debate is frowned upon. Of course there have been an increasing number of public meetings about smoking bans but they tend to involve mutually admiring 'experts' who reassure each other about the righteousness of the antismoking cause. Speakers argue only over minor points of implementation, asking each other how far they can go and how soon. The scientific foundations on which smoking bans are being promoted are rarely questioned.

Q *But if the science on ETS is so flawed, surely we could expect some doctors and scientists to speak out about it?*

A Some doctors and scientists *have* spoken out but they are shouted down by an anti-smoking movement which is well-organised, well-funded, and on a roll. Those who have dared stick their heads above the parapet include Dr Ken Denson of the Thame Thrombosis and Haemostasis Research Foundation who wrote, 'The ill effects of passive smoking are still intuition rather than scientific fact, and billions have been spent by the medical institutions in pursuing this illusory myth' (August 2004). Liewise Dr Michael Fitzpatrick wrote, 'The alarming estimates of deaths attributable to passive smoking result from multiplying miniscule risks of dubious validity by vast population numbers – an effective propaganda device but statistical sharp practice' (www.spiked-online.com, November 2004). And Dr Richard Smith, former editor of the *British Medical Journal*, famously commented (July 2003), 'We must be interested in whether passive smoking kills, and the question has not been definitively answered' (August 2003).

Q *Why is the passive smoking argument so important to antismokers?*

A Before the antismoking movement created what Dr Denson (above) calls 'the myth of passive smoking', smokers were largely tolerated by non-smokers. The general attitude was 'They're only harming themselves.' Antismokers therefore decided to argue that smokers were harming others around them which of course makes smoking 'socially unacceptable'.

It's a clever tactic and it's working. By constantly giving the impression (without ever proving) that ETS is a serious cause of ill health among non-smokers, the antismoking lobby has been able to increase people's intolerance of smoking and deprive smokers of the important argument of free choice. In short: the antismoking movement has found passive smoking to be such an effective propaganda tool that objective science cannot be allowed to get in the way.

Q *But if primary smoking is as harmful as they say it is (and few people deny there are serious health risks associated with primary smoking), surely passive smoking must be bad for non-smokers too? And even if it doesn't kill them, why should they be exposed to ANY risk?*

A It is true that ETS contains 3-4,000 chemicals. But so does any organic compound (wood, for example) that is burned. The important thing is that ETS (and even primary

smoke) has minute amounts of those compounds. In the case of ETS the smoke compounds undergo numerous chemical processes that make it quite different from primary smoke, even chemically, and a further process involves heavy dilution in the surrounding air. (For further information see Table 3, page 50, which compares toxins in passive smoke in a set of very extreme theoretical conditions to government workplace standards in the US, and Table 4, page 51.)

As for making ourselves safe from any risk (as in the antismokers' latest mantra, 'There is no safe level of environmental tobacco smoke'), it is simply not possible. We have to take *some* risks in life, even to get to work in the morning. The real issue is where to draw the line on what constitutes acceptable risk. This is a hot topic in science and public policy circles where there is an ongoing battle between those who champion a 'zero risk' approach and those who think such an approach is impractical and undesirable. (See our comments on the Precautionary Principle, page 26.)

Popular culture reflects the trendy 'no risk' mentality in the health scares that pepper the media and lead to uninformed hysterias about 'risky' things – or those that 'may' be risky according to someone's epidemiological study. Examples of health scares (there are hundreds to choose from) include the notion that the use of antibiotics might be a cancer risk, or that plastic packaging might be making men impotent. Might. As we will see from our examination of multifactorial epidemiological evidence, modern researchers attach very exact and scary-sounding numbers to risks that often boil down to 'might', 'may', 'can', or 'well, it's not impossible'.

Today, having been bombarded with antismoking propaganda, many people intuitively believe that ETS must be harmful simply because it involves tobacco smoke, albeit massively diluted. (As FOREST president Lord Harris of High Cross puts it, 'There's no smoke without death.') Perhaps it's time to do a study to see whether we should ban cooking – especially frying – in restaurants. Don't laugh. Several studies have been conducted on the 'carcinogenic' effects of cooking as well as of incense in churches. Let's not even talk about cars!

Q *What exactly is 'epidemiology'? And what is 'multifactorial epidemiology'?*

A Epidemiology is the study of the causes of disease. It is most reliable when it looks at diseases that have one obvious cause, infectious diseases like cholera, influenza, etc. They are called 'monofactorial' diseases. Diseases in which multiple factors are believed to play a part (eg lung cancer and heart disease) are called 'multifactorial' and the study of those diseases is called 'multifactorial epidemiology'.

When epidemiology starts to tackle diseases or disorders that do NOT have a single cause, things get complicated. The less that is known about the causal mechanisms, and the more factors are suspected to be involved in creating the disease (such factors could include genetics, toxic exposures, exposure to micro organisms, stress levels experienced over time, etc), the harder it is to say anything meaningful about actual causation.

Q *Do the terms 'correlation' or 'link' mean the same thing as 'cause'?*

A No. A 'correlation' or 'link' simply means that there is a correspondence or parallel between two phenomena. In the case of epidemiology, this parallel is between the frequency with which a disease appears in a group of people under study and the occurrence of something that scientists suspect may cause (or help cause) the disease. The suspected cause of the disease could be one of hundreds of 'suspects' whose effects cannot be isolated.

Q *So what is the basis of the anti-smoking movement's claims that ETS poses a serious risk to the health of non-smokers?*

A There is almost no basis for the claim, other than intuition. Many studies have been carried out, some of them rather poorly designed, others more impressive in their methodology. But, viewed as a whole, the studies are demonstrably inconclusive – and this is particularly true of the larger, better designed studies.

Q *What do you mean by 'inconclusive'?*

A It means there is simply no conclusive proof – no persuasive evidence – that ETS as we typically encounter it in ordinary life is a risk to anyone (although common sense would suggest that serious asthmatics should steer clear of very smoky environments). It also means there is no proof of the *absence* of risk. This in turn implies that if a risk does exist it is likely to be too small to be measured. That would lump ETS in with scores of everyday activities and exposures that we all take for granted as part of life. This is what the 'mountain of evidence' so often mentioned by proponents of smoking bans actually amounts to.

Q *I've heard there is a 20-30% increased risk of lung cancer or heart disease from passive smoking. That sounds alarming.*

A It sounds scary (and is meant to) but, in layman's terms, unless the risk is at least doubled (ie 200% or more) it isn't a significant risk according to the established rules of epidemiology. This is a vital point to understand. Epidemiology has set these rules for the kinds of studies that examine diseases caused by more than one factor because there is an enormous margin of error that is accumulated in even the most diligently conducted study. However, even a statistically significant study can have methodological flaws – and the secondhand smoke studies usually do because they are almost always based on individuals recalling events that took place many years, sometimes decades, earlier.

Q *OK, but tobacco smoke still stinks and can make your eyes water. Isn't that evidence of harm?*

A Without question, strong concentrations of secondhand smoke can be annoying to non-smokers, and even to smokers themselves. But harmful? The point is worth closer examination. When ETS is concentrated enough to become uncomfortable, that's an indication of poor air exchange – and when eyes are watering, that means the air exchange is down to dangerous levels. If we run an analysis of that air we often find all sorts of stuff that is bad for our health: elevated levels of carbon dioxide, lack of oxygen,

various chemical compounds, high levels of dust, viruses, bacteria. Those elements are there regardless of ETS, which indeed becomes a useful tool to indicate that it is time, literally, to change the air. Without the ETS marker people would be exposed for much longer to harmful elements without realising it, as most of those elements are invisible and odourless.

It is also worth noting that despite having eliminated ETS in enclosed public places for several years, states such as California continue to experience increasing rates of asthma. One problem is that once smoke is eliminated and people think the air is 'clean', air circulation systems are turned down to save energy. Isolated claims that there is a decrease in lung cancer incidence since tough antismoking measures were enforced are just propaganda because lung cancer requires decades to develop. (The state-wide ban in California was introduced in 1998, in New York it was 2003.)

Q *How then do we explain all those deaths that are said to be caused by ETS?*

A Public health activists love to use the word 'caused' to induce people to believe there is an established causality. As we will see in detail later there is no demonstrated causality of death and disease from ETS. It is interesting, for example, that while the US Environmental Protection Agency still sticks to its original estimate of 3,000 ETS-related deaths a year in the USA, other sources claim 53,000 – a figure over 17 times larger. Likewise in the UK projections of deaths caused by passive smoking range from 'several hundred' per annum to 10,000, including one barworker every week! What does this tell us about the accuracy or credibility of such figures?

Q *Why don't we see such discrepancies for polio, tuberculosis or malaria, for example?*

A Because in those cases there is a monofactorial established causality, which is not the case for 'tobacco-related' diseases. There is no need to be a specialist, or even to look at the methodologies, to understand a simple truth about the huge discrepancies in 'tobacco-related deaths': the figures are made up through arbitrary statistical computer models.

