ETS
FOR DUMMIES II

The complete, updated evidence of the

PASSIVE SMOKE
EPIDEMIOLOGICAL
FRAUD

www.forces.org
P.O. Box 591257 - San Francisco, CA 94159 – USA

FORCES INTERNATIONAL (Forces, Inc.) is an independent, non-profit educational corporation organized under the laws of the Commonwealth of Virginia.

Forces, Inc. has received a charitable tax exemption under Internal Revenue Code 501(c)3.
Your contribution is tax deductible.

"There is a worrying trend in academic medicine which equates statistics with science, and sophistication in quantitative procedures with research excellence. The corollary of this trend is a tendency to look for answers to medical problems from people with expertise in mathematical manipulation and information technology, rather than from people with an understanding of disease and its causes.

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*.

The root of most instances of statistical malpractice is the breaking of mathematical neutrality and the introduction of causal assumptions into the analysis without scientific grounds. This amount to performing science by sleight-of-hand: the quickness of statistics deceives the mind. … Statistical analysis has expanded beyond its legitimate realm of activity."

- B.G. Charlton, MD, University of Newcastle upon Tyne**,** in *Statistical Malpractice*, Journal of the Royal College of Physicians in London, March-April, 1996, pp. 112-114.

GENERAL INDEX

INDEX OF TABLES

PART I

An overview

Nothing works better than a slick slogan, as the advertising industry has always known. That's why, when antismoking campaigners started claiming that "passive smoke kills", they were on to 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 second-hand smoke as a danger because that, they reasoned cynically, would put pressure on smokers to quit or be marginalized.

The only problem with their claim that passive smoke is a danger is this: it just isn't so. At the time of writing (2004), studies on the health effects of passive smoke have been going on for a quarter century. And despite the "spin" put on the results by the antismoking campaigners, the best that can be said of these efforts is, "no cigar."

In this first section, we'll address some common questions about this issue. The latter part of the document is a more detailed primer on the "science" behind the alarmist but essentially groundless claims that are moving governments towards smoking bans everywhere.

☞ **Environmental Tobacco Smoke (ETS), Passive Smoke (PS), Second-hand Smoke –** these are all simply names for the smoke dissipated into the air, and which may be breathed by bystanders, as a result of a smoker smoking.

Q. If it's not true that passive smoke is dangerous, why does everyone say so? Why do we hear about it every time a legislature or town council proposes a smoking ban?

In a word, politics.

Antismoking activists want smoking bans passed. Their jobs depend on it. Even when they know that the "mountain of evidence about the dangers of passive smoke" is a mountain of something quite different, they believe it doesn't matter. They have found a very effective weapon for getting their way.

After all, when the issue is just the annoyance of smoky environments, the hospitality industry and the market can come up with effective measures that give customers choice. The antismoking lobby doesn't want this. They want to go all the way – perhaps even as far as outright prohibition in a few years time.

Q. But doctors and health authorities support this claim. Why would they if it weren't true?

Again, politics. There is a "party line" on this issue, and dissent is not welcome. We also live in an age of specialization. Not everyone in the health community can possibly be an

expert on this. Some people simply accept the “consensus”. Some understand that the claims aren’t sound, but figure that lying to the public is fine in the cause of antismoking. Some fail to speak out for fear of professional retaliation. “Going against the grain” has its costs.

In such an atmosphere, it’s no wonder that debate does not exist. The only public “debates” that are still open are those between mutually admiring “experts” who reassure each other over the righteousness of the cause at hand (**the scientific foundations, however are never questioned**). They argue only over minor points of implementation of restrictive policies, asking each other *how far* they can go and *how soon*. We do not see public debates on smoking and lung cancer between two experts who disagree on causality or number of deaths. National TV in prime time does not feature programmes with epidemiologists disagreeing with each other on fundamental scientific grounds over the effects of passive smoke on health and the problems of seeking to measure them. Such debates are possible, but they are *suppressed*, often to oblige specific requests from ministries of health to “act responsibly” and not stir up dissent. It’s easier for them if the public accepts what they have to say as an act of faith. If such debates were held, however, the public perception would be very different.

Q. Yet, if the science on ETS is so flawed, we would still expect more doctors and scientists to speak out about it -- in spite of politics. Why don’t they?

Firstly, many *have* spoken out despite the risks of doing so, but they are being drowned out by an anti-smoking movement which is well-organised, well-funded, and very much on a roll.

Q. Where is there strong public opposition to the antismoking movement? I don’t see it.

It exists, it has existed for a long time, and it’s growing. Unfortunately, the forces opposed to prohibitionism and smoker-bashing have historically “missed the target” and have thus lost some momentum. They have focused on such issues as economic damage, lifestyle choice, and appeals to tradition and tolerance. While these issues are undoubtedly important, they pale when put beside the vicious and untrue claim that people who smoke are assaulting – perhaps even killing – the people around them. Those opposing the antismokers have been disorganised and reactive rather than proactive. They have tended to address the issue locally, failing to realise that the problem is global, and concerns international institutions and lobbies that are highly organised and that set goals and decide on strategies years in advance.

Finally, and most importantly, the opposition has shied away from the “magic chalice” of the antismoking movement – the so-called “science of second-hand smoke.” This has happened for reasons of ignorance, unwillingness to learn, and especially for fear of challenging authority. It’s time to change that.

Q. Why would the antismoking movement keep harping on “the dangers of ETS” if this were a weak argument for them to use?

It depends on what you mean by “weak”. In terms of its basis in fact, the argument is fatally weak (but only scientific insiders who check out the research thoroughly could

normally be expected to know this). In terms of its public propaganda value, “ETS kills” has proven a godsend to the antismoking movement.

In fact, it is the antismokers' trump card, and they need it so badly that they cannot admit that it's essentially based on smoke and mirrors.

Before the antismoking movement created the perception of a danger from ETS, it was having great difficulty cutting smoking rates, which had stayed the same for a long time. Smoking was widely tolerated by non-smokers; the general attitude was, 'oh, well, they're only harming themselves.' *Antismokers therefore had to find a way to show that smokers were harming others around them, so as to portray smokers as anti-social, selfish, or even as murderers - and thus to make smoking 'socially unacceptable'.* And, unfortunately, it is working. By constantly giving the impression (though never proving) that ETS is deadly, the antismoking lobby has been able to greatly increase intolerance of smoking, and deprive smokers of the argument of free choice. In short: the antismoking lobby has found ETS to be such an effective propaganda tool that objective science cannot now be allowed to get in the way!

This, however, prompts some fundamental questions. Can we allow, in a free society, the philosophy of “the means justify the ends” to work itself out in the world unchecked? Specifically, can we allow institutions to make use of frauds and misrepresentation of evidence to achieve what is unilaterally portrayed as a “good end”? Where and when will this stop? Where is the demarcation line?

The same abuse of statistics used in the antismoking crusade has already been expanded to target those who drink or eat the “wrong stuff”. Can we allow epidemiological frauds to become a standard tool in the hands of governments and health authorities? Aren’t we just dishonest if we embrace the notion of passive smoke “dangers” 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? And, finally, should we permit “concerns 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 associations, unlimited taxation, massive redirection of public funds, and endless regulatory power by an exploding bureaucracy?

Q. Smoking is deadly. Surely if 'active' smoking is as bad as everyone 'knows' it is, then 'passive' smoking MUST be bad for us. Even if it doesn't kill us, it may be making us sick. Why should we, non-smokers, be exposed to any risk?

At the outset, it must be said that health authorities have begun to greatly exaggerate the dangers of even primary smoke. But we will limit ourselves to discussing passive smoke, since that is our purpose here.

The antismoking movement, in bad faith – bad faith because it is deliberately deceptive – has been working to entangle the issue of passive smoke and primary smoke in the public imagination, so that people believe that passive smoke = primary smoke. The wave of smoking bans that we have seen in the world speaks to the success of that effort.

It is true that passive smoke has 3,000 to 4,000 chemicals in it. It is also true that any organic compound (wood, for example) that is burned has similar chemical composition. However, both primary and passive smoke have extremely small amounts of those

compounds. In the specific case of passive smoke, the smoke compounds undergo numerous chemical processes that make it quite different from primary smoke even chemically, and a further process consists in heavy dilution in the surrounding air. Further information is available in table 3 (page 47), 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 48).

As far as making ourselves safe from "any risk", that is simply impossible! In fact, it is not even desirable, since we have to take some risks even to get to work in the morning. The real issue is *where to draw the line* as to what constitutes acceptable risk. This is a hot topic today in science and public policy circles. In fact, there is a battle going on between those who champion a "zero risk" approach and those who think such an approach is plainly crazy. For more on this, see our comments on the Precautionary Principle (page 17).

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 at least "may" be risky according to someone's epidemiological study. Examples of health scares include (among many, many others) the notion that use of antibiotics might be a cancer risk, or that plastic packaging might be putting men at risk for infertility. *Might.* As we will see from our examination of multifactorial epidemiological evidence later in this document, modern researchers attach very exact and scary-sounding numbers to risks that often boil down to: "*might"* or even "*well, not impossible".*

Today, having been battered with propaganda, people think it is "common sense" to believe that passive smoke puts them at risk simply because it is smoke. Perhaps it's time to do a study to see if 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"? What is "multifactorial epidemiology"?

Epidemiology is the study of the causes of disease. It is most reliable when it looks at diseases that have one necessary and sufficient cause. Such is the case with infectious diseases like cholera, influenza, etc. They are called "monofactorial" diseases.

Diseases in which multiple factors are believed to play a part in cause are called "multifactorial", and the study of these diseases is called "multifactorial epidemiology".

When epidemiology starts to tackle diseases or disorders that do NOT have one 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 at all about actual causation.

Q. Do the terms "correlation" or "link" mean the same thing as "cause"?

No, they do not. 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. Why do tobacco companies not defend themselves against junk science accusations? Doesn't this silence prove that the accusations must be true?

No, it does not. Perhaps it shouldn’t need to be said, but a failure to defend oneself is not proof of guilt.

For a long time, the tobacco companies did mount a defence of themselves, but they were silenced by mounting controversy and a very effective effort by activists and the media to portray everything said by tobacco companies, or any research supported by tobacco companies, as illegitimate.

With the 1998 Master Settlement Agreement in the United States, some prominent tobacco companies capitulated in a “damage control” effort. Faced with superior power, lawsuits, harassment and vilification, the tobacco industry accepted making payments of US $5 billion per year for 25 years as compensation for the damages of active smoking -- in spite of the fact that, even today, the causality of **not even one single death** from smoking can be established with scientific certainty.

Certain tobacco multinationals are now even siding with their aggressors, confirming antitobacco’s statistical frauds, distortions and exaggerations. Paradoxically, they use the same paternalistic rhetoric as their enemies. They accept the “bad guy” role in society, act against the product which is the reason for their existence, and *officially apologise to the “health” authorities for having attempting to defend themselves in the past* – in the empty hope of regain a public image that assures their survival. This, of course, is an error, for the ultimate goal of antismoking advocates is the extinction of the industry, and the relegation of tobacco to the status of an illegal drug. But the reaction of the accused against an accuser with virtually unlimited power is as predictable as it is human.

In short, certain large tobacco companies that are heavily diversified have adopted the “if you can’t beat ‘em, join ‘em” philosophy. Others continue to fight.