Q *Nevertheless it is said that when ETS is removed from enclosed working environments people's health improves. For example, a six-month public smoking ban in the city of Helena, Montana, USA, apparently demonstrated a reduction in heart attacks during that period. Surely that proves something?*

A The highly publicised (but unpublished) Helena 'study' showed that just six months after public smoking became illegal in Helena, the rate of heart attacks decreased by a whopping 50%. Writing for *Reason* magazine in the States, journalist Jacob Sullum reported that the study was conducted by Richard Sargent and Robert Shepard, two local physicians and enthusiastic backers of Helena's smoking ban, as well as Stanton Glantz, 'a well-known anti-smoking activist who directs the University of California at San Francisco's Center for Tobacco Control Research and Education'.

According to Sullum, 'Sargent says they discovered that the smoking ban "led to an immediate and dramatic decline in the number of heart attacks experienced in Helena."

But regardless of his position on smoking, any honest doctor will tell you (at least privately) that it takes years for heart conditions to develop – even for heavy-duty primary smokers.' Sullum went on to describe how the study was presented:

> 'According to data Sargent presented at a recent meeting of the American Academy of Cardiology, St Peters Community Hospital in Helena saw an average of fewer than four heart attacks a month from June, when the smoking ban took effect, until December when the ordinance was suspended because of a legal challenge. From 1998 through 2001, by contrast, the hospital recorded an average of about seven heart attacks a month during the same part of the year. In a UCSF press release, Glantz declared, "This striking finding suggests that protecting people from the toxins in secondhand smoke not only makes life more pleasant, it immediately starts saving lives."
>
> 'To a more sceptical eye, this striking finding suggests the perils of drawing conclusions from a single study involving tiny, volatile numbers. As Richard Pasternak, an associate professor of medicine at Harvard, told UPI, "This is a small study, so we have to be cautious in how we interpret these results." Unless we work for the news service at Glantz's university. The headline on the UCSF press release, which was echoed by news outlets, announced, "Public Smoking Ban Slashes Heart Attack Rate in Community."'

Q *Even if it doesn't present a serious risk, I've heard it said that 'Smoking around others is like peeing in a swimming pool'. Advocates of smoking bans say we shouldn't have to put up with it. That sounds persuasive.*

A A vast library of short, easy-to-remember slogans and soundbites has been developed to hammer home a consistent message about ETS. The well-worn 'peeing in a swimming pool' jibe is calculated to suggest that a common good enjoyed by all (air) is polluted in some disgusting, anti-social way by a few to the discomfort of all in an enclosed public place. But given the tiny amounts of smoking pollution in a normal enclosed environment and the inconclusive evidence of harm, the truth is that 'Smoking around others is like peeing in the ocean' – as, eventually, we all do.

Q *If there is so much dubious science concerning ETS, truth will out in the end, won't it?*

A One would like to think so. Unfortunately it's not looking good – the adoption of a highly controversial guiding principle called the Precautionary Principle by the European Union and others (it was incorporated into the French constitution in June 2004) has seen to that. Beginning as a rather loose 'better safe than sorry' idea that stemmed primarily from the environmental field, the Precautionary Principle as a formal statement is quickly becoming universal in application.

At first, it seems common sense: prevention is better than cure. That, however, would be an erroneous and superficial interpretation. The implications of this principle are actually astounding and mostly unknown to the public. Most of us probably assume that if a government body attacks an industry for producing a dangerous or environmentally damaging product, the onus is on the government to prove that danger.

The Precautionary Principle reverses that onus and if the industry concerned fails to prove that the product is not dangerous then government has the power to completely forbid and/or regulate at will. Suspicion of possible harm is evidence enough. But every scientist will tell you that it is very difficult if not impossible to prove a negative. So too will every lawyer. This ensures that the accusing authority is always 'right'.

But it doesn't stop there. Even if one was able to prove a negative, suspicion of possible future harm is still enough to permit intervention. You don't need much imagination to see how nicely this 'principle' fits the passive smoke fraud in particular, and epidemiological junk science in general. The Precautionary Principle protects junk science and its perpetrators and enables bureaucrats and politicians to regulate, tax and prohibit at will.

Q *If the attack on passive smoking helps rid society of tobacco, what's wrong with that?*

A It's corrupt. The quality of information that the public receives is vital for the proper functioning of a democracy. We may not always get high ethical standards concerning the information given to us, but we must always fight for them. Moreover, in many jurisdictions it is taxpayers' money that at least partially pays for anti-tobacco programmes. We have every right to demand accountability. More important, many people still think that individuals and minorities should be able to choose for themselves whether they wish to enjoy a cigarette, a pint and a pizza, or go fishing, fox hunting, hang-gliding, mountaineering etc.

Q *Why isn't the public aware of these issues?*

A One, the anti-smoking movement has been very successful. Among other things they have discouraged debate. Dissenters are accused of being 'stooges of Big Tobacco'. Like an accusation of witchcraft, it works – on the media, on politicians, on scientists, on the public. Even high profile antismokers who go 'off message' are forced to repent, publicly and quickly.

Two, the issues are hard to explain. In order to understand something about the real state of the evidence on ETS it is necessary to know at least a little about statistics, and the uses and limitations of epidemiological investigation. Few people do, and that includes reporters, politicians and bureaucrats, all of whom are regularly lobbied by well-funded anti-smoking activists.

Q *Why doesn't the media take more interest in this issue?*

A They aren't motivated. Politicians and lobbyists want them to help participate in the 'health revolution' and so do the pharmaceutical companies who in many jurisdictions can now advertise directly to the public (for products that include smoking cessation pills and patches). No-one – especially journalists or politicians – wants to be accused of defending 'unhealthy choices'. And there's another reason. Most people lack the background to check the claims about ETS. It's much easier – even for reporters – simply to accept arguments from authority and 'take the experts' word for it'.

Q *I don't smoke. Why should I care about any of this stuff?*

A Everyone should be concerned that we have honest public discourse and decision-making, whether or not we have a personal interest in the subject matter. The most important general message we want to convey in this document is that the propaganda on ETS has been dishonest and deliberately confusing. You may not care because it's 'about smoking'. You may cheer because you don't like smoke in pubs and restaurants. But how will you feel when the same tried-and-tested techniques are used to attack something you do care about?

To be anti-smoking is one thing, but must we be anti-science as well? If junk science can be successfully applied to smoking it can be used in other areas too. In a free society is it acceptable that the end justifies the means? Specifically, can we allow institutions and campaigners to misrepresent evidence to achieve what they argue is a 'good' cause? Where and when will this stop?

The same abuse of statistics used in the antismoking crusade has already been adopted to target those who drink or eat the 'wrong' things. Can we allow epidemiological frauds to become a standard tool in the hands of governments and health authorities? Aren't we simply being dishonest if we embrace the notion that ETS is a 'danger' simply because we have been induced to hate smoking? If we see the fraud will we acknowledge it as such or will we reject that knowledge because the fraud works in our emotional interest?

Should we, in short, permit 'concern for health' to become the political ticket for limitless restrictions of choice and lifestyle, control of free commerce, conditioning of our children, misrepresentation of evidence by medical authorities, unlimited taxation, massive redirection of public funds, and endless regulatory power by an exploding bureaucracy?

Of course those directly affected by smoking bans, including those in the hospitality industry, also have an urgent and vital interest in this issue – whether they smoke or not. After all, legislation that will force you to ban smoking on your premises is a direct violation of your right to run your business as you see fit, within existing health and safety regulations.

As for ETS being annoying to some non-smokers (ie the comfort factor), it is surely not beyond the wit of man to devise a suitable compromise (eg smoking and non-smoking rooms plus decent ventilation) that can accommodate those who want to light up without inconveniencing those who do not want to breathe other people's tobacco smoke. If you believe in tolerance, courtesy and common sense solutions to social 'problems' such as environmental tobacco smoke, you should not only care – you should make your views known and support the overwhelming majority who want a choice of smoking and no-smoking pubs, clubs and bars – not a blanket ban on smoking in all public places.

PART TWO

epidemiology and ETS

THE EPIDEMIOLOGY of environmental tobacco smoke (ETS) – aka secondhand smoke (SHS) or passive smoke (PS) – aims at finding correlations between various diseases and exposure to ETS, from which it attempts to infer cause-effect connections. Epidemiologists dealing with this issue insist that ETS investigations are scientific, even though most prominent epidemiologists agree that the epidemiology of ETS is not and cannot be scientific.

To get a better understanding of why this is we need to take a closer look at the way ETS and disease is studied. We have made every effort in this report to avoid excessive technical detail because our goal is to provide an accurate but simple explanation for the layman. The basic ideas expressed here are not difficult to grasp if the reader exercises a little patience.

WHY SCIENCE AND ETS ARE INCOMPATIBLE

A TRULY scientific epidemiologic study of ETS would work like this: an experiment would be set up where non-smokers were randomly assigned to groups before being exposed or not exposed to ETS, during a time adequate for the development of the possible diseases. However, such a study would be unethical because it could conceivably result in harm to study participants. It would also be impractical because it would take decades to complete.

The epidemiology of ETS is therefore limited to retrospective surveys in populations of self-declared non-smokers who happen to have been exposed to ETS in the course of their lives. Indeed, the subjects usually would be of an advanced age where possible effects such as lung cancer or cardiovascular diseases could be observed.