The tobacco companies have failed to counter their opponents, partly through their own errors and omissions. It’s not that they haven’t pointed out the problems with junk science – they have. But for many years now, no one has listened. Researchers who produce good, reputable work that deserves publication and a proper hearing simply aren’t published any more if even a small part of their research has been funded by the tobacco industry.

Q. I don’t even smoke. Why should I care about any of this stuff?

Everybody should be concerned that we have **honest** public discourse and decision-making, whether or not we have a personal concern in the specific subject matter.

The most important general message we want to convey in this document is that the propaganda on passive smoke 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-true 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 used on this issue, it can be used on others too. And in fact, it is already happening.

Of course, those directly affected by prohibitionist policies, such as those in the hospitality industry, have an urgent and vital interest in this issue -- whether they smoke or not.

As to the *annoyance* of smoking, a compromise between smokers and non-smokers CAN be reached through **setting air quality standards** and the use of **modern ventilation technology** (more on that later).

Q. If the attack on passive smoke helps rid society of tobacco, why not go for it? Even if passive smoke is just a "smokescreen", what's wrong with that?

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. By the way, in many jurisdictions it is our tax money that at least partially pays for antitobacco programs. We have every right to demand accountability!

Moreover, many people in our society still think that individuals should be able to make their own personal choices – about whether or not to enjoy cigarettes, pints, and pizzas, for example.

Q. Why doesn't the media look into this more?

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 – including reporters or politicians -- wants to be accused of defending "unhealthy choices".

There's another reason. Most people lack the background to check out the claims about passive smoke. It's much easier – even for reporters -- to simply accept arguments from authority and "take the experts' word for it."

Q. What is the basis of the antismoking movement's claims that passive smoke poses a risk to health?

Surprising, there is virtually no basis. Well over 100 studies have been done since 1981, some of them rather poorly designed, others more impressive in their methodology. But at the end of the day, taken in their totality, the studies are inconclusive. And this is particularly true of the larger, more well-designed "serious" studies.

Q. What exactly does "inconclusive" mean?

It means that there is simply no proof – indeed, no particularly persuasive evidence -- that passive cigarette smoke as we typically encounter it in ordinary life is a risk to people. It also means that there is no proof of the absence of a risk. This in turn implies that if a risk does indeed exist, it is likely to too small to be measured.

That would lump passive smoke 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 smoking ban proponents actually amounts to.

Q. I've heard there is generally a 20% risk elevation from passive smoke. Isn't that something to worry about?

No, never. We go into more detail about this later in the document, but briefly, here is the reason why:

- the studies do not know what they've measured

- most studies show at the same time a risk and a benefit from passive smoking, thus they do not pass an important epidemiological criterion: statistical significance. (Don't panic – we'll explain this later!)

- when showing either a benefit OR a risk, they either

    - examine too small a number of subjects, resulting in an enormous distortion

    OR

    - the risk/benefit elevation is so small that it does not fit the criteria of 200-300% elevation set forth by the most authoritative epidemiologists and institutions in the world.

In laymen's terms, unless the risk elevation is **at least 200%**, it "doesn't count" according to the established rules of epidemiology. That 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. But 20% "sounds like a lot" and the figure is used by antismoking activists to set off emotional alarm bells in public discussions.

- Even a statistically significant study can have other methodological flaws – and the second-hand smoke studies do.

Q. I've heard that urine tests prove that people are being poisoned by the toxins in passive smoke. What do you say about that?

There are studies showing that people exposed to passive smoke have **cotinine in their urine**, and it is suggested that this is evidence of danger.

A little background. Cotinine is a naturally-produced nicotine metabolite, so if you've got it in your urine it means you've been exposed to nicotine. But nicotine abounds even in vegetables (especially tomatoes); therefore, a non-smoker living in a non-smoking environment and who eats certain vegetables may have abundant cotinine in his urine without ever having been exposed (those studies almost never check for diet.) Some studies measure cotinine directly on the subjects (i.e. in the hair) rather than in urine. Still, the presence of cotinine only indicates exposure to passive smoke and/or other sources of nicotine. It does not prove risk, and is therefore meaningless.

Q. Advocates of smoking bans say that *"passive smoking is like peeing in a swimming pool".* They argue that we should simply not have to put up with it in the environment. That sounds persuasive.

A vast library of short, easy-to-remember slogans and sound bites has been developed to hammer home a consistent belief about passive smoke – but none of them stands to reason, although they do appeal to emotion. The well-worn *"passive smoking is like peeing in a swimming pool"* is calculated to suggest that a common good enjoyed by all (air) is polluted in a disgusting way by the few (smokers) to the damage of all in an enclosed environment. But, given the tiny amounts of smoking pollution in a normal, enclosed environment and the non-existing evidence of harm, a more realistic sound bite should be: *"passive smoking is like peeing in the ocean"* – and, in fact, we all eventually do. Actually, it's the word "pee" that really makes this quote effective, designed as it is to evoke a "yuck" reaction.

Q. The statistical and scientific evidence of the tobacco industry should not be believed. How many of the studies listed in Section II are funded by that industry?

None of the studies on passive smoke has ever been financed by the tobacco industry, with the partial exception of one major study. It was minimally funded in its very final stages by the tobacco industry when it did not turn out to have the "right" results (therein lies a story! See Appendix II). Except for that, all of the passive smoke studies have been done with public funds and by the pharmaceutical industry. The tobacco industry has only financed some papers of commentary and some writers who interpreted the studies – but not the studies themselves.

Q. Isn't it wrong for the tobacco industry to fund research?

Although it shouldn't need saying, funding by the tobacco industry – or any other industry, for that matter – does not determine whether research is good or not. But there are real – and growing – concerns with potential conflict of interest in the business of science funding today. (It should be noted that, due to controversy, the tobacco industry no longer funds research).

Questions about who funds studies (and why), and the influence of corporate support for health-related science journals, is stirring a lot of controversy these days. With the private sector shouldering more of the burden of funding science, scientists and journals are increasingly worried that disinterested science is being compromised. This issue goes beyond the scope of the present discussion, but it is a serious concern.

We should note in this connection that the antismoking establishment is frequently allied with the pharmaceutical industry. That industry, which manufactures the smoking cessation devices so widely promoted today, is involved with funding antismoking lobby efforts. It is also largely in control of scientific publications through advertising sponsorship. Pharmaceutical companies are officially partnered with the WHO, particularly for antismoking programmes. So this is another industry that has its own interests. Yet few would say that pharmaceutical companies should be barred from funding studies in health journals. Indeed, such companies are often the only entities with funds available. Yet they too present the problem of possible corporate agendas that could corrupt the scientific enterprise. There is no "quick fix" for such legitimate concerns.

In any case, in the passive smoke controversy, the studies have not come from the tobacco industry. We should also note that there are not two sets of evidence here, representing two "sides" -- it's not a case of "antitobacco" studies versus "pro-tobacco" studies. *There is only one body of studies. The question is: what does that body of evidence really represent?* In the following pages, we will see how the public has been given a **false representation of evidence** by people who ignore the rules of epidemiology in order to score political points.

Q. Still, passive smoke bothers people. It stinks, and makes eyes water in enclosed environments. Isn't that a clear indication that it must be harmful?

Years ago there was smoking literally everywhere: airplanes, restaurants, workplaces – even hospitals and buses, and nobody complained - even those who truly hated smoking. Why? Because there was *no hysteria*. Today things have changed, and suddenly smoking bothers everybody. Left to common sense, the issue of annoyance has a solution built in: separation of areas and/or ventilation are practicable and economical virtually everywhere, and they alone would be sufficient to peacefully resolve the issue. But that is not the agenda of the antismoking industry, whose goal is the elimination of smoking everywhere first, and ultimately, the de-legalisation of tobacco.

Unarguably, strong concentrations of passive smoke annoy everybody – including smokers themselves. An indication of harm? The point is certainly worth closer examination.

When passive smoke is concentrated enough to become an annoyance, that's an indication of poor air exchange – and when eyes are watering, that means that the air exchange is down to dangerous levels. If we run an analysis of that air, we often find "stuff" that is really bad for health: elevated levels of carbon dioxide, lack of oxygen, various chemical compounds, high levels of dust, viruses, bacteria. Those elements are there regardless of passive smoking, which indeed becomes a useful tool to indicate that it is time, literally, to change the air. Without the passive smoke marker, people are exposed for much longer to harmful elements without realising it, as most of those elements are invisible and odourless. It is noteworthy that despite having eliminated passive smoke for several years, places such as California – where a state-wide ban has been in place since 1998 -- the trend of increases in asthma, for example, continues at staggering rates. [1] Once smoke is eliminated and people think that the air is "clean", air circulation systems are *turned down to save energy*. Isolated claims – such as that of California -- that there is a decrease in lung cancer incidence since tough antismoking measures were enforced are just propaganda, [2] as that disease actually requires decades to develop, and the time elapsed is too short (for example, the 1988 ban did not include smoking at home) to observe any effect whatsoever.

Q. How do we explain, then, all those deaths caused by passive smoke? We hear that in the United States there are as many as 53,000 premature deaths each year.

[1] See: Points of Interest, California Department of Health Services and the Public Health Institute, Cancer Surveillance Section, Survey Research Group, Number 9, May 2003 - http://www.dhs.ca.gov/ehib/ehib2/PDF/BRFSPointofInterest.pdf

[2] See, for example: "State lung cancer rates largest drop in nation", The Associated Press Dec. 2, 2000 - http://www.berkeleydaily.org/article.cfm?archiveDate=12-02-00&storyID=2458

Media, "public health" and activists love to use the word "*caused"* to induce people to believe there is an established causality. As we will see in detail in the following pages, there is no absolutely no demonstrated causality of death and disease by passive smoke. *Attribution* is the word that should be used, rather than cause. But *attribution does not mean causality* – it is *just an opinion on causality* that is not substantiated by science. It is interesting, for example, that while the EPA still claims its original attribution of 3,000 passive smoke-related yearly deaths in the US [3], other authoritative entities claim 53,000 – a figure over 17 times larger. [4] Others even say 100,000. [5] The same discrepancy applies to the primary smoking "death toll". While the World Bank claims an average smoker's life loss of 20-25 years (!) [6], the WHO has gone from claiming 3 million deaths a year in 1990 to projecting ten million for 2010 [7]– the bigger, the better, it seems. On May 31, 2003 (the date of the WHO-trumpeted World No-Tobacco Day) at an official event in Milan, the Italian League Against Cancer claimed 90,000 tobacco-related deaths in that country. [8] On the same day in Rome, at another official event, the minister of health claimed 53,000 [9] – a difference of 43 percent.

Why don't we see those discrepancies for polio, tuberculosis or malaria, for example? 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 these huge discrepancies in "tobacco-related deaths": *the figures are made up through arbitrary statistical computer models.* Here is another significant indication: nowhere in the world is there *one* death certificate stating that death was caused by passive smoking. Every physician is aware of the legal difference between propaganda and a cause of death that is known and can be stated with reasonable certainty on a death certificate.