What this means is that epidemiologic studies of ETS are not experimental, they are observational – an attempt to look back in time after the fact and observe what may have happened. The researchers don't have the opportunity of taking pre-emptive steps to ensure the independent reliability of the outcomes they observe. Such studies obviously have a lot of built-in problems.

Ultimately the reliability of any empirical evidence, scientific or not, depends on having met three basic benchmarks:

- An assurance of identity, namely that what is being measured is indeed what is claimed to be measured, and measured with sufficient accuracy.

• An assurance of the absence of other explanations, namely that the effects observed are due exclusively to what is being measured (in this case, ETS) and not to other disturbances that interfere with and may alter and confound the results.

• An assurance of consistency, namely that results are consistently reproduced by different reports.

Without meeting these three guarantees no hypothesis can aspire to reach any degree of credible evidence and cannot be credibly taken as the basis for reasoned policy decisions, either public or private.

As we are about to find out, not only are the epidemiologic studies of ETS unable to ensure that they have measured ETS exposures with any degree of accuracy, they are also unable to ensure the absence of other explanations for their observations. They are also inconsistent with some studies claiming a slight *increase* in risk, others suggesting a *decrease*, although the majority show neither an increase nor a decrease in risk.

EPIDEMIOLOGY AND PASSIVE SMOKING

THE epidemiology of ETS claims correlations of ETS exposure with cancer, cardiovascular, and other diseases that are not caused by single entities such as viruses or bacteria, but depend on a constellation of possible causes, none either necessary or sufficient. Laboratory and clinical studies have been unable to determine specific causal mechanism for such diseases. In this regard, Doll and Peto, arguably the most prominent epidemiologists today, have concluded that:

> 'Epidemiological observations … have serious disadvantages … They can seldom be made according to the strict requirements of experimental science and therefore may be open to a variety of interpretations. A particular factor may be associated with some disease merely because of its association with some other factor that causes the disease, or the association may be an artefact due to some systematic bias in the information collection
>
> 'It is commonly, but mistakenly, supposed that multiple regression, logistic regression, or various forms of standardization can routinely be used to answer the question: "Is the correlation of exposure (E) with disease (D) due merely to a common correlation of both with some confounding factor (or factors)?"
>
> 'Moreover, it is obvious that multiple regressions cannot correct for important variables that have not been recorded at all … These disadvantages limit the value of observations in humans, but … until we know exactly how cancer is caused and how some factors are able to modify the effects of others, the need to observe imaginatively what actually happens to various different categories of people will remain.'
>
> *Doll R, Peto R, The Causes of Cancer, JNCI 66:1192-1312, 1981, page 1281*

(The 'multiple regression' and 'logical regression' referred to in the quotes above are techniques used in the statistics of epidemiology.)

Thus, while epidemiologists insist that their discipline is a science, clearly it is not a mainstream experimental science that produces reliable causal connections that could be repeatedly tested and yield the same results. A brief inquiry into how studies of multifactorial epidemiology are conducted makes it clear why they do not conform to a scientific framework and why the evidence produced cannot be other than judgemental.

RISK MEASUREMENT – TWO TYPES OF STUDY

EPIDEMIOLOGIC risks in general are estimated from observing differences in the frequency with which diseases appear (incidence) among groups more, or less, exposed to whatever agent the researchers are studying. Various types of studies are used in epidemiology but only two have been used in the case of ETS. Let's briefly examine how these studies are conducted, and what they actually measure.

1. Retrospective cohort (or longitudinal) studies
These studies record different individual recalls of disease incidence in groups of people possibly exposed to ETS to varying degrees during the previous course of their lifetimes. In such studies, risk is estimated from differences of incidence in relation to differences in ETS exposure. Only a handful of such studies have been performed with regard to ETS.

2. Case-control studies
These constitute by far the majority of ETS studies. They record different individual recalls of possible lifetime ETS exposure in two groups of people. One of these groups is composed exclusively of subjects all having the disease under study (lung cancer, for instance): this group is called the 'cases'. The other is composed of subjects who are all free of the disease under study: this group is called the 'controls'. In case-control studies the incidence is 0% in the controls and 100% in the cases. Therefore, a key understanding is that in such studies risks are conjectured as differentials of exposure recall. Increased risk is inferred if exposure is found to be higher among cases, and protection is inferred if exposure is found to be higher among controls.

Note that the term 'individual recall' means the recollections of individual people concerning the phenomenon that the researchers are interested in. In other words, researchers in these studies use people's memories to guess the actual amount of second-hand smoke that they were exposed to, and the comparison of the case and control groups is based on this recollection. Obviously this fact alone is a considerable 'wild card' when it comes to the reliability of the basic data upon which the study depends.

RISK CALCULATION

THE arithmetic of risk calculation is the same for cohort and case-control studies, except for a difference in terminology. The ratio used for risk calculation is called RR (relative risk). The case-control studies assume risk from differentials of exposure. Thus the relative risk (RR) can be indicated by dividing the estimated exposure of cases by the estimated exposure of controls.

In the above ratio, if the exposure is the same in exposed and non-exposed subjects the RR =1.0 and there is no inference of difference in risk. If RR is greater than 1.0 there is

an inference of increased risk in the cases. If RR is smaller than 1.0 there is an inference of decreased risk for the cases, suggesting that exposure may possibly protect people against the disease under scrutiny.

CORRUPTING INFLUENCES

BIASES are common. In simple terms a bias is a type of error that alters the base of comparison in the study to some extent and thereby effects the result. When we want to compare two groups of people in an effort to get information about the possible effect of a particular factor upon them, we must start by comparing 'apples with apples' as much as possible.

A selection bias occurs when control subjects mismatch the case subjects with regard to characteristics that cannot be adjusted for age, gender, etc. In fact, selection bias can only be reduced, for it is impossible to eliminate. Its presence can only be guessed but not measured with any precision.

Information bias relates to inevitable inaccuracies in data collection. Recall bias – that is, inaccurate data resulting from people's inaccurate memories – is most frequent and is of special concern in case-control studies where cases with a disease are apt to 'recall' more intense and longer exposures than the controls without the disease, thus contributing to a false appearance of increased risk. Recall bias and error may be increased when exposure information is retrieved from next of kin of deceased subjects.

In general recall data are based exclusively on vague individual statements that cannot be verified. It is only natural that people with cancer or other diseases would be more inclined than people without disease to blame ETS exposure, in an effort to rationalise their disease and explain it.

Thus, no direct measurements are performed to determine the real level of exposure, nor are they possible. So-called measures of exposure are obtained simply by asking people how many cigarettes a day were smoked by smokers in their households over a lifetime. The persons interviewed might have been children at the time in question and the questions may concern events or impressions dating back decades – as much as 60 years. The inevitably vague answers obtained are then reported as specific numbers – numbers which, while totally unreliable, are then taken and manipulated statistically as if they were confirmed reality.

MISCLASSIFICATION BIAS

MISCLASSIFICATION bias occurs when subjects wrongly declare themselves to be non-smokers and are mistakenly classified as such. The tendency to cheat and misclassify themselves as non-smokers would be naturally more prevalent among subjects with cancer than in control subjects that are healthy, thus contributing to a false impression of elevated risk.

Confounders are defined as hidden risk factors that could also influence the RR. Study subjects with cancer must have been more exposed to other cancer risk factors than the

healthy controls – and there are many risks for lung cancer that studies in general do not bother to control, and cannot be accurately allowed for.

For instance, ETS studies dealing with lung cancer should consider some three dozen risk factors as potential confounders reported in the literature. (For a list of other factors associated with lung cancer see Table 2, page 43). It should be apparent that without a credible control for at least all major known confounders, epidemiologic studies of ETS cannot be validly interpreted.

APPLES, ORANGES AND ABSURDITY

IN THE particular instance of case control studies (by far the majority of ETS studies), the epidemiologic reports indicate that control subjects without lung cancer may recall exposure to ETS at a base rate of 100%, and that the cases with lung cancer may recall exposure at a rate of 115%. Thus, a claim of causality requires the incongruous assumption that the exposure of controls without cancer does not lead to cancer, and that a claimed 15% excess of that exposure is the cause of lung cancer in all the cases.

Not only have we compared apples to oranges by comparing people with cancer of unknown origin exposed to ETS with people without cancer and also exposed to ETS, but we can even express this absurdity with a theorem of appropriate name – theorem of the absurd: *The exposure to 100% ETS does not cause cancer because the controls are all healthy. The exposure to 115% ETS is an absolute and sufficient cause of cancer, since all the cases selected had cancer!*

Large as they might be, epidemiologic surveys sample only a small fraction of a population and therefore incur a statistical error of measurement. This means that a given risk estimate is not precise but may be plus or minus a certain degree of error. In other words, the true estimate may lie in the interval *between* the higher and lower figures that the error comports.

Given that such error is inevitable, the question arises of how much statistical error is tolerable. In what clearly amounts to an arbitrary judgement call, a standard consensus has been adopted that no more than a 5% margin of statistical error is tolerable. The complementary implication is that no less than a 95% probability of statistical certainty is acceptable. By this convention, statistical results that display less than a 5% margin of error are said to be statistically significant at the 95% level, whereas if the error exceeds 5% the results are said to be not statistically significant.

STATISTICAL SIGNIFICANCE

IN INTERPRETING a confidence interval it is important to recall how risk ratios are calculated. A value of 1.0 means no change in risk because exposure is the same in cases and controls. Values below 1.0 imply risk reduction, or protection. Values above 1.0 imply increased risk. The 95% confidence interval gives an immediate impression of statistical significance, which is characterised by confidence intervals whose values are either above or below 1.0. Intervals containing values above and below 1.0 define non-significant results because they simultaneously imply an increase and a decrease of risk.

Detailed accounts of epidemiologic methodologies for dealing with biases, confounders, standardisations, and statistics can be found in textbooks. Still, it should be useful to acquire a perspective on statistical significance. The 5% convention is equivalent to a 1 in 20 threshold of acceptable error, which would be disastrous in most everyday activities. Would it be sensible to drive a car if one time in 20 the brakes failed?

An added warning is that statistical error and certainty are both figures of probability that refer to the numerical context of the data available, but have nothing to say about the reliability of the data themselves. For instance, statistics cannot know or determine whether ETS exposure recall data are credible or not, nor whether biases or confounders may be present. Statistics is blind to whatever influences may be corrupting the underlying study data.

THE DUBIOUS MAGIC OF META-ANALYSIS

META-ANALYSIS is a statistical technique used to pool results from different studies. Originally it was developed for summarising the results of homogeneous randomised clinical trials, a use that remains its legitimate application. However, using meta-analysis for pooling the results of diverse observational ETS studies of contrasting outcomes is fraught with irresolvable difficulties.

The procedure gives different weights to studies, primarily in relation to their size. However, meta-analysis does not pool the discrete data that originated each result, but only the final results of each study regardless of whether concordant or discordant, credible or not. The procedure does not discriminate for characteristics of each study, such as design, data collection, standardisations, biases, confounders, adjustments, statistical procedures, etc.

Meta-analysis, therefore, produces only a weighted average of the final numerical results of the studies, but does not standardise, relieve, or control for differential corruptions that may be present in each study. If characteristics other than study size are used in weighing studies (eg an estimate of study quality), those characteristics are likely discretionary, judgemental, and conducive to different meta-analysis results at the hands of different analysts.

Therefore, with the exception of its use for summarising homogeneous randomised clinical studies, it is abundantly clear that meta-analysis can be used as a stratagem to conjure meaning from studies that have no apparent meaning.

EPIDEMIOLOGY – FUZZY LOGIC

MORE importantly there is a general but crucial warning in reading and interpreting epidemiologic reports. Numerical displays in epidemiology should be seen as having 'an analogue rather than digital' meaning. Most numbers in epidemiology are metaphorical proxies of uncertain real quantities, for epidemiology rarely measures reliably, and more commonly evokes, conceives, assesses, sizes up, adjusts, rounds up, and appraises.

Indeed, numerical transformations and renditions impart an undeserved sense of accuracy

and credibility to a background of vagueness caused by study design deficiencies, asymmetries in data collection, statistical error, biases, confounders, limitations of adjustments and standardisations, prejudice, and more.

As a further cautionary note, the greater the complexity of the statistical analysis in epidemiologic reports, the greater the weakness of the data is likely to be. In a practice known as 'data dredging', epidemiologists like to squeeze every conceivable signal from the data that has been amassed.

EPIDEMIOLOGISTS AND UNCERTAINTY

EPIDEMIOLOGISTS have reacted to the inherent uncertainty of their findings by adopting a vague set of causality criteria, known as the Hill criteria. However, none of the ETS studies, alone or together, come even close to satisfying even this vague set of criteria. Strength of an association is a clue to causation, although a strong association is neither necessary nor sufficient to *affirm* causality, and a weak one is neither necessary nor sufficient to *deny* causality. In the case of the ETS studies it is obvious that the associations claimed are extremely weak, as we can see from the following quotes:

National Cancer Institute
'In epidemiologic research, relative risks of less than 2.0 are considered small and usually difficult to interpret. Such increases may be due to chance, statistical bias or effects of confounding factors that are sometimes not evident.' National Cancer Institute, *Abortion and possible risk for breast cancer: analysis and inconsistencies,* October 26, 1994

Sir Richard Doll
'When (we are dealing with) relative risk lies between 1.0 and 2.0 … 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.' *The Causes of Cancer* by Richard Doll, F.R.S. and Richard Peto. Oxford-New York, Oxford University Press, 1981, page 1219

WHO/IARC
'Relative risks of less than 2.0 may readily reflect some unperceived bias or confounding factor, those over 5.0 are unlikely to do so.' Breslow and Day, 1980, *Statistical methods in cancer research, Vol. 1, The analysis of case control studies*. Published by the World Health Organization, International Agency for Research on Cancer, Sci. Pub. No. 32, Lyon

Food and Drug Administration (FDA)
'My basic rule is if the relative risk isn't at least 3.0 or 4.0, forget it.' Robert Temple, director of drug evaluation at the Food and Drug Administration

Food and Drug Administration (FDA)
'Relative risks of 2.0 have a history of unreliability.' Robert Temple, MD *FDA Journal of the American Medical Association* (JAMA), Letters, September 8, 1999

PART THREE

the evidence on ETS

PART TWO highlighted the critical benchmarks for evaluating the validity of epidemiologic studies on environmental tobacco smoke. These are summarised as follows:

- A study must warrant that its numerical representations of individual lifetime ETS exposure recalls are true measures of actual exposures
- A study must warrant that an exposure recall bias affects at the same rate cases and control groups, and exposed and non-exposed groups
- A study must warrant that subject selection and misclassification biases are affecting at the same rate cases and control groups, and exposed and non-exposed groups
- A study must warrant that known causal confounders are affecting at the same rate cases and control groups, and exposed and non-exposed groups
- A study must warrant the accuracy of pathological and diagnostic records
- The results from different studies addressing the same subject must be consistently reproducible
- In any study the statistical margin of error of reported risks should reach no less than the 95% level of significance
- If the above criteria are met the results of a study should be consistent with Hill's criteria of causality
- Meta-analysis summations shall not be credible unless performed on the basis of all available studies, which must be of homogenous design and conduct and must have met the above criteria of validity

A detailed consideration of each individual study of ETS and lung cancer would make this report inaccessible to non-specialists. For the sake of brevity and clarity we focus here on the epidemiologic studies of ETS and lung cancer which their sponsors claim to represent the best and strongest evidence of the risks of ETS exposure. On this basis, a consideration of the reliability of claims for other conditions (eg heart disease) allegedly linked with ETS exposure would be subordinate to the considerations for lung cancer.

As an overall summary, here listed are all the studies available to 2004 with regard to ETS exposure and lung cancer risk, classified in three categories:

SPOUSAL STUDIES: Non-smoking spouses living with smoking husbands, wives or partners.
WORKPLACE STUDIES: Non-smokers working in occupational settings where smoking is allowed.
CHILDHOOD STUDIES: Non-smokers exposed to ETS in their homes during childhood-adolescence.

TABLE 1 **STUDIES ON PASSIVE SMOKE AND LUNG CANCER**

KEY

Not statistically significant risk elevation

Not statistically significant risk reduction (protection)

Statistically significant risk elevation

Statistically significant risk reduction (protection)

CA97 Figures from Final Report CALEPA 1997

CA03 Figures from Final Report CALEPA 2003

SGR 1986 Surgeon General's Report

+/– Estimated

NR No Risk. Reported as having no correlation, namely RR = 1.0

SPOUSAL STUDIES

Studies & Authors	Year	Nation	Sex	Number of lung cancers	Relative Risk ie 1.18=18% risk elevation	95% Confidence Interval
Garfinkel et al. 1 SGR	81	United States	F	153	1.18	0.90-1.54
Chan et al. SGR	82	Hong Kong	F	84	0.8	0.43-1.3
Correa et al. SGR	83	United States	F	22	2.07	0.81-5.25
Correa et al. SGR	83	United States	M	8	1.97	0.38-10.32
Trichopouls et al. SGR	83	Greece	F	77	2.08	1.20-3.59
Buffler et al.	84	United States	F	41	0.8	0.34-1.9
Buffler et al.	84	United States	M	11	0.51	0.14-1.79
Hirayama et al. SGR	84	Japan	F	200	1.6	1.00-2.4
Hirayama et al. SG	84	Japan	M	64	2.24	1.19-4.22
Kabat et al. 1SGR	84	United States	F	24	0.79	0.25-2.45
Kabat et al. 1SGR	84	United States	M	12	N R	0.2-5.07
Garfinkel et al. 2SGR	85	United States	F	134	1.23	0.81-1.87
Lam W. et al.	85	Hong Kong	F	60	2.01	1.09-3.72
Wu et al. SGR	85	United States	F	29	1.4	0.4-4.2
Akiba et al. SGR	86	Japan	F	94	1.5	0.9-2.8
Akiba et al. SGR	86	Japan	M	428	1.8	0.4-7.0
Lee et al. SGR	86	United Kingdom	F	32	N R	0.37-2.71
Lee et al. SGR	86	United Kingdom	M	15	1.3	0.38-4.39
Bownson et al. 1	87	United States	F	19	1.68	0.39-6.9
Gao et al.	87	China	F	246	1.19	0.82-1.73
Humble et al.	87	United States	F	20	2.2	0.80-6.6
Humble et al.	87	United States	M	8	4.82	0.63-36.56
Koo et al.	87	Hong Kong	F	86	1.64	0.87-3.09