Junk science debunker Steven Milloy [10] explains the body count statistical trickery quite well in his book *Junk Science Judo – Self-defense Against Health Scares & Scams.* [11]

---

[3] See: Environment, Health and Safety Online, Environmental Protection Agency, Respiratory Health Effects of Passive Smoking - An EPA Fact Sheet; Reprinted by EHSO - http://www.ehso.com/SmokingRespHealth.htm

[4] See for example, the Frontline interview (PBS television, USA) with antismoking guru Stanton Glantz: http://www.pbs.org/wgbh/pages/frontline/smoke/interviews/glantz2.html

[5] This figure was publicly mentioned by NY City majoralty candidate Mark Gree in 1997 during his election bid.

[6] See http://www.worldbank.org/html/extdr/hnp/hddflash/hcnote/hrn001.html

[7] See Morbidity and Mortality Weekly Report, May 21, 1999, World No-Tobacco Day press release, US Centers for Disease Control. -- http://193.78.190.200/who/cdc1.htm

[8] The figure was reported by the Corriere della Sera on June 27, 2003 (http://193.78.190.200/italy/corriere_27_giugno.htm), among others. This year (2004) League is using the figure of 85,000 (http://www.legatumori.mi.it/prevenzione/fumoPrevenzione.htm).

[9] As reported in "Il Nuovo", June 1, 2003 (http://193.78.190.200/italy/54000.htm)

[10] See his website at http://www.junkscience.com .

[11] pp. 115-117 ' "Body counts" are a great way to grab the public's attention. Smoking kills 400,000 per year. Obesity kills 300,000 per year. Medical mistakes kill 98,000 per year. Secondhand [passive] smoke kills 50,000 per year. Radon kills 10,000 per year. Body counts, or rather "non-counts," are simply more statistical malpractice.'

'… Epidemiologists estimate the body counts by using the formula for "attributable fraction" (AF):

$$AF = \frac{\text{Proportion of population with characteristics X (relative risk} - 1)}{\text{Proportion of population with characteristics X (relative risk} - 1) + 1} \times 100\%$$

Without even understanding the formula or actually working through an example, you should already see what the problem is – reliance on relative risk. The attributable risk formula **pretends that relative risk indicates risk**' *[let us*

Milloy demonstrates the absurdity of the procedure used to calculate the premature deaths from active and passive smoke. Is it conceivable that such levels of incompetence truly exists in "public health?" We question that.

Q. Still, this is an admission that smokers and passive smokers die a premature death. Right?

Firstly, as far as so-called "passive smokers" are concerned, since the case for risk has not been made, any claims about "premature death" are mendacious nonsense.

But the most intriguing question about this issue is simply this: what is a **premature death?** Premature *next to what,* exactly?

Actually, people should die at the exact same age – at least, according to statisticians! In the world of statistics there is a person we'll never meet. He is Mr. Joe Statistical, a peculiar guy, born in a few nanoseconds from theoretical models where hypotheses and assumptions are reduced to numbers and entered in computers. JS (for short) lives *exactly* the average number of days, has the average vices, suffers the average ailments, and surrenders an average number of sleepless nights to work and sex. JS eventually dies an average death. This theoretical individual is the metre against which real people are measured.

JS lives a set number of years that changes for every country: let's say **74** years, for example. His wife, as a woman, lives to 76. When a real person dies (let's call him Mr. Real), he is compared with JS, and if his life was shorter, his death is defined as "premature". But premature deaths **are not supposed to happen**, as in the family of JS they never occur. Thus, there must be *an explanation of why he failed to be average*. If Mr. Real smoked and died at **73** – well, *there you go: smoking kills*. Thus his death is officially attributed to smoking. And because he lost one statistically potential year of life to boot, *his death is considered premature*. That year becomes a *cost to society*, quantified through a

---

remember that relative risk is, instead, a ratio between a sample and another sample, as described above – and in the case of passive smoke that does not even mean that the risk is real].

'As we know, relative risk can be used **only** for statistically associating an exposure with a disease. But relative risk **does not prove that a cause-and-effect association exists**. The attributable fraction formula simply ignores this inconvenient fact.'

'…Let's say that 20 percent of the US population lives in areas where the level of fine particulate air pollution exceeds a level statistically associated with a 17 percent increase in death from cardiopulmonary diseases. The question for the junk scientist, then, is: how many deaths will be caused by the fine particulate air pollution? Plugging in the numbers into our attributable fraction formula, we have:

$$\text{AF of cardiopulmonary deaths from air pollution} = \frac{.20 \times (1.17 - 1)}{.20 \times (1.17 - 1) + 1} \times 100\% = 3.3 \text{ percent}$$

'The junk scientist would conclude that 3.3 percent of all cardiopulmonary deaths are **caused** by fine particulate air pollution. So, if there are 1 million deaths in the United States annually from cardiopulmonary causes, then 3.3% of these – or 33,000 – are **caused** by fine particulate air pollution. The fatal defect in this junk science exercise is the **assumption** that the relative risk really MEANS that fine particulate air pollution increases death rates by 17%.'

'Attributable fraction and attributable risk – the number derived by applying the attributable fraction to the number of deaths – is statistical abuse squared. […] And here you have it: Body non-counts = Attributable risk = "Non-sensical." '

complex set of formulas. Furthermore, it may be that Mr. Real had been ill for some time before his death. So, all the medical expenditures are attributed to smoking and increase the cost to society.

What if Mr. Real had died at 73 and did not smoke? In that case, if we are running a study on passive smoke, we ***ask*** Mrs. Real, a smoker, how many cigarettes she smoked while living with her husband to establish how many "passive cigarettes" it took to kill him. If Mrs. Real does not smoke we turn to co-workers, friends and relatives who smoke. The killers must be found! Remember: junk science *has already decided what Mr. Real died of*.

What if Mr. Real died at 73 and smoked, but also ate and drank a lot? In that case, he died once because of smoking, once because of his diet and once because of alcohol dependency. People have many lives in statistics – actually, many deaths: it all depends on how many studies they feature in and what those studies must show. [12] Finally, the information is passed on to the media for the final statement: "Vice X **kills** *(number of death attributions here)* per year, at the social cost of (*attributed billions here*), along with a call for restrictive policies.

What if active/passive smoker Mr. Real lives to the age of 90? Well, that is an *aberration* due to chance and **luck** – and it is certainly not attributed to the benefits of smoking. Smoking kills. But what if too many smoking Mr. Reals live to 90? In that case, the average life of Mr. JS becomes longer – say 76 years instead of 74. But that also means that all the smoking Mr. Reals who made it to **75** instead of the previous 74 will now *join the ranks of "premature deaths caused" by smoking* -- and that will cost even more to society! Junk science always wins in the end.

Q. Nevertheless, studies show that when passive smoking is removed from enclosed environments, the general health of people gets better. For example, the removal of passive smoking in the city of Helena, Montana, clearly demonstrated a reduction in heart attacks. Doesn't that prove something*?*

We won't send you to Appendix II, where we deal with various well-publicised studies, for an explanation of this shameless piece of junk science, which we would prefer to dismiss here.

The highly publicized (but unpublished) Helena "study" shows that just **six months** after public smoking became illegal in Helena, the rates of heart attacks decreased by a whopping 50%. As journalist Jacob Sullum related the story in an article for Reason Magazine, the study came from 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." Glanz, incidentally, has been a recipient of very generous pharmaceutical funding to help with this work. In Sullum's account, "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

[12] This point is hammered home by economist Pierre Lemieux, of the University of Hull, Québec, in his book "Smoking and Liberty" (page 32): *"Although the infamy cast on tobacco may be partially explained by our natural fear of disease and death as well as our tendency to look for scapegoats and panaceas, the irrationality of it remains striking. Bertrand Daveaud and Bertrand Lemennicier have observed that by adding together the proportions of lung cancer traced to tobacco (85%), radon (30%) and working conditions (40%), all 155% of the cases of lung cancer are accounted for."*

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 goes 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 second-hand smoke not only makes life more pleasant; it immediately starts saving lives."*
>
> *To a more skeptical 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."*
>
> *A little calculation shows how preposterous this claim is, even if you believe that second-hand smoke causes heart disease. The American Heart Association attributes 35,000 heart disease deaths a year, about 5 percent of the total, to second-hand smoke.*
>
> *It seems reasonable to assume that the proportion would be similar for heart attacks, fatal or not. So even if a city completely eliminated second-hand smoke (which Helena's ban did not do, since it did not apply to smoking at home), how could that possibly cut heart attacks in half?*
>
> *Sargent and Glantz note that smoking bans also encourage smokers to cut back or quit. Inconveniently for them, that point suggests that any drop in heart attacks could be due to less smoking rather than less exposure to second-hand smoke.*
>
> *In any case, the numbers still don't add up. According to the U.S. Centers for Disease Control and Prevention, smoking accounts for about one-fifth of heart disease deaths. So even if every smoker in Helena quit (which no one claims happened), you would not get anything like the drop that Sargent, Shepard, and Glantz attribute to the ban.*
>
> *If smoking bans cut heart attacks in half, it's odd that no one has noticed it before, especially in big cities such as Los Angeles and San Francisco, where an effect of such magnitude should have been obvious.*[13]

The Helena "study" is absolute junk science nonsense, and does not deserve any further consideration. But such epidemiological malpractice is not restricted to one study -- unfortunately. Junk science attached to political agendas is in fact routinely used to induce us to support predetermined public policies. In his book *Junk Science Judo – Self-defense*

13 See "Heartstopping Discovery: Do smoking bans cut heart attacks in half?" By Jacob Sullum, Reason Magazine, April 4, 2003. http://reason.com/hitandrun/001308.shtml

Against Health Scares & Scams, Steven Milloy documents another sham smoking ban health study. [14]

Q. Well, if there is so much bad or dubious science around these issues, truth will out in the end. Won't this just blow over as an overreaction?

That seems unlikely .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 commonsensical: isn't prevention 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. A simple hypothetical example should suffice to illustrate.

Most of us probably assume that if a government entity 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 fails to prove that the product is not dangerous, then the government has the power to completely

---

[14] Milloy refers to a study that created the headline, "Smoking ban boosted health of bartenders, study reports", in the *Los Angeles Times* headline on December 9, 1998. He quotes the Times article as follows:

> *San Francisco bartenders showed dramatic improvements in lung health within* ***two months*** *after the January implementation of the California's indoor smoking ban, UC San Francisco Researchers report today.*
>
> *Bartenders were exposed to unusually high levels of secondhand smoke before the ban – about four to six times the level found in other workplaces.*
>
> *Examining 53 bartenders before and after the ban was implemented, Dr. Mark D. Eisner and his colleagues at UC San Francisco found that 59% of those reporting respiratory problems, such as wheezing, shortness of breath and morning coughing, were symptom-free less than two months after the ban began.*
>
> *Moreover, they report in today's Journal of the American Medical Assn., 78% of those with eye, nose or throat irritation were also symptom-free.*
>
> *"That's a pretty big change over a short period of time," Eisner said. Although the number of people studies is relatively small, the results are considered statistically significant…*
> *In an editorial in the same issue of the journal, Dr. Ronald M. Davis of the Henry Ford Health System in Detroit called for further smoking bans across the country.*

Milloy's comments:

> But of the study's 53 bartenders, 24 smoked – and smoking is a risk factor for lung illness. And what the *Times* didn't report was a key information describing when the data was collected:
>
> > *From December 1 to 31, 1997, we interviewed and performed spirometry on participating bartenders in their workplaces (bar or tavern). Follow-up interviews and spirometry were performed from Fabruary 1 to 28, 1998, to evaluate changes in symptoms or lung function following the institutions of smoke-free bars.*
>
> Notice anything? Bartenders were interviewed about their respiratory health in the middle of flu season and then re-interviewed when the flu season was over. Perhaps this is why the study authors were compelled to note at the and of the study. "Confounding by personal smoking and upper respiratory infections could potentially explain the observed improvement in respiratory health."
>
> But the *Times* apparently wasn't interested in the headline, "Bartenders Get Flu and Recover, Study Says." Such a headline would also not go far in justifying a smoking ban.

forbid and/or regulate at will. *Suspicion of possible harm is evidence enough*. But every scientist will tell you that it is impossible to prove a negative. So will every lawyer. This impossibility ensures that the accusing authority is always "right".

But it doesn't stop there! Even if one *were* able to prove the negative, *suspicion of possible future harm is still enough to permit intervention*. If you think this is nonsensical, you are absolutely right!

With a such principle now enshrined and forming the mentality of a generation, we can only hope that the Precautionary Approach does not seep into criminal law. Will we one day see the principle of reverse burden of proof in criminal courts, so that we can all feel "safer"? Most of us know enough history to appreciate how dangerous and uncivilized this would be.

We 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 mockeries of science and their perpetrators, and enables bureaucrats and politicians to grab power, regulate, tax and forbid at will – and get away with it without victims having a legal recourse.

Q. Why isn't the public more aware of all these issues?

1) The antismoking movement has been very successful. Among other things, they have been successful in cutting off debate. They accuse any dissenter on any point of being "a stooge of Big Tobacco". Like an accusation of witchcraft, it works. On the media. On politicians. On scientists. On the public.

2) The issues are hard to explain. In order to understand something about the real state of the evidence on passive smoke, 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 government bureaucrats, all of whom are regularly lobbied by well-funded antismoking activists.

In the pages that follow, we'll take readers through the ABCs of the epidemiology used for passive smoking studies in order to provide that basic understanding. Have some patience – in order to understand, it's essential to read carefully, and you will have to wrestle with some new concepts. But we've done our best to make this explanation accessible to the layman. And if you or your business has an interest in defending yourself from smoking prohibitionism, it's important to arm yourself with the right information so that you can identify the lies and distortions when they are presented to you. This is information that your opponents are hoping – and betting -- that you never get your hands on!

PART II

SUMMARY OF ARGUMENTS

The following is a short summary of the key points that the layman should understand about the “science” on passive smoke. In the section that follows, we’ll examine in detail how research is actually performed and what it actually “says”.

The epidemiologic (epi) studies of PS (passive smoke) affirm to have reached risk estimates by measuring the frequency of certain diseases in relation to measures of PS exposures.

Alleged risks of PS exposures are estimated by measuring differences in disease frequency and PS exposure in groups of many individuals **that must be non-smokers**. An increased risk is estimated if the frequency of diseases is higher in groups more exposed to PS, and vice versa.

Public accountability is vital…

The risk claims of epi studies of PS are not research propositions of theoretical use restricted to epidemiologists and others, but are made to justify forcible social policies and police interventions that trouble millions and millions of people around the globe. Therefore such **claims and their foundations must be accountable**, at least for the free citizens of free democratic societies.

...and so is reliable evidence

Alleged risk of PS exposure are expressed as numbers, reflecting numerical measures of disease frequencies and of PS exposures in different people. Such **measures are therefore subject to elementary tests of reliability**.

The measures of PS exposure **must warrant to have actually measured exposures** and not something else, and that those measures provide **reliable accurate statistical representations** of the various extents of PS exposure and disease frequency in groups of people.

There must be **a warrant that the people studied are indeed non-smokers**, or else the results could be corrupted by the hidden risks of active smoking.

Lung cancer, cardiovascular diseases, and other diseases that are allegedly associated with PS can have many causes (asbestos, air pollution, diet, lack of exercise, obesity, family predisposition, and many, many others). Therefore the epi **studies of PS exposures must warrant that no other causes are responsible for the apparent risks** that could be wrongly attributed to PS exposure.

The results of **different epi studies of PS exposures must be similar, comparable, consistent, and reproducible**.

In the following pages we are about to demonstrate how and why none of the epi studies of PS exposure so far performed meets the elementary requirements of reliable evidence listed above. On this basis, we affirm that the current policies seeking to restrict active smoking and PS are based on bogus claims of non-existent scientific evidence. We further affirm that such claims are consciously and wilfully fabricated and imposed on free citizens and societies by special interests that are no less false and meretricious than the cigarette industry.

EPIDEMIOLOGY AND PASSIVE SMOKE

The studies on passive smoke: what they are and what they are not

The epidemiology of passive smoke (PS) aims at finding correlations between various diseases and exposure to PS, from which it attempts to infer cause-effect connections. Epidemiologists dealing with this issue insist that PS investigations are scientific, even though most prominent epidemiologists agree that the epidemiology of PS *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 passive smoke and disease is studied. We have made an effort in these pages to avoid excessive technical detail, since our purpose is to provide an accurate but simple explanation for the layperson. The basic ideas expressed here are not difficult to grasp if the reader exercises a bit of patience.

☞ **PS** – Passive Smoke

☞ **Correlation** – a correspondence or parallel between phenomena.

☞ **Epidemiology** - the study of the distribution and determinants of health-related states or events in specified populations.

Why a scientific approach *can't* be taken

A truly scientific epidemiologic study of PS 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 PS, during a time adequate for the development of the possible diseases. However, such a study would be unethical because it could conceivably mean harm to study participants. It would also be impractical, because it would take decades to complete.

The epidemiology of PS is therefore limited to retrospective surveys in populations of self-declared non-smokers that happen to have been exposed to PS in the course of their lives. Indeed, the subjects usually would be of the advanced ages where such possible effects as lung cancer or cardiovascular diseases could be observed. What this means is that epidemiologic studies of PS 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.

☞ **Experimental** - the use of controlled observations and measurements to test hypotheses.

☞ **Observation** – the passive watching of a phenomenon and the recording of what happens.

☞ **Empirical evidence** - evidence relating to or based on experience or observation.

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** (exposure to PS, in our case), and not to other disturbances that interfere with the observations 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.

☞ **Hypothesis** -an original assumption that must be demonstrated or rejected through experimentation.

As we are about to consider, the epidemiologic studies of PS cannot ensure they have measured PS exposures with any degree of identity or accuracy, cannot ensure the absence of other explanations for their observations, and show a remarkable inconsistency of outcomes. **Indeed, some studies may claim a slight increase of risk, others a decrease, and most neither an increase or decrease of alleged risks!**

Epidemiologic studies of passive smoke – not the same as science

The epidemiology of PS claims correlations of PS 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 proven unable to determine specific causal mechanism for such diseases. In this regard, Doll and Peto, arguably the most prominent epidemiologists today, have concluded that:

> *[E]pidemiological observations...have serious disadvantages... [T]hey 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……..*
>
> *[I]t 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."…….[T]hese 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. p. 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 justify public and private policies.

☞ **Multi-factorial disease** - a disease that can be caused by different factors acting by themselves or in combination with other factors (confounders, concomitant factors). This is the case of cancer, heart disease, and stroke, for example. The relative influence of concomitant causes and factors greatly changes for each individual. Further, the body of each individual continuously changes, and so does his environment throughout life. For those reasons it is virtually *impossible* to isolate a single factor and quantify its contribution to the disease of one individual. Very rough statistical approximations can be made on population masses through decades of observations on hundreds of thousands of people. But even the best studies must be considered vague pointers – the reason why epidemiology cannot **ever** establish causality.

☞ **Mono-factorial** – A disease with only one known cause, such as polio, tuberculosis, or smallpox. The control or eradication of this kind of disease was a triumph of epidemiology in the past. In this type of diseases, if the cause is known there is no doubt about causality and effect.

☞ **Incidence** - The frequency at which a disease appears in people.

☞ **Retrospective study** – A study that identifies groups of subjects with different incidences of diseases and attempts to reconstruct their past exposures through memory.

☞ **Longitudinal or cohort**

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 judgmental.

study – A study that identifies groups of subjects exposed or not exposed to potentially toxic conditions.

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 PS. Let's briefly examine how these studies are conducted, and what they actually measure.

***Retrospective* cohort (or *longitudinal*) studies**

These studies record different individual recalls of disease incidence in groups of people possibly exposed to PS 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 PS exposure. Only a handful of such studies have been performed in regard to PS.

***Case-control* studies**

These constitute by far the majority of PS studies. They record different individual recalls of possible lifetime PS 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**, and not actually estimated as differentials of disease incidence. Increased risk is inferred but not directly estimated if exposure is found to be higher among cases, and protection is inferred but not directly estimated if exposure is found to be higher among controls.

Please 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 as 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.

Now, some arithmetic

The arithmetic of risk calculation is the same for cohort and case-control studies, except for a difference in terminology. In cohort studies the ratio used for risk calculation is called RR (relative risk), while in case control studies the ratio is called OR (odds ratio).

However, the important difference is that the cohort studies estimate risk directly as differentials of disease incidence, since cohort studies observe only the health outcomes that could be associated with exposure to a particular factor. The case-control studies only assume risk from differentials of exposure, since they are designed to observe the exposure that subjects may have had in the past to the factor of interest.

Let us go into a bit more detail as to how the calculations are done – but don’t worry, it is simple arithmetic!

In cohort studies, risk is measured as a difference in *disease incidence* between exposed and non-exposed subjects. The risk is defined as relative risk (*RR*), and it is expressed this way:

RR = Incidence rate in exposed / Incidence rate in non-exposed
Thus, the disease incidence rate in the exposed subjects is simply divided by the incidence rate in non-exposed subjects. The *RR* ratio reflects that a certain incidence of disease is observed in both non-exposed and exposed subjects, due to multiple background causes operating in conjunction with, or entirely separate from the exposure under study. Therefore, risk in the exposed is said to be an increment or decrement of incidence, *relative* to the basic incidence of the non-exposed subjects.

☞ **Case-control study** - done to observe differences of postulated toxic exposures in groups of people with or without disease. Case-control studies are necessarily retrospective and their weakness is that they do not measure differences of disease incidence.

☞ **Relative Risk (RR), Odds Ratio (OR)** – A **ratio** between the incidence of diseases in a group of people believed to be exposed to the risk (in our case, passive smoke) and the incidence in a group of people believed not to be exposed to that risk.
If the incidence of disease is the same in the exposed and in the non-exposed groups, the ratio is **1** and there is no change in risk. If the incidence of disease is greater in the people exposed, the ratio becomes greater than 1 and the risk has increased. Conversely, the risk is smaller than 1 when the incidence is lower in the exposed people, implying that the exposure protects from the disease.

☞ **Incidence** - The frequency at which a disease appears in people.

☞ **Multiple background causes** – As we have seen in multi-factorial diseases, many causes can concur in any possible combination to the occurrence of a disease. Multiple background causes simply means all those causes operating on the background of the life of the examined subjects, having an unquantifiable effect on the results.

☞ **Relative Risk (RR), Odds Ratio (OR)** – A **ratio** between the incidence of diseases in a group of people believed to be exposed to the risk (in our case, passive smoke) and the incidence in a group of people believed not to be exposed to that risk.