SPOUSAL STUDIES

Studies & Authors	Year	Nation	Sex	Number of lung cancers	Relative Risk ie 1.18=18% risk elevation	95% Confidence Interval
Lam Tet al.	87	Hong Kong	F	199	1.65	1.16-2.35
Pershagen et al. SGR	87	Sweden	F	70	1.2	0.7-2.1
Butler et al.	88	United States	F	8	2.2	0.48-8.56
Geng et al.	88	China	F	54	2.16	1.08-4.29
Inoue et al.	88	Japan	F	22	2.25	0.8-8.8
Shimizu et al.	88	Japan	F	90	1.08	0.64-1.82
Choi et al.	89	Korea	F	75	1.63	0.92-2.87
Choi et al.	89	Korea	M	13	2.73	0.49-15.21
Hole et al.	89	Scotland	F	6	1.89	0.22-16.12
Hole et al.	89	Scotland	M	13	3.52	0.32-38.65
Svensson et al.	89	Sweden	F	34	1.26	0.57-2.81
Janerick et al.	90	United States	F&M	191	0.93	0.55-1.57
Kalandidi et al.	90	Greece	F	90	2.11	1.09-4.08
Sobue et al.	90	Japan	F	144	1.13	0.78-1.63
Wu-Williams	90	China	F	417	0.7	0.60-0.9
Liu Z et al.	91	China	F	54	0.77	0.30-1.96
Brownson et al. 2 CA97	92	United States	F	431	NR	0.80-1.2
Stockwell et al. CA97	92	United States	F	62	1.6	0.80-3.0
Liu Q et al. CA97	93	China	F	38	1.66	0.73-3.78
Wu et al.	93	China	F	75	1.09	0.64-1.85
Fontham et al. CA97	94	United States	F	651	1.29	1.04-1.60
Layard et al.	94	United States	F	39	0.58	0.30-1.13
Layard et al.	94	United States	M	21	1.47	0.55-3.94
Zaridze et al.	94	Russia	F	162	1.66	1.12-2.46
Du et al.	95-96a	China	F	69	1.19	0.66-2.16
Kabat et al. 2 CA97	95	United States	F	67	1.08	0.60-1.94
Kabat et al. 2 CA97	95	United States	M	39	1.6	0.67-3.82
Wang et al.	96a	China	F	99	2.5	1.3-5.1
Wang et al.	96b	China	F	92	1.11	0.65-1.88
Schwartz et al. CA97	96	United States	F	175	1.1	0.72-1.68
Schwartz et al. CA97	96	United States	M	72	1.1	0.60-2.03
Sun et al.	96	China	F	230	1.16	0.80-1.69
Want SY et al.	96	China	F	82	2.53	1.26-5.10
Wang TJ et al.	96	China	F	135	1.11	0.67-1.84
Cardenas et al. CA97 CA03	97	United States	F	150	1.2	0.80-1.6
Cardenas et al. CA97 CA03	97	United States	M	97	1.1	0.60-1.8
Jöckel-BIPS CA03	97	Germany	F	53	1.58	0.74-3.38

SPOUSAL STUDIES

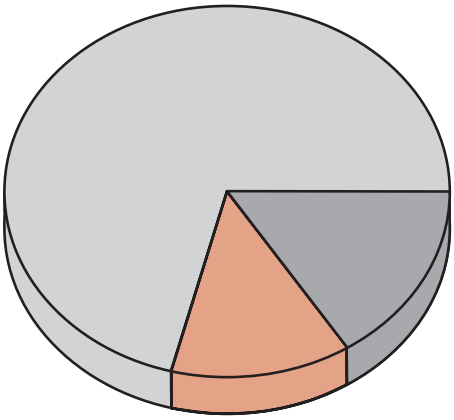
Studies & Authors	Year	Nation	Sex	Number of lung cancers	RelativeRisk ie. 1.18=18% risk elevation	95% Confidence Interval
Jöckel-BIPS CA03	97	Germany	M	18	1.58	0.52-4.81
Jöckel-GSF CA03	97	Germany	F	242	0.93	0.66-1.31
Jöckel-GSF CA03	97	Germany	M	62	0.93	0.52-1.67
Ko et al. CA97 CA03	97	Thailand	F	105	1.3	0.7-2.5
Nyberg et al. CA03	97	Sweden	F	89	1.2	0.74-1.94
Nyberg et al. CA03	97	Sweden	M	35	1.2	0.57-2.55
Jockel et al. CA03	98	Germany	F&M	71	1.12	0.54-2.32
Nyberg et al. CA03	98a	Sweden	F	89	1.05	0.6-1.86
Nyberg et al. CA03	98a	Sweden	F&M	58	1.17	0.73-1.88
Boffetta et al. CA03	98	Europe	F	508	1.15	0.86-1.55
Boffetta et al. CA03	98	Sweden	F&M	70	2.29	0.65-8.07
Boffetta et al. CA03	98	Germany 1	F&M	76	0.88	0.40-1.95
Boffetta et al. CA03	98	Germany 2	§F&M	142	1.22	0.66-2.2
Boffetta et al. CA03	98	Germany 3	F&M	31	2.01	0.71-5.67
Boffetta et al. CA03	98	England	F&M	26	1.38	0.43-4.28
Boffetta et al. CA03	98	France	F&M	77	0.72	0.36-1.25
Boffetta et al. CA03	98	Portugal 1	F&M	49	2.04	0.71-5.8
Boffetta et al. CA03	98	Portugal 2	F&M	33	2.03	0.76-5.38
Boffetta et al. CA03	98	Spain	F&M	71	1.1	0.48-2.68
Boffetta et al. CA03	98	Italy 1	F&M	40	0.73	0.28-1.65
Boffetta et al. CA03	98	Italy 2	F&M	19	1.12	0.35-3.56
Boffetta et al. CA03	98	Italy 3	F&M	16	1.36	0.30-6.45
Zaridze et al. CA03	98	Russia	F	189	1.53	1.06-2.21
Jee et al. CA03	99	Korea	F	79	1.9	1.0-3.5
Rapiti et al. CA03	99	India	F	52	1.2	0.5-2.9
Zhong et al. CA03	99	China	F	504	1.1	0.7-1.7
Lee et al. CA03	00	Taiwan	F	186	1.2	0.7-2.0
Wang et al. CA03	00	China	F&M	200	1.19	0.7-2.0
Kreuzer at al. CA03	00/01	Germany	F	234	0.96	0.7-1.33
Kreuzer at al.	00/01	Germany	F&M	292	0.99	0.73-1.34
Johnson et al. CA03	01	Canada	F	56	1.2	0.5-3.0
Nishino et al. CA03	01	Japan	F	23	1.8	0.67-4.6

WORKPLACE STUDIES

Studies & Authors	Year	Nation	Sex	RelativeRisk ie. 1.18=18% risk elevation	95% Confidence Interval
Kabat et al. 1 CA97	84	United States	F	0.70	0.30-1.50
Kabat 1 et al. CA97	84	United States	M	3.3	1.1-10.4
Garfinkel 2 CA97	85	United States	F	0.93	0.7-1.2
Wu et al. CA97	85	United States	F	1.3	0.5-3.3
Lee et al. CA97	86	United Kingdom	F	0.63	0.17-2.33
Lee et al. CA97	86	United Kingdom	M	1.61	0.39-6.6
Koo et al. CA97	87	Hong Kong	F	0.91	0.15-5.37
Shimizu et al. CA97	88	Japan	F	1.18	0.70-2.01
Janerich et al. CA97	90	United States	F&M	0.91	0.80-1.04
Kalandidi et al. CA97	90	Greece	F	1.39	0.80-2.5
Wu-Williams et al. CA97	90	China	F	1.2	0.90-1.6
Brownson et al. 2	92	United States	F	0.79	0.61-1.03
Stockwell et al. CA97	92	United States	F	NR	N S
Fontham et al. CA97	94	United States	F	1.39	1.11-1.74
Zaridze et al.	94	Russia	F	1.23	0.74-2.06
Kabat et al. 2 CA97	95	United States	F	1.15	0.62-2.13
Kabat et al. 2 CA97	95	United States	M	1.02	0.5-2.09
Schwartz et al. CA97	96	United States	F&M	1.5	1.0-2.2
Sun et al.	96	China	F	1.38	0.94-2.04
Wang et al.	96a	China	F	2.0	p=0.05
Wang et al.	96b	China	F	0.89	0.45-1.77
Jockel-BIPS CA03	97	Germany	F&M	2.37	1.02-5.48
Jockel-GSF CA03	97	Germany	F&M	1.51	0.95-2.4
Ko et al. CA97 CA03	97	Thailand	F	1.1	0.40-3.0
Nyberg et al. CA03	98a	Sweden	F&M	1.61	0.91-2.85
Zaridze et al. CA03	98	Russia	F	0.88	0.55-1.41
Boffetta et al. (WHO) CA03	98	Europe	F&M	1.17	0.94-1.45
Zhong et al. CA03	99	China	F	1.7	1.30-2.3
Kreuzer et al. CA03	98/00	Germany	F	1.03	0.78-1.36
Lee et al. CA03	00	Taiwan	F	1.2	0.50-2.4
Johnson et al. CA03	01	Canada	F	1.21	0.50-2.8 min.

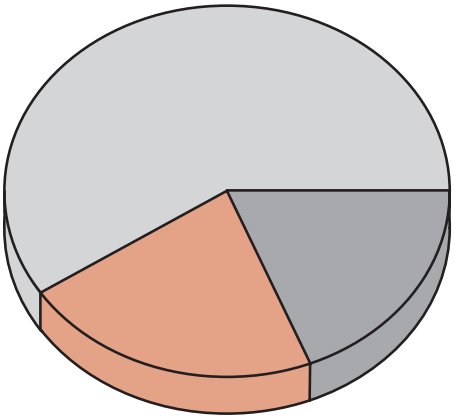
CHILDHOOD STUDIES

Studies & Authors	Year	Nation	Sex	Relative Risk ie 1.18=18% risk elevation	95% Confidence Interval
Correa et al. SG	83	United States	F	NR	N.S
Kabat & Wyn CA97	84	United States	F	0.92	0.40-2.08
Kabat & Wyn CA97	84	United States	M	1.26	0.33-4.83
Garfinkel et al. 2 SG	85	United States	F	0.91	0.74-1.12
Wu et al. SGR	85	United States	F	0.6	0.20-1.7
Akiba et al. SG	86	Japan	F&M	N.R	N.S
Gao et al. CA97	87	China	F	1.1	0.7-1.7
Koo et al. CA97	87	Hong Kong	F	1.73	0.6-6.4
Pershagen et al. CA97	87	Sweden	F	N.R	0.4-2.3
Svensson et al. CA97	89	Sweden	F	3.3	0.5-18.8
Janerich et al. CA97	90	United States	F&M	1.09	0.68-1.73
Sobue et al. (CA97)	90	Japan	F	1.28	0.71-2.31
Wu-Will et al. (CA97)	90	China	F	N.R	N.S
Brownson et al. 2 CA97	92	United States	F	0.8	0.60-1.1
Stockwell et al. CA97	92	United States	F	1.1	0.50-2.6
Fontham et al. CA97	94	United States	F	0.89	0.72-1.1
Zaridze et al.	94	Russia	F	0.98	0.66-1.45
Kabat 2 CA97	95	United States	M	0.9	0.43-1.89
Kabat et al. 2 CA97	95	United States	F	1.55	0.95-2.79
Sun et al.	96	China	F	2.29	1.56-3.37
Wang et al.	96a	China	F	1.91	p.=0.01
Wang et al.	96	China	F	0.91	0.56-1.49
Jockel-BIPS CA03	97	Germany	F&M	1.05	0.50-2.22
Jockel-GSF CA03	97	Germany	F	0.95	0.64-1.4
Ko et al. CA97 CA03	97	Thailand	F	0.80	0.4-1.6
Boffetta et al. (WHO) CA03	98	Europe	F&M	0.78	0.64-0.96
Jockel et al. CA03	98	Germany	F&M	2.02	0.60-6.75
Nyberg et al. CA03	98a	Sweden	F&M	1.02	0.63-1.66/F
Nyberg et al. CA03	98a	Sweden	F&M	0.72	0.28-1.87/M
Zhong et al. CA03	99	China	F	0.9	0.50-1.9
Rapiti et al. CA03	99	India	F	3.99	1.90-8.2
Kreuzer et al. CA03	98/00	Germany	F	1.03	0.78-1.36
Lee et al. CA03	00	Taiwan	M	1.7	1.10-2.6/F
			F	0.9	0.30-3.1/M
Wang et al. CA03	00	China	F&M	1.52	1.1-2.2
Rachtanet al. CA03	01	Poland	F	3.31	1.26-8.69
Johnson et al. CA03	01	Canada	F	0.54	0.1-2.7



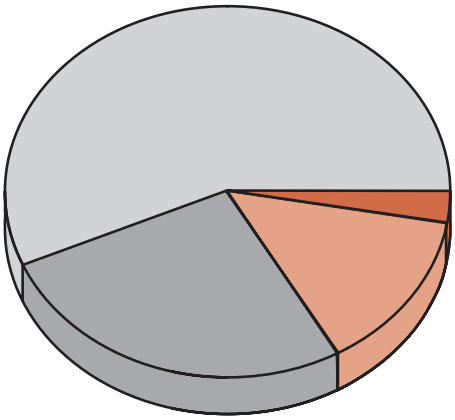
SPOUSAL STUDIES

71.3%	Statistically insignificant risk elevation:	57 studies
12.5%	Statistically insignificant risk reduction:	10 studies
16.2%	Statistically significant risk elevation:	13 studies
	TOTAL	**80 studies**



WORKPLACE STUDIES

58%	Statistically insignificant risk elevation:	57 studies
22.7%	Statistically insignificant risk reduction:	10 studies
19.3%	Statistically significant risk elevation:	13 studies
	TOTAL	**31 studies**



CHILDHOOD STUDIES

55.6%	Statistically insignificant risk elevation:	20 studies
27.8%	Statistically insignificant risk reduction:	10 studies
13.9%	Statistically significant risk elevation:	5 studies
2.7%	Statistically significant risk reduction:	1 study
	TOTAL	**36 studies**

CONCLUSION

IN CONSIDERING the tables and charts on the previous pages it is necessary to mention that a publication bias has been ascertained, but not measured, whereby it is certain that studies purporting a positive association, statistically significant or not, are more likely to have been published than studies reporting a null or negative association.

The flimsiness of the alleged risk reported, the manifest lack of consistency of results from different studies, and the failure of statistical significance, should have been a clear warning – since the very first attempts – that such studies of ETS exposures could not possibly reach verifiable conclusions. Instead, a dogged determination to fabricate risks at any cost has produced a remarkable sequence of largely impotent studies. Hence:

- for spousal studies, 83.8% of the results are not statistically significant and could mean either an elevation or a reduction of risk under universally accepted standards of statistical significance;

- for workplace studies, 80.7% of the results are not statistically significant and could mean either an elevation or a reduction of risk under universally accepted standards of statistical significance;

- for childhood studies, 83.4% of the results are not statistically significant and could mean either an elevation or a reduction of risk under universally accepted standards of statistical significance.

We have previously seen in Part One that the 95% level of statistical significance is a very lax standard that accepts a 1 in 20 chance of error. In the case of epidemiologic studies of ETS and lung cancer, that error is compounded by the discrepancy of the results of the studies.

A fair appraisal leads directly to some questions, the answers to which depend on whether a person is a blind follower of received dogma (ie 'passive smoking kills'), or one who still values self determination and the transparency of evidence. The statistical significance of the studies is abysmal. The results of different studies are wholly inconsistent. Would anyone accept to drive a car the brakes of which are guaranteed to fail at least four times in five when applied?

Likewise is it rationally defensible or legitimate for governments in democratic societies to use such data to raise false public anxieties and to enforce draconian policies that victimise millions of citizens, and trample on and restrict their recognised freedoms?

APPENDIX I

risk factors for lung cancer independent of smoking

EVEN IF one were to use the improper statistical and epidemiologic distortions introduced by the US Environmental Protection Agency to dream up a conclusion about the lung cancer risk of ETS (USEPA, 1992), the currently available studies on this topic could not conjure up a relative risk greater than 1.05, namely an incongruous 5% elevation (Gori and Luik, 1999). Assuming for sake of argument that such an assessment was technically correct, it would be 20 to 300 times smaller as compared respectively to the lowest and highest risk factors here listed.

It is undeniable, therefore, that even a slight confounding by any or many of the confounders could have a much greater impact in any study of ETS and lung cancer than the possible impact of ETS itself. Yet the ETS and lung cancer studies so far published have accounted erratically for no more than a handful of the confounders here listed, thus making very difficult any interpretation of the possible association of ETS exposure and lung cancer.

HOW TO READ THE TABLE OPPOSITE

THE FIRST column in Table 2 describes the nature of the risk factors investigated. The second column gives the name of the first author of each study that has reported separately on the risk factor listed on the first column to the left.

The third column lists the most likely and best estimate of the highest risk reported by each study. The risks are given as relative risks, namely relative to the risk of people NOT exposed to the risk factor under study.

The fourth and last column gives the 95% confidence interval for each risk listed. The interval is a measure of the statistical uncertainty of the risk values listed in the third column, and indicates that the true value of the risk may be anywhere between the low and high figures in the fourth column, with a 1 in 20 probability of error.