In the RR ratio above, if the rates are the same in exposed and non-exposed subjects, the *RR*=1 and therefore there is no risk differential**. If *RR* is greater than 1, the risk is said to be increased in the exposed subjects. If *RR* is smaller than 1, the risk is said to be**

decreased in the exposed subjects, indicating that the exposure under study might be possibly protective.

Because case-control studies infer but do not directly estimate possible risk, their results are expressed as odds ratios (*OR*), namely the ratio between the odds (expressed as % or other rate) of being exposed for the cases and the controls:

OR = Odds that cases were exposed / Odds that controls were exposed

In the above ratio, if the odds are the same in exposed and non-exposed subjects**, the *OR*=1 and there is no inference of difference in risk. If *OR* is greater than 1, there is an inference of increased risk in the cases. If *OR* is smaller than 1, there is an inference of decreased risk for the cases**, presuming that the exposure may possibly protect for the disease under study.

Both cohort and case-control studies are affected by similar difficulties of design, data collection, and interpretation — difficulties that are far worse for case-control studies that uniquely rely on vague recollections of exposure.

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 thus "throws the results off". 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. For the sake of illustrating the point, let us imagine an extreme example of bias: the comparison of a group of eight-year-old girls with an 80-year-old group of male former prisoners of war! The differences between these groups are so extreme as to such factors as age, life experience and medical history that any comparison would hopelessly compromise the results of the study.

A selection bias occurs when control subjects mismatch the test or case subjects in 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.

☞ **Bias** – A type of error, sometimes in the basic selection of elements for the study, that distorts of the final results.

☞ **Recall bias** – the bias that is caused by using people's memories as data. Here is a good, realistic example of bias and recall bias. Question*: "How many cigarettes a day do you think that uncle Joe smoked in the presence of aunt Annie between 1962 and 1968, when Annie died and you were 7 years old?"* Answer: *"I don't know, perhaps between 15 and 25…?"* The number of **25** cigarettes is marked in the interviewer's questionnaire as if it was a ***verified, measured data***. Later, those 25 cigarettes will be computed as part of the *attribution of death* for a certain amount of population.

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 recall statements, and no verification is possible. It is only natural that persons with cancer or other diseases would be more inclined than persons without disease to blame PS exposure, in an effort to rationalize their disease and explain it.

Measuring memories

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 sixty 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**.

More bias

Differential accuracy of disease diagnostics and death certificates may affect the classification of subjects. A misclassification bias occurs when subjects wrongly declared 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 or other diseases than in control subjects that are otherwise healthy, thus contributing to a false impression of elevated risk.

The trouble with confounders

Confounders are defined as hidden risk factors that could also participate in an association. Study subjects with cancer must, as a matter of course, 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.

☞ **Confounder, Concomitant factor** - Factors and circumstances that contribute to the occurrence of a disease which has more than one known and certain cause.

For instance, PS studies dealing with lung cancer should consider some three dozen risk factors as potential confounders reported in the literature, and studies of cardiovascular conditions face over 300 published accounts of risk factors as potential confounders. For a complete list of lung cancer risks, see *Appendix 1.* It should be apparent that without a credible control for at least all major known confounders, epidemiologic studies of PS could not be validly interpreted.

Apples, oranges and absurdity

In the particular instance of case control studies, by far the majority of PS studies, the epidemiologic reports indicate that control subjects without lung cancer may recall exposure to PS 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. Conversely, the claim of a 15% increased risk of cancer is logically untenable, being a fictitious excess over that of controls who have no cancer.

The framework of such studies also rests on numerous unverifiable assumptions. First, that the difference in ETS exposure between cases and controls is a true difference -- and

second, that both cases and controls are equally affected by confounding factors that may increase or decrease the risk of lung cancer. In reality, those presumed conditions cannot possibly be met.

Not only have we compared apples to oranges by comparing people with cancer of unknown origin exposed to passive smoke with people without cancer and also exposed to passive smoke, but we have taken this bizarre absurdity further. We can even express this absurdity with a theorem of appropriate name:

THEOREM OF THE ABSURD

The exposure to 100% passive smoke does not cause cancer because the controls are all healthy. The exposure to 115% passive smoke is an absolute and sufficient cause of cancer, since all the cases selected had cancer!

The few PS studies of the longitudinal kind (the cohort studies) might be expected to be more reliable, but those studies also rely on vague and unreliable individual recalls to reconstruct passive smoke exposure over lifetimes, and are plagued by the same biases and confounders that affect case-control studies. In the end, such studies remain equally inconclusive.

… and the famous "statistical error"

Large as they might be, epidemiologic surveys usually 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 off plus or minus a certain amount 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 judgment 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 of certainty, whereas if the error exceeds 5%, the results are said to be not statistically significant.

Statistical significance is important!

Statistical error is characterized by tests of significance defined as p-values or as 95% confidence intervals (*CI*). Statistical procedures calculate the p- value of estimated risks, with the threshold of significance being at p=0.05, which is the numerical representation of a 5% margin of error. Values lower than p=0.05 are

☞ **Statistical significance** – A numerical expression of the fact that the data of a study shows a risk increase OR decrease. If a study's data shows BOTH at the same time, this is considered to be an incoherent result. In layman's terms, such a study is meaningless. In epidemiology, the term used is **statistically insignificant**.

A study is statistically significant when all data shows either a risk or a benefit.

The majority of studies on passive smoke are statistically insignificant.

"Statistical significance" **does not** mean that the data are accurate, nor does it mean that the shown risk or benefit exists, nor does it mean that the risk or benefit is "big".

In short, "statistically significant" ***does not mean "significantly large risk"***, as antismoking propaganda lets people understand.

☞ **Confidence interval, CI, "p", level of certainty** – The margin of certainty that errors are not made. In the kinds of studies we examine here, 95% is considered acceptable. That means that it is OK to miss once every 20 times. Try that with the brakes of your car, or with your tax payments!

said to characterize statistically significant results at the 95% level, and vice-versa. Thus, a relative risk or odds ratio of 1.6 with a p-value of 0.03 is said to be statistically significant at the 95% level, whereas a relative risk of 2.3 with a p-value of 0.07 is not.

The 95% confidence interval is more informative than p-values, although based on the same concepts and calculations. In standard format it gives a range of values within which the statistically true value of a relative risk or odds ratio is likely to be located with a 5% probability of error and a 95% probability of certainty.

In interpreting a confidence interval it is important to recall how risk or odds ratios are calculated. For both indexes **a value of 1 means no change in risk** because incidence is the same in non-exposed and exposed subjects, or because exposure is the same in cases and controls. **Values below 1 imply risk reduction or protection, values above 1 imply increased risk.** The 95% confidence interval gives an immediate impression of statistical significance, which is characterized by confidence intervals whose values are all either above or below 1, that is all in negative or positive risk territory. By contrast**, intervals containing values above and below 1 define non-significant results, which remain moot because they simultaneously imply an increase and a decrease of risk**.[15]

Detailed accounts of epidemiologic methodologies for dealing with biases, confounders, standardizations, 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, or it turned left when attempting to steer right? Science valid enough for reliable applications must attain much lower margins of error, usually *far less than one in a million*.

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 PS 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.

[15] •*RR* or *OR* = 1.9 (95%CI 1.2-4.6) means that the best estimate of the risk may be 1.9, but that its true value could be between 1.2 and 4.6, with a probability of 95%. It also means that within that range all values are statistically significant at the 95% level, because all would mean an increase of risk, the lowest value still being >1.

•*RR* or *OR* = 1.9 (95%CI 0.7–2.3)) means that the best estimate of the risk may be 1.9, but that its true value could be between 0.7 and 2.3, with a probability of 95%. It also means that some values could be <1 and could mean protection, others could be >1 and could mean risk. As a consequence the result is said to be equivocal and not statistically significant.

•*RR* or *OR* = 0.7 (95%CI 0.2-0.9)) means that the best estimate of the risk may be 0.7, but that its true value could be between 0.2 and 0.9, with a probability of 95%. It also means that within that range all values are statistically significant at the 95% level, because all would mean a reduction of risk, the highest value still being <1.

•*RR* or *OR* = 0.7 (95%CI 0.3-1.9) means that the best estimate of the risk may be 0.7, but that its true value could be between 0.3 and 1.9, with a probability of 95%. It also means that some values could be <1 and could mean protection, others could be >1 and could mean risk. As a consequence the result is said to be equivocal and not statistically significant.

The dubious magic of meta-analysis

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

☞ **Meta-analysis** – A "study of studies". No direct study is performed on subjects, but a compendium of pre-existing studies is made and analysed. A meta-analysis is only good if **all** the studies done with the same methods ever performed are kept into account. This is **never** the case for passive smoke meta-analyses, where only certain studies are "cherry picked" in the attempt to demonstrate that passive smoke is a threat to public health.

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, standardizations, 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 standardize, relieve, or control for differential corruptions that may be present in each study. If characteristics other than study size are used in weighing studies (e.g. an estimate of study quality), those characteristics are likely discretionary, judgmental, and conducive to different meta-analysis results at the hands of different analysts.

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

Epidemiology – fuzzy at best

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 standardizations, prejudice, and more. Tests of statistical significance are equally speculative, being no more than approximate summaries of metaphorical primary data.

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 out of what is usually a congeries of data.

☞ **Data dredging** – The sifting of pre-existing data from studies for the purpose of finding associations that may not have been originally made in the studies. It's a way of recycling data to create new studies – almost instantly – at a low cost.

How do epidemiologists approach the inherent fragility of their data?

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 PS studies, alone or together come 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 PS studies, it is obvious that the associations claimed are extremely weak, as we can see from the following citations:

National Cancer Institute - *"In epidemiologic research, relative risks of less than 2 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 and 2 ... problems of interpretation may become acute, and it may be extremely difficult to disentangle the various contributions of biased information, confounding of two or more factors, and cause and effect."* - ("The Causes of Cancer," by Richard Doll, F.R.S. and Richard Peto. Oxford-New York, Oxford University Press, 1981, p. 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, p. 36

FDA - *"Relative risks of 2 have a history of unreliability"* - Robert Temple, M.D. Food and Drug Administration Journal of the American Medical Association (JAMA), Letters, September 8, 1999
FDA - *"My basic rule is if the relative risk isn't at least 3 or 4, forget it."* - Robert Temple, director of drug evaluation at the Food and Drug Administration.

Consistency of results from different studies is an obvious attribute of true causal relationships.

- Epidemiologic studies of PS are grossly inconsistent, and epidemiologic associations that are inconsistent are quite unlikely to be true.

Specificity requires that a cause leads to a single effect, which is seldom the case in multi-factorial epidemiology.

- PS has been claimed to cause many different effects.

Temporality. That effects must occur after the cause has a chance to act is a self-evident and trivial criterion of causality.

Dose-effect relationship is a useful criterion of causation, but does not resolve the matter.

- Such an effect is the exception in PS studies.

Plausibility. Whether an association is biologically plausible or not remains a matter of individual speculation and is far from being objective or conclusive.

Coherence. Agreement with other information may be a corollary attribute but not evidence of causation.

Experimental evidence. Experimental evidence in humans would indeed constitute proof of causation, but it is unavailable in the case of PS.