TABLE 2 SOME WIDELY VARIED FACTORS FOR LUNG CANCER INDEPENDENT OF CIGARETTE SMOKING

Factor	Reference	Reported Relative Risk at Highest Exposure	95% Confidence Interval
Family history of lung cancer	Samet (1986)	5.3	(2.2-12.8)
	Ooi (1986)	2.4	
	Horwitz (1988)	2.8	(1.0-7.7)
	Wu (1988)	3.9	(2.0-7.6)
	Brownson (1997)	2.7	(1.2-6.1)
Personal history of tuberculosis	Hinds (1982)	10.0	(1.1-90.1)
	Gao (1987)	6.4	
	Wu (1988)	1.7	(1.1-2.4)
	Sakurai (1989)	8.2	(1.3-54.4)
ß-carotene/Vitamin A deficiency	Ziegler (1986)	2.2	
ß-carotene/Vitamin A intake	Wu (1985)	0.3	(P=0.06 trend)
	Byers (1987)	0.2	
	Pastorino (1987)	0.4	(0.2-0.9)
	Candelora (1992)	0.4	(0.2-0.8)
Alcohol intake	Pollack (1984)	2.19	(1.3-5.0)
Dietary cholesterol/fat	Goodman (1988)	2.2	(1.3-3.8)
Dietary fat intake	Wynder (1987)	4-6	
	Alavanja (1993)	6.14	(2.63-14.40)
	De Stefani (1997)	2.85	(1.73-4.69)
Pork meat intake	Mettlin (1989)	2.4	(1.4-4.2)
Vegetable diet	Le Marchand (1989)	0.6	(0.4-0.88)
	Jain (1990)	0.3	(P=0.009 trend)
	Candelora (1992)	0.2	(0.1-0.5)
	Alavanja (1993)	0.61	(0.37-0.99)
	Axelsson (1996)	0.37	(0.23-0.61)
	Sankaranarayanan	0.32	(0.13-0.78) (1994)
Fruit intake	Koo (1988)	0.4	(0.2-0.9)
	Candelora (1992)	0.6	(0.3-1.1)
Milk intake	Mettlin (1989)	2.1	(1.4-3.2)
	Rylander (1996)	1.73	(1.0-3.01)
	Axelsson (1996)	1.73	(1.0 3.01)
Hormone therapy in women	Adami (1989)	1.3	
Radon	Edlin (1984)	4.3	(1.7 10.6)
	Lees (1987)	2.4	(0.8 7.1)

Continued ...

TABLE 2 Continued

Factor	Reference	Reported Relative Risk at Highest Exposure	95% Confidence Interval
Cooking methods	Gao (1987)	1.4-2.6	(1.1-5.0)
	Mumford (1987)	5.6	(3.4-9.1)
	Geng (1988)	1.9	(1.1-3.3)
	Sobue (1990)	2-3	
	Ko (1997)	8.3	(3.1-22.7)
Motor exhaust exposure	Hayes (1989)	1.5	(1.2-1.9)
	Jacobsson (1997)	2.0	(1.5-2.6)
	Gustavsson (1990)	2.4	(1.3-4.5)
Socio economic class	Brown (1975)	2.6-3.8	
Ventilatory function	Lange (1990)	2-4	
Cardiac anomalies	Tenkanen (1987)	2.4	
Physical inactivity	Albanes (1989)	1.6	(1.2-3.5)
Severson (1989)	1.4	(1.0-2.1)	
Psychosocial traits	Kulessa (1989)	2-3	
	Knekt (1996)	3.32	(1.53-7.20)
Urban/rural risk ratio	Shy (1984)	1.2-2.8	
Arsenic ingestion	Tsuda (1995)	15.69	(7.38-31.02)
Vitamin E	Yong (1997)	0.36	(0.16-0.83)
High education	van Loon (1997)	0.53	(0.34-0.82)
Vitamin A,C and E intake	Yong (1997)	0.32	(0.14-0.74)
Vegetables & fruit intake	Agudo (1997)	0.45	(0.22-0.91)
Asbestos exposure	Oksa (1997)	10.0	(6.9-14.0)
	Zhu & Wang (1993)	5.32	
	Dement (1994)	2.3	(1.88-2.79)
	Raffin (1993)	3.31	
Physical activity	Thune (1997)	0.39	(0.18-0.85)
	Lee (1994)	0.39	(0.18-0.85)

APPENDIX II

ETS, ventilation and everyday risks

ENVIRONMENTAL tobacco smoke isn't clean air – but what is? If you live in an urban industrial society it is very difficult to breathe 'clean air' – after all, we're surrounded by pollutants. However the toxicity of ETS is very small and its components are well below any legal threshold of danger of exposure to its individual components. Of course this is not what the antismoking propaganda wants us to believe. With activists now claiming that 'There is no safe level of environmental tobacco smoke' it naturally follows that the only acceptable level of exposure is zero!

(The claims of anti tobacco 'experts' are often quite risible. James Repace worked for 30 years with the US federal government as a research physicist for the navy and then with the Occupational Safety & Health Administration, as well as the Environmental Protection Agency. Now retired, he has become a highly paid consultant. In March 2004 he claimed that 100,000 air changes per hour (the equivalent of a 'tornado') would be necessary to bring ETS down to an unspecified 'safe level of contamination'. That was quite a jump from the 34,000 air changes per hour he claimed were necessary on 24 January of the same year!)

The removal of any safety threshold is one of the several core problems of what has come to be known as 'junk science'. The refusal to consider safety thresholds implies, at the extreme, that what is poisonous at tremendous dosage is also toxic at infinitesimal dosage. If such an assumption were true, we would all be in mortal danger because of the presence of the toxins in our bodies, some of which are even essential to life in extremely tiny amounts – arsenic, for example.

Good ventilation, properly maintained, can easily create a comfortable environment that removes not just passive smoke but also, and especially, the potentially serious contaminants that are independent from smoking, such as viruses and bacteria. Before examining ventilation, however, we need to look at what passive smoke actually contains (see Table 3 overleaf) and how many cigarettes would have to be smoked in order to reach dangerous levels of pollution in an enclosed environment.

FINE RESPIRABLE PARTICLES IN ETS

IF THE case in Table 3 were to occur in real life the smokers in the room would run out of oxygen long before reaching the limit for methyl chloride. By definition, fine particles (Respirable Suspended Particles or RSP) are dust grains with a calliper of 10 microns or less (PM_{10}, $PM_{2.5}$ … PM = Particulate Matter); a micron is one thousandth of a millimetre. Fine particles come from exhaust emissions, ETS, and from a large number of natural

TABLE 3 **HOW MANY CIGARETTES ARE NEEDED TO REACH DANGEROUS LEVELS OF POLLUTION IN AN ENCLOSED ENVIRONMENT?**

Estimated number of cigarettes required to reach Threshold Limit Value (TLV) levels from side stream smoke emission of selected chemicals in a sealed and unventilated 100m³ enclosure (Gori and Mantel, 1991). Note: 100m³ are equivalent to a room 22 x 21 x 8 feet ceiling, or 3,696 cubic feet.

Side Stream Smoke Component (1)	Side Stream Smoke output mg/cigarette (2)	Safety Limit mg/m³ (3)	Cigarettes required to reach safety limit (4)
Methyl chloride	0.88	0.30	1,170
Hydroquinone	0.16	2.00	1,250
Cadmium	0.0007	0.001	1,430
Acetaldehyde	1.26	180.00	1,430
Acetic Acid	1.50	25.00	1,660
Nitrogen oxides	2.80	50.00	1,780
Phormic Acid	0.525	9.40	1,790
Pyridine	0.39	16.00	4,100
Phenol	0.25	19.00	7,600
Methylamine	0.1	13.00	13,000
Benzene	0.24	32.00	13,300
Catecol	0.14	23.00	16,500
Nickel	0.0025	1.00	40,000
Diethylamide	0.036	18.00	50,000
Hydrazine	0.00009	0.13	145,000
Acetone	1.00	1,780.00	178,000
Benzo(a)pyrene	0.00009	0.20 [3]	222,000
2-Toluidine	0.003	9.00	300,000
Polonium 210	0.4pCi	3.pCi/l [4]	750,000
Toluene	0.000035	375.00	1,000,000

TLV = Threshold Limit Value. This is the threshold used in the USA, a country with the most restrictive standards for toxic exposure.

(1) Side stream smoke is the smoke from the cigarette released right downstream of the hot tip. That type of smoke is the 'worst', since the chemical catalysis processes that occur a few instants after the smoke leaves the burning tip still have to blend with the surrounding air before it becomes ETS.

But it is very unlikely that non-smokers stand motionless four inches directly above the tip of a cigarette, as the sensor was positioned for these measurements. Thus even the number of cigarettes estimated above to reach the safety limits is understated.

(2) EPA data1990a, Table C-2, pages C-19, 20

(3) Based on the lower exposure limit for tar volatile compounds

(4) EPA 1990b

causes. The theory is that, since they are so small, they can get into the alveoli of the lungs and cause cancer and other disease.

However, fine particles have been with us since the dawn of time, and we have developed strong resistance to them through natural selection. Cavemen burned wood and grease fires in caves to keep warm during the long winter months, and those who had less resistance to fine particles and other pollutants died and thus were prevented from reproducing. Through the eons, resistance to ETS and its fine particles may have been one of the reasons for our survival as a species.

The impact on human health of those particles is still the object of great controversy and indecision by environmental authorities. Recently, even the US Environmental Protection Agency reassessed its estimate on the negative impact of the particulate matter on human health by greatly reducing its assessment of that impact.

If fine particles are as deadly for non-smokers as they want us to believe, *all smokers would die just a few months after picking up the habit* since their exposure to those particles is substantially greater than that of the 'involuntary' smoker – *up to 75,000 times greater*. But we know that the majority of even heavy smokers live to old age. How is that possible? There are only two explanations: either smokers have a special genetic make-up (a ridiculous concept) or the impact on health of fine particles is grossly exaggerated.

Even if we assume a 10-hours-a-day exposure instead of the 1.5 Table 4 refers to (concerning mainly non-smokers exposed occasionally, ie in restaurants), it means that a non-smoking waiter who works in a smoky restaurant for 10 hours each day still inhales 11,250 times *less* fine particles than a 30-cigs-a-day smoker, and 7,500 times less than a pack-a-day (20 cigs) smoker.

TABLE 4 **RELATIVE DOSE ESTIMATE OF RSP IN TYPICAL ACTIVE SMOKERS & PASSIVE SMOKE (ETS) EXPOSED NON-SMOKERS**

Smoker	**Non smoker exposed to passive smoke**
30 cigarettes per day	0.05 mg RSP/cubic meter of air
15 mg RSP inhaled per cigarette	1.5 hours per day exposure
Lung retention efficiency: 90%	0.7 cubic meters per hour inhaled
Daily dose: about 400 mg	Lung retention efficiency: 10%
	Daily dose: about 0.00525 mg
Dose Ratio 0.00525: 400 = about 1:75,000	

From Luik and Gori, 1999

THE VENTILATION SOLUTION

WITH SUCH low level of pollutants it becomes apparent that the biggest problem concerning ETS is the annoyance or 'discomfort' factor. The truth is this can be solved with modern, well-maintained ventilation systems. Experiments performed in the Black Dog Tavern in Scarborough, Ontario, Canada, reported in the study 'Environmental Tobacco Smoke in the Non-Smoking Sections of a Restaurant: A Case Study', demonstrated in an empirical way how a low-budget ventilation system can easily get rid of the annoyance factor with the extra bonus that the air in the area where smoking was free and unrestricted was considerably cleaner than that of no-smoking restaurants.

Likewise, tests in the UK have demonstrated that a good ventilation system can remove up to 90% of the gases and particles from environmental tobacco smoke in a city centre public bar. It is clear that separating smokers from non-smokers (ie smoking and non-smoking areas) combined with decent ventilation offers a perfectly acceptable solution to the 'problem' of ETS. All it needs is for regulatory authorities to set standards for indoor air quality and let technology, with a bit of help from proprietors, do the rest.

Air quality standards are common in industrial and environmental contexts but, to date, no country in the world has set them for smoking areas. It seems clear that the reasons are not scientific, nor are they economic or even technical: they are political. Politicians, like antismoking campaigners, aren't interested in a fair yet practical solution to the 'problem' of ETS. The goal is to ban smoking so that smokers will be forced to quit.

FEARING EVERYDAY RISKS?

IF WE ACCEPT that small risk elevations based on flimsy evidence really constitute a danger for public health then we should literally stop existing to protect our existence. Table 5 (page 53) looks at some examples of statistical risk elevation concerning everyday life. As you can see, the 'risk' of getting lung cancer from passive smoking (RR: 1.5) is minute in comparison to the risk of getting breast cancer from wearing a bra (RR: 12.5) or leukaemia from eating 12 or more hotdogs each month (RR: 9.5) or, more seriously, advanced prostate cancer from eating red meat (RR: 2.6).

Where does it all end? Should we forbid, control and regulate everything? (Incidentally, the risks in Table 5 are calculated using the same 'scientific' methodology used for ETS.) Is a 30% risk elevation a cause for real concern? If an ailment is mono-factorial – known to be caused by one agent whose effect can be precisely measured – the answer may be yes. Where causality is multi-factorial (like lung cancer or heart disease), and the effects of any one agent are in question or cannot be isolated, any measurement of risk is highly unreliable.

In view of this, why should somebody arbitrarily determine that 30% risk elevation from three cups of coffee a week is less dangerous than 30% risk elevation from passive smoking? Perhaps it's time to reclaim the middle ground in the name of common sense.

TABLE 5 EVERYDAY RISKS

Exposure and disease	Relative Risk (RR)	Risk elevation in %
Passive smoke and lung cancer (EPA)	1.19	19%
Passive smoke and lung cancer (IARC)	1.25	25%
Consuming olive oil and breast cancer	1.25	25%
Vasectomy and prostate cancer	1.3	30%
Obesity in women and premature death	1.3	30%
Sedentary job and colon cancer	1.3	30%
Three cups of coffee per week and premature death	1.3	30%
Birth weight of 8+ lbs. and breast cancer	1.3	30%
Baldness in men under 55 and heart attack	1.4	40%
Eating margarine every day and heart disease	1.5	50%
Drinking tap water and miscarriage	1.5	50%
Regular use of mouthwash and mouth cancer	1.5	50%
Abortion and breast cancer	1.5	50%
Eating yogurt and ovarian cancer	2.0	100%
Drinking whole milk and lung cancer	2.14	114%
Obesity in non-smoking women and premature death	2.2	120%
Eating red meat and advanced prostate cancer	2.6	160%
Chlorinated drinking water and bladder cancer	2.0 – 4.0	100% – 300%
Douching and cervical cancer	4	300%
Workplace stress and colorectal cancer	4.5	350%
Eating 12+ hotdogs per month and leukaemia	9.5	850%
Wearing a brassiere all day and breast cancer	12.5	12,000% +

Chart from Science Without Sense – The Risky Business of Public Health Research, Steven Milloy. Cato Institution, 1997, pp 14

APPENDIX III

glossary of terms

BIAS: type of error, sometimes in the basic selection of elements for the study, that distorts the final result

CONFIDENCE INTERVAL: margin of certainty that errors have not been made. In the kinds of studies examined in this report, a 95% confidence interval is considered acceptable

CONFOUNDER, CONCOMITANT FACTOR: factors and circumstances that contribute to the occurrence of a disease which has more than one known and certain cause

DATA DREDGING: sifting of data from existing studies for the purpose of finding associations that may not have been found first time around: often used to create new studies at low cost

EPIDEMIOLOGY: study of the distribution and determinants of health-related states or events in specified populations

ENVIRONMENTAL TOBACCO SMOKE (ETS): smoke dissipated into the air which may be inhaled by bystanders as a result of someone smoking; also known as passive smoke (PS) and secondhand smoke (SHS)

META-ANALYSIS: analysis of a group of existing studies

MONO-FACTORIAL DISEASE: disease with only one known cause, such as polio or tuberculosis

MULTI-FACTORIAL DISEASE: diseases such as cancer, heart disease and stroke that can be caused by different factors acting by themselves or in combination with other factors

RECALL BIAS: bias caused by using people's memories as a source of data

RELATIVE RISK (RR): ratio between the incidence of diseases in a group of people believed to be exposed to the risk and the incidence in another group of people believed not to be exposed to that risk

RETROSPECTIVE STUDY: identifies groups of subjects with different incidences of diseases and attempts to reconstruct their past exposures through memory and recall

STATISTICAL SIGNIFICANCE: numerical expression of the fact that the data of a study shows a risk increase (or decrease). If a study suggests both at the same time it is statistically insignificant

STATISTICALLY SIGNIFICANT: a study is statistically significant when the confidence interval demonstrates either a risk or a benefit. Note: 'statistical significance' does not necessarily mean that the data is accurate nor does it necessarily mean 'significantly large risk'

About FOREST

FOUNDED in 1979 FOREST neither promotes smoking nor do we deny the serious health risks associated with smoking. Instead we defend the interests of adults who choose to smoke; we promote freedom of choice for employers and proprietors who wish to accommodate smokers on their premises; and we speak out against those who want to discriminate against smokers or ban smoking in all public places. Last but not least, we promote greater consideration from smokers towards non-smokers.

Our patron is TV chef and restaurateur Antony Worrall Thompson; our president is Lord Harris of High Cross, former general director of the Institute of Economic Affairs; and our Supporters Council includes artist David Hockney, musician Joe Jackson, inventor Trevor Baylis and Oscar-winning playwright Ronald Harwood.

FOREST spokesmen are regularly quoted in the national and local press and frequently appear on television and radio. In recent years we have been invited to submit written and oral evidence to the Health and Safety Commission (1999), House of Commons Health Select Committee (2000), Greater London Authority Investigative Committee on Smoking in Public Places (2001), the Scottish Parliament Community Care Committee (2002), the Scottish Parliament Health Committee (2004) and the Welsh Assembly Committee on Smoking in Public Places (2004).

Like most smokers FOREST accepts many of the current restrictions on smoking in public places. There is a huge difference however between restrictions and a total ban and our goal is a society that can accommodate those who want to smoke without inconveniencing those who do not want to breathe other people's tobacco smoke.

Specifically, we welcome the introduction of more smoke free areas (together with designated smoking areas), and we positively encourage the installation of better ventilation and modern air filtration systems that will improve air quality for everyone, smokers and non-smokers alike.

For further information see our website which is updated daily with news and information about the smoking debate.

www.forestonline.org

FOREST
Sheraton House
Castle Park
Cambridge CB3 0AX
Telephone 01223 370156
contact@forestonline.org
www.forestonline.org