Analogy **is open to imagination and remains an invalid criterion of causation.**

Individual risk

Epidemiologic claims of risks allegedly associated with PS exposures cannot be applied to infer the risk of individuals, because of statistical and common sense considerations.

There are general arguments against such a transfer of risk. For instance, a polled group showing a 60% preference for proposition X cannot mean that single individuals in that group are 60% in favour of X, because each may have different levels of agreement, or may be either for or against the proposition. If an epidemiologic study should say that an exposed population might have the probability of a 20 % increased risk of a disease, it does not mean that every individual in the population has a 20% increased risk for the disease. Obviously not all individuals in the population will develop the disease and it is not possible to state in advance which ones will develop the disease or not. It can only be said that single individuals in the population will either develop the disease or not, namely that individual chances are either 100% or 0%.

PART III

EXAMINING THE EVIDENCE ON PASSIVE SMOKE

Part II has highlighted the critical benchmarks for evaluating the validity of PS epidemiologic studies, which are here summarized as follows:

1. A study must warrant that its numerical representations of individual lifetime PS exposure recalls are true measures of actual exposures.

2. A study must warrant that an exposure recall bias affects at the same rate cases and control groups, and exposed and non-exposed groups.

3. 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.

4. A study must warrant that known causal confounders are affecting at the same rate cases and control groups, and exposed and non-exposed groups.

5. A study must warrant the accuracy of pathological and diagnostic records.

6. The results from different studies addressing the same subject must be consistently reproducible.

7. In any study, the statistical margin of error of reported risks should reach no less than the 95% level of significance.

8. If the above criteria are met, the results of a study should also be consistent with Hill's criteria of causality.

9. Meta-analysis summations shall not be credible unless performed on the basis of all available studies, which studies also must be of homogeneous design and conduct , and must have met the above criteria of validity.

For sake of brevity and clarity we focus here on the epidemiologic studies of PS and lung cancer, which their sponsors claim to represent the best and strongest evidence of the risks of PS exposure. On this basis, a consideration of the reliability of claims for other conditions allegedly linked with PS exposure would be subordinate to the considerations for lung cancer.

The credibility of the above criteria is self evident, plainly understandable and acceptable to anyone interested in the verification of factual evidence. On this basis we challenge anyone to disagree with the following appraisal, which we affirm to be valid for any individual study or for any meta-analysis summation so far conducted.

1. It is incontrovertible that no extant study can warrant that the numerical representation of individual lifetime PS exposure recalls is a reliable measure of actual exposures.

2. It is incontrovertible that no extant study can warrant that PS exposure recall bias affects at the same rate cases and control groups, and exposed and non-exposed groups.

3. It is incontrovertible that no extant study can warrant that subject selection and misclassification biases (and other biases) are affecting at the same rate cases and control groups, and exposed and non-exposed groups.

4. It is incontrovertible that no extant study can warrant that known causal confounders affect at the same rate cases and control groups, and exposed and non-exposed groups.

5. It is incontrovertible that no extant study has warranted the accuracy of pathological and diagnostic records.

6. It is incontrovertible that results from different studies addressing the same subject have been grossly inconsistent and not reliably reproducible.

7. It is incontrovertible that only for a random minority of studies the numerical margin of error of reported risks has been at or below the 95% confidence level of statistical significance.

8. It is incontrovertible that no study of PS has met Hill's criteria of causality.

9. It is incontrovertible that no meta-analysis summation of PS studies has been performed on the basis of all available studies, of studies that are of homogeneous design and conduct, and of studies that have met the above criteria of validity.

A detailed consideration of each individual study of PS and lung cancer would make this report inaccessible to non-specialists. Upon request we pledge to offer a detailed analysis of each and every study here listed, in regard to the affirmations above.

However, the issues of consistency of results and of statistical significance can be grasped and presented immediately with a simple listing of studies and their reported risks, as in the following pages. Graphic summaries are also provided after the listing.

The reader is reminded that statistical significance relates only to the numerical context of a study, and simply refers to the numerical range of error of a particular estimate. No statistical manipulation can improve the quality of the collected data, and therefore, if the data are unreliable and corrupt any derived statistical estimate remains equally unreliable and corrupt.

For instance, no statistical manoeuvre could improve the lack of reliability of lifetime PS exposure recall answers from individual study subjects. It follows that **a statistically significant risk elevation gives no assurance of being credible**. On the other hand, a statistically not significant estimate, whether of elevated or reduced risk, remains ***doubly unreliable***: first because of the unreliability of the underlying data, and second because of the margin of numerical error is not acceptable. In any event, larger studies have a statistical advantage, because the extent of numerical error usually correlates inversely with the number of subjects in any given study.

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

SPOUSAL STUDIES
Studies of non-smoking spouses living with smoking husbands, wives, or partners.

WORKPLACE STUDIES
Studies of non-smokers working in occupational settings where smoking is allowed.

CHILDHOOD STUDIES
Studies of non-smokers exposed to PS in their homes during childhood-adolescence.

TABLE 1 – ALL THE STUDIES ON PASSIVE SMOKE AND LUNG CANCER

COLOUR CODING AND NOTATIONS:

Not statistically significant risk elevation [invalid study]
Not statistically significant risk reduction (protection) [invalid study]
Statistically significant risk elevation
Statistically significant risk reduction (protection)

STUDIES ON PASSIVE SMOKE 1981-2003

^ = Figures from Final Report CALEPA 1997
^^ = Figures from Final Report CALEPA 2003
SG = 1986 Surgeon General's Report
( ) = Estimated
NR = No Risk. Reported as having no correlation, namely RR=1.00.
See bibliographic references at page 47

SPOUSAL STUDIES

Studies and Authors	Year	Nation	Sex	Number of lung cancers	Relative Risk (i.e. 1.18=18% risk elevation)	95% Confidence Interval
Garfinkel et al. 1 (SG)	81	United States	F	153	1.18	0.90-1.54
Chan et al. SG	82	Hong Kong	F	84	0.8	0.43-1.3
Correa et al. (SG)	83	United States	F	22	2.07	0.81-5.25
Correa et al.(SG)	83	United States	M	8	1.97	0.38-10.32
Trichopouls et al. (SG)	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. (SG)	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. 1(SG)	84	United States	F	24	0.79	0.25-2.45
Kabat et al. 1(SG)	84	United States	M	12	NR	0.2-5.07
Garfinkel et al. 2(SG)	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. (SG)	85	United States	F	29	1.4	0.4-4.2
Akiba et al. (SG)	86	Japan	F	94	1.5	0.9-2.8
Akiba et al. (SG)	86	Japan	M	428	1.8	0.4-7.0
Lee et al. (SG)	86	United Kingdom	F	32	NR	0.37-2.71
Lee et al. (SG)	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
Lam T et al.	87	Hong Kong	F	199	1.65	1.16-2.35
Pershagen et al. (SG)	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 ^	92	United States	F	431	NR	0.80-1.2
Stockwell et al. ^	92	United States	F	62	1.6	0.80-3.0
Liu Q et al. ^	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. ^	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 ^	95	United States	F	67	1.08	0.60-1.94
Kabat et al. 2 ^	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. ^	96	United States	F	175	1.1	0.72-1.68
Schwartz et al. ^	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. ^ ^^	97	United States	F	150	1.2	0.80-1.6
Cardenas et al. ^ ^^	97	United States	M	97	1.1	0.60-1.8
Jöckel-BIPS ^^	97	Germany	F	53	1.58	0.74-3.38
Jöckel-BIPS ^^	97	Germany	M	18	1.58	0.52-4.81
Jöckel-GSF ^^	97	Germany	F	242	0.93	0.66-1.31
Jöckel-GSF ^^	97	Germany	M	62	0.93	0.52-1.67
Ko et al. ^ ^^	97	Thailand	F	105	1.3	0.7-2.5
Nyberg et al. ^^	97	Sweden	F	89	1.2	0.74-1.94
Nyberg et al. ^^	97	Sweden	M	35	1.2	0.57-2.55
Jockel et al. ^^	98	Germany	F&M	71	1.12	0.54-2.32
Nyberg et al. ^^	98a	Sweden	F	89	1.05	0.6-1.86
Nyberg et al. ^^	98a	Sweden	F&M	58	1.17	0.73-1.88
Boffetta et al. (meta-analysis) ^^	98	***Nation***	***Sex***	***Cancers***	***RR***	***CI 95%***
		Europe	F	508	1.15	0.86-1.55
		Sweden	F&M	70	2.29	0.65-8.07
		Germany 1	F&M	76	0.88	0.40-1.95
		Germany 2	F&M	142	1.22	0.66-2.2
		Germany 3	F&M	31	2.01	0.71-5.67
		England	F&M	26	1.38	0.43-4.28
		France	F&M	77	0.72	0.36-1.25
		Portugal 1	F&M	49	2.04	0.71-5.8
		Portugal 2	F&M	33	2.03	0.76-5.38
		Spain	F&M	71	1.1	0.48-2.68
		Italy 1	F&M	40	0.73	0.28-1.65
		Italy 2	F&M	19	1.12	0.35-3.56
		Italy 3	F&M	16	1.36	0.30-6.45
Zaridze et al. ^^	98	Russia	F	189	1.53	1.06-2.21
Jee et al. ^^	99	Korea	F	79	1.9	1.0-3.5
Rapiti et al. ^^	99	India	F	52	1.2	0.5-2.9
Zhong et al. ^^	99	China	F	504	1.1	0.7-1.7
Lee et al. ^^	00	Taiwan	F	186	1.2	0.7-2.0
Wang et al. ^^	00	China	F&M	200	1.19	0.7-2.0
Kreuzer at al. ^^	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. ^^	01	Canada	F	56	1.2	0.5-3.0
Nishino et al. ^^	01	Japan	F	23	1.8	0.67-4.6

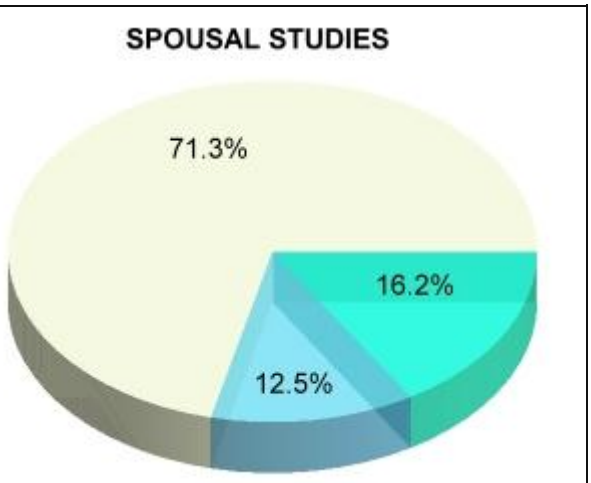
WORKPLACE STUDIES

Studies and Authors	Year	Nation	Sex	R.R.	95% C.I.
Kabat et al. 1 ^	84	United States	F	0.70	0.30-1.50
Kabat 1 et al. ^	84	United States	M	3.3	1.1-10.4
Garfinkel 2 ^	85	United States	F	0.93	0.7-1.2
Wu et al. ^	85	United States	F	1.3	0.5-3.3
Lee et al. ^	86	United Kingdom	F	0.63	0.17-2.33
Lee et al. ^	86	United Kingdom	M	1.61	0.39-6.6
Koo et al. ^	87	Hong Kong	F	0.91	0.15-5.37
Shimizu et al. ^	88	Japan	F	1.18	0.70-2.01
Janerich et al. ^	90	United States	F&M	0.91	0.80-1.04
Kalandidi et al. ^	90	Greece	F	1.39	0.80-2.5
Wu-Williams et al. ^	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. ^	92	United States	F	NR	NS
Fontham et al. ^	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 ^	95	United States	F	1.15	0.62-2.13
Kabat et al. 2 ^	95	United States	M	1.02	0.5-2.09
Schwartz et al. ^	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 ^^	97	Germany	F&M	2.37	1.02-5.48
Jockel-GSF ^^	97	Germany	F&M	1.51	0.95-2.4
Ko et al. ^ ^^	97	Thailand	F	1.1	0.40-3.0
Nyberg et al. ^^	98a	Sweden	F&M	1.61	0.91-2.85
Zaridze et al. ^^	98	Russia	F	0.88	0.55-1.41
Boffetta et al. (WHO) ^^	98	Europe	F&M	1.17	0.94-1.45
Zhong et al. ^^	99	China	F	1.7	1.30-2.3
Kreuzer et al. ^^	98/00	Germany	F	1.03	0.78-1.36
Lee et al. ^^	00	Taiwan	F	1.2	0.50-2.4
Johnson et al. ^^	01	Canada	F	1.21 1.71	0.50-2.8 min. 0.7-4.3 max.

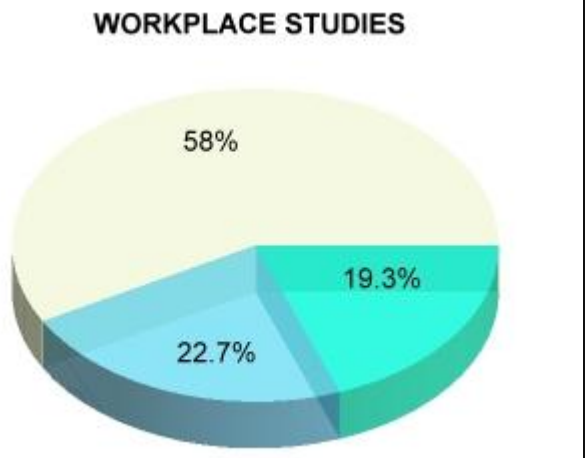
CHILDHOOD STUDIES

Studies and Authors	Year	Nation	Sex	R.R.	95% C.I.
Correa et al. SG	83	United States	F	NR	NS
Kabat & Wyn ^	84	United States	F	0.92	0.40-2.08
Kabat & Wyn ^	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. (SG)	85	United States	F	0.6	0.20-1.7
Akiba et al. SG	86	Japan	F&M	NR	NS
Gao et al. ^	87	China	F	1.1	0.7-1.7
Koo et al. ^	87	Hong Kong	F	1.73	0.6-6.4
Pershagen et al. ^	87	Sweden	F	NR	0.4-2.3
Svensson et al. ^	89	Sweden	F	3.3	0.5-18.8
Janerich et al. ^	90	United States	F&M	1.09	0.68-1.73
Sobue et al. (^)	90	Japan	F	1.28	0.71-2.31
Wu-Will et al. (^)	90	China	F	NR	NS
Brownson et al. 2 ^	92	United States	F	0.8	0.60-1.1
Stockwell et al. ^	92	United States	F	1.1	0.50-2.6
Fontham et al. ^	94	United States	F	0.89	0.72-1.1
Zaridze et al.	94	Russia	F	0.98	0.66-1.45
Kabat 2 ^	95	United States	M	0.9	0.43-1.89
Kabat et al. 2 ^	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 ^^	97	Germany	F&M	1.05	0.50-2.22
Jockel-GSF ^^	97	Germany	F	0.95	0.64-1.4
Ko et al. ^ ^^	97	Thailand	F	0.80	0.4-1.6
Boffetta et al. (WHO) ^^	98	Europe	F&M	0.78	0.64-0.96
Jockel et al. ^^	98	Germany	F&M	2.02	0.60-6.75
Nyberg et al. ^^	98a	Sweden	F&M	1.02 0.72	0.63-1.66 father 0.28-1.87 mother
Zhong et al. ^^	99	China	F	0.9	0.50-1.9
Rapiti et al. ^^	99	India	F	3.99	1.90-8.2
Kreuzer et al. ^^	98/00	Germany	F	1.03	0.78-1.36
Lee et al. ^^	00	Taiwan	M	1.7	1.10-2.6 father
			F	0.9	0.30-3.1 mother
Wang et al. ^^	00	China	F&M	1.52	1.1-2.2
Rachtanet al. ^^	01	Poland	F	3.31	1.26-8.69
Johnson et al. ^^	01	Canada	F	0.54	0.1-2.7

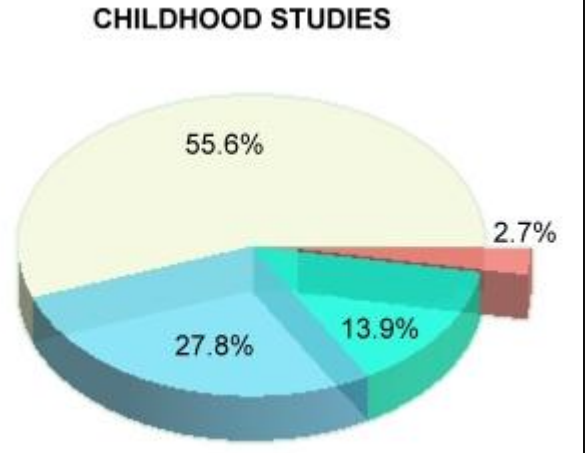
Coding	Total number of spousal studies: 80	Perc.
	Not statistically significant risk elevation: **57**	71.3%
	Not statistically significant risk reduction (protection): **10**	12.5%
	Statistically significant risk elevation: **13**	16.2%



Coding	Total number of workplace studies: 31	Perc.
	Not statistically significant risk elevation: **18**	58%
	Not statistically significant risk reduction (protection): **7**	22.7%
	Statistically significant risk elevation: **6**	19.3%



Coding	Total number of childhood studies: 36	Perc.
	Not statistically significant risk elevation: **20**	55.6%
	Not statistically significant risk reduction (protection): **10**	27.8%
	Statistically significant risk elevation: **5**	13.9%
	Statistically significant risk reduction (protection): **1**	2.7%



CONCLUSION

In considering the graphs above 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 a negative association. Such a bias, therefore, did shrink by an immeasurable but significant amount the true extent of the green and red sections in the graphs above.

The tables above clearly indicate the flimsiness of the risk values reported, which are most likely the result of the recall and misclassification biases that intuitively must be more pronounced among cases than among controls, and of the influence of lung cancer causal confounders that by necessity must be more prevalent among subjects that have lung cancer.

Indeed, 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 PS exposures could not possibly reach verifiable conclusions. Instead, a dogged determination to fabricate risks at any cost has produced such a sequence of impotent studies.

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 section I 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 PS and lung cancer, that error is compounded by the outcome discrepancy 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, 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?

The data of the studies are not only of undefined quality, but are most certainly corrupted. Would anyone accept to drive a car for which there is no guarantee that the engine or the chassis are made of durable materials and not – say – of cardboard?

Is it rationally defensible, fair, and legitimate for governments in democratic societies to use such data as above to falsely raise public anxieties, and to enforce Draconian policies that victimize millions of citizens, and trample and restrict their recognized freedoms?

APPENDIX I

SOME LUNG CANCER RISKS INDEPENDENT OF CIGARETTE SMOKING

The epidemiologic literature reports many risk factors for lung cancer that are independent of cigarette smoking. In other words, they are independent lung cancer risks for smokers and non-smokers alike. Hence, epidemiologic studies of the association of lung cancer with active smoking or with environmental tobacco smoke must investigate and measure the interference of any and all independent risk factors. These factors carry the appropriate technical designation of confounders, and it is an illegal technical and professional procedure to draw conclusions from a study without a meticulous accounting of such confounders.

Active cigarette smoking. Obviously it is not possible to draw conclusions about the magnitude of cigarette smoking risk for lung cancer without a reasonably accurate account of the possible influence of confounders. Yet, none of the studies of cigarette smoking and lung cancer reported by the U.S. Surgeon General (USSG, 1979) have taken into account any of the confounders here listed. The likely reason is that most of those studies were performed in the '50s and '60s, when the confounding risk factor here listed had yet to be reported.

Thus, it if it were possible to account for the inevitable interferences of confounders, the magnitude of the relative risk and the numbers of lung cancers now attributed to smoking would have to be corrected downward, and likely substantially so. Sir Richard Doll, the dean of antismoking epidemiologists, acknowledged the discrepancy as he wrote: "*...[active] smoking seems to act synergistically with other aetiologic agents such as consumption of alcohol; various aspects of the diet; levels of blood pressure, blood lipids, or other cardiovascular risk factors; or exposure to asbestos, radon, or possibly some infective factors. The quantitative effect of smoking will, therefore, vary with variation in the prevalence of these other factors.*" (Doll et al., 1994). Although Doll did not mention all the risk factors here listed, his admission is tantamount to stating that the magnitude of the risk and the number of lung cancer cases that could be attributed to cigarette smoking remain unknown.

Environmental tobacco smoke (ETS). Even if one were to use the illegal statistical and epidemiologic distortions introduced by the U.S. 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 were 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 here listed 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 absolutely moot any interpretation of the possible association of ETS exposure and lung cancer.

How to read the following table. The first column 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 complete bibliography of each study can be found alphabetically by author in the list of references.

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 RIKS 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)
β-carotene/ vitamin A deficiency	Ziegler (1986)	2.2	
β-carotene/ vitamin A	Wu (1985)	0.3	(P=0.06 trend)
	Byers (1987)	0.2	
intake	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	Wynder (1987)	4-6	
intake	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 (1994)	0.32	(0.13-0.78)
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)

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	Hayes (1989)	1.5	(1.2-1.9)
exposure	Jacobsson (1997)	2.0	(1.5-2.6)
	Gustavsson (1990)	2.4	(1.3-4.5)
Socioeconomic class	Brown (1975)	2.6-3.8	
Ventilatory function	Lange (1990)	2-4	
Cardiac anomalies	Tenkanen (1987)	2.4	
Physical	Albanes (1989)	1.6	(1.2-3.5)
inactivity	Severson (1989)	1.4	(1.0-2.1)
Psychosocial	Kulessa (1989)	2-3	
traits	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 and 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)
Beer drinking	Potter (1992)	2.0	(1.02-3.8)

APPENDIX II

SOME NOTEWORTHY STUDIES

THE ENVIRONMENTAL PROTECTION AGENCY STUDY

The 1993 EPA study of PS is considered a benchmark for antismoking policies. Yet, this study ranks very low in reliability, for the following reasons:

- It is a meta-analysis.
- It did not consider all the studies ever performed on PS, but only some, "cherry-picked" amongst those that leaned most towards the existence of a risk.
- The confidence interval was lowered to 90%, in the attempt to emphasize risk.
- Even the EPA consultants recommended against classifying passive smoke as a carcinogen (see original documentation at: http://www.forces.org/evidence/files/epa-epid.htm, and http://www.forces.org/evidence/files/epa-asse.htm).
- In spite of all of the above, the resulting RR was only 1.19 (19% risk elevation) – well below any threshold of epidemiological credibility.
- The study was essentially declared a methodological fraud *by a U.S. Federal Court* in 1998 (http://www.forces.org/evidence/epafraud/etsfrau.htm), which also ordered the removal of passive smoke from the "Class A" list of carcinogens.
- The claim of 3,000 estimated "deaths caused by passive smoke" was also vacated by the decision.

Later, the US Court of Appeal overruled the decision of the Federal Court on grounds of juridical jurisdiction. However, the Court of Appeal specifically stated that it would not question the scientific merits of the Federal Court decision.

THE IARC STUDY

The 2002 International Agency for Research on Cancer Monograph Vol. 83 "Tobacco Smoke and Involuntary Smoking" study is of the same kind as the EPA study mentioned above – a meta-analysis of cherry-picked studies previously performed. In June 2002, the IARC announced that "passive smoke causes cancer" and dared to state that a causal link was established. Again, only about 50 cherry-picked studies from more than 100 that had been performed on passive smoke and cancer were selected, and the study has been published only very recently. In the press release announcing publication (http://www.iarc.fr/pageroot/PRELEASES/pr152a.html), the IARC states that the study "puts a final stop to all controversies fuelled at various degrees by the tobacco industry".

In the 1986 IARC Monographs on the Evaluation of the Carcinogen Risks of Chemicals to Humans, Tobacco Smoking, Volume 38, page 308, 10-12 February, 1985, the IARC stated:

"Several epidemiological studies have reported an increased risk of lung cancer in non-smoking spouses of smokers, although some others have not. In some studies, the risk of lung cancer in non-smokers increased in relation to the extent of spouses' smoking. Each of the studies had to contend with substantial difficulties in determination of passive exposure to tobacco smoke and to other possible risk factors for the various cancer studied. The resulting errors could arguably have artefactually depressed or raised

estimated risks, and, as a consequence, each is compatible either with an increase or with an absence of risk. As the estimated relative risks are low, the acquisition of further evidence bearing on the issue may require large-scale observational studies involving reliable measures of exposure both in childhood and in adult life. The studies on childhood cancer do not provide clear evidence as to whether or not there is a clear association with parental smoking."

We see in the table in Section II that, up to 1985, only 10 studies had been performed and, of those, only four were statistically significant. Of those, three showed an RR greater than 3. Since 1986 over 100 studies have been performed on passive smoke on all kinds of populations. Only 20 are statistically significant. Of those, nine show an RR greater than 2, and only two report a RR greater than 3. Furthermore, the largest studies performed since 1986 (amongst them the non-meta-analytical WHO-IARC "Multicenter case-control study of exposure to environmental tobacco smoke and lung cancer in Europe" - Journal of the National Cancer Institute, Vol. 90, No. 19, October 7, 1998, and "Environmental Tobacco Smoke and Tobacco Related Mortality in a Prospective Study of Californians, 1960-98", BMJ Volume 326, 17 May 2003) indicated no connection between passive smoke and disease.

Therefore, the numerous studies that have been amassed since 1986 have demonstrated more than abundantly the lack of evidence of connection between passive smoke and disease.

The position of IARC (based on a mere 25% risk elevation – that is, RR 1.25) can therefore only be explained with a political posture supportive of prohibition, and not as founded on science.

THE ENSTROM AND KABAT STUDY

In May 2003 Enstrom and Kabat "shook the floors" of the international antismoking establishment by publishing one of the largest studies ever conduced on passive smoke: *Environmental tobacco smoke and tobacco related mortality in a prospective study of Californians, 1960-98*. The study was a longitudinal one, examining over 118,000 people from 1960 to 1998. *"The results do not support a causal relation between environmental tobacco smoke and tobacco related mortality, although they do not rule out a small effect. The association between exposure to environmental tobacco smoke and coronary heart disease and lung cancer may be considerably weaker than generally believed,"* says the abstract.

In spite of its enormous size and duration, the study was not statistically significant. Enstrom and Kabat proved conclusively the only thing that seems can be proven about passive smoke: its "dangers" cannot be demonstrated.

Started under the auspices of the American Cancer Society in 1959 with a one million dollar start-up fund, the huge study examined and followed 118,000 people, 35,561 of them non-smokers. The data was collected for about 40 years. The study continued under the auspices of the State of California, which granted some funds from the Tobacco-Related Disease Research Program (Proposition 99) – a tax system that uses the super-tax on cigarettes to finance antismoking programs and propaganda. In 1999 it became clear that the data was not showing the "expected" results. In spite of tremendous pressures to suppress the study, the two scientists refused to do so, and the funds from

Proposition 99 suddenly dried up. At this point, Enstrom and Kabat had no choice but to turn to the Center for Indoor Air Research – a group supported by the tobacco industry – to obtain the $75,000 necessary to complete the study. After that, the financing of the tobacco industry was used as a reason by all scientific journals not to publish the results. Fortunately, the British Medical Journal eventually accepted the study for publication. However, Richard Smith, the editor of BMJ, instantly went *"From hero to pariah in one easy jump",* as he himself wrote on May 17, 2003 (http://bmj.bmjjournals.com/cgi/eletters/326/7398/1057#32390): *"Not long ago I was something of a hero of the antitobacco movement-- because I resigned my professorship at Nottingham University when it accepted money from British American Tobacco. I felt somewhat embarrassed by the whole episode. I was no hero. But now I'm a pariah for publishing a piece of research funded by the tobacco industry"*.

THE LUFTHANSA FLIGHT ATTENDANT STUDY

A sizeable longitudinal study that went unreported by mass-media was published by the American Journal of Epidemiology on May 1st, 2002: *Mortality from Cancer and Other Causes among Airline Cabin Attendants in Germany, 1960–1997* (study available at: http://193.78.190.200/39/cabin.pdf). Completed in 1997, the study followed 16,014 women and 4,537 men, all Lufthansa flight attendants, for 37 years, monitoring for lung cancer and cardiovascular disease. The peculiarity of this study is that it concerns the confined air cabin environment where fight attendants were exposed throughout their professional lives to passive smoking. (It is worth pointing out that, since smoking has been banned in airplanes, the air quality has significantly decreased because of the diminished air exchange.)

The study concluded: "We found a rather remarkably low SMR [standardized incidence ratio] for lung cancer among female cabin attendants and no increase for male cabin attendants, indicating that smoking and exposure to passive smoking may not play an important role in mortality in this group. Smoking during airplane flights was permitted in Germany until the mid-1990s, and smoking is still not banned on all charter flights. The risk of cardiovascular disease mortality for male and female air crew was surprisingly low (reaching statistical significance among women)."

This study is not an isolated case. Other studies on airline cabin environment and passive smoke show similar results (See Appendix V).

THE 1989 US DOT STUDY

Although not an epidemiological study, this study, *Airliner Cabin Environment: Contaminant Measurements, Health Risks, and Mitigation Options* (US Department of Transport, P-15-89-5, available at http://193.78.190.200/dot/dot-p-15-89-5.pdf), gives us perhaps the best intuitive dimension of the dilution of passive smoke in the environment. This direct-measurement study states that a passenger sitting in the row bordering with the smoking section would have to fly non-stop for 48,440 hours (5 ½ years) to inhale the equivalent of one cigarette. The study also compares passive smoke with the exposure to cosmic rays (a bombardment we are all exposed to, even in caves), and states that cosmic rays constitute a risk of cancer from 150 to 641 times higher than passive smoking. The political conclusion of the study, however, was that smoking should be banned from all flights!

OTHER ISSUES

FINE RESPIRABLE PARTICLES IN PASSIVE SMOKE

Passive smoke is not clean air - nor is the air we breathe outside. In fact, “clean air” never existed, even in antiquity. However, passive smoke’s toxicity 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 says. As activists claim that “there is no safe level of the exposure to passive smoke”, it conveniently follows that the only acceptable level of exposure is **zero**. [16] 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, all of us would 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.

Air ventilation 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.

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

Estimated number of cigarettes required to reach TLV levels from side stream smoke emission of selected chemicals in a sealed and unventilated 100 m^3 enclosure (Gori and Mantel, 1991)
(Please note: 100 m^3 are equivalent to a room 22' x 21' x 8' ceiling, or 3,696 cubic feet) - Click here to download the study.

Side Stream Smoke Component [*]	Side Stream Smoke output mg/cigarette [*]	Safety Limit mg/m^3 [**]	Cigarettes required to reach safety limit
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 [***]	222,000
2-Toluidine	0.003	9.00	300,000

[16] Sometimes the claims of antitobacco “experts” on passive smoke are quite risible. Mr. 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 of the pharmaceutical conglomerates for the purpose of banning smoking. In March 2004 he claimed that **100,000** air changes per hour would be necessary (“a tornado”, he said) in a room to bring passive smoke 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 January 24th of the same year!

Polonium 210	**0.4pCi**	**3pCi/l** [****]	**750,000**
Toluene	**0.000035**	**375.00**	**1,000,000**

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

[*] 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 (in short, before becoming real "passive smoke"). But it is very unlikely that non-smokers stand motionless 4 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.
[**] EPA data1990a, Table C-2, pages C-19, 20
[***] Based on the lower exposure limit for tar volatile compounds
[****] EPA 1990b

If the case above 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 - 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, passive smoke, and from a large number of natural 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 "passive smoke" 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 EPA reassessed its estimate on the negative impact of the PM on human health, by greatly reducing its assessment of that impact. That information, however, is not advertised by antismoking propaganda, as it would adversely affect the belief that smoking kills in any form.

If fine particles were 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 great majority of even heavy smokers get to a healthy old age. How is that possible? There are only two explanations: either smokers have a special, superior genetic make-up (a ridiculous concept), or the impact on health of fine particles is grossly exaggerated.

TABLE 4 - *Relative dose estimate of RSP in typical active smokers and passive smoke* (*ETS) exposed non-smokers*

SMOKER	**30 cigarettes per day** **15 mg** RSP inhaled per cigarette Lung retention efficiency**: 90%** **DAILY DOSE** about ***400 mg***
NON SMOKER EXPOSED TO PASSIVE SMOKE	0.05 mg RSP/cubic meter of air 1.5 hours per day exposure 0.7 cubic meters per hour inhaled Lung retention efficiency**: 10%**

DAILY DOSE about ***0.00525 mg***

DOSE RATIO 0.00525 : 400 = about 1 : 75,000

From Luik and Gori, 1999

Even if we assume a 10 hour-a-day exposure instead of the 1.5 the table refers to (concerning mainly non smokers exposed occasionally, i.e., in restaurants), that 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.

THE VENTILATION SOLUTION

With such low level of pollutants it becomes apparent that the most important "pollutant" of passive smoke is the annoyance factor. That can be easily solved with 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* demonstrate 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.

It is clear that separation of smokers from non-smokers combined with air exchange technology is a complete solution to this largely artificial problem. All it takes is regulating authorities setting the standards for indoor air quality on passive smoke, and the technology does the rest. *Such 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 technical: they are political.

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. Let us look at some examples of statistical risk elevation concerning everyday life:

TABLE 5 - FEARING EVERYDAY RISKS?

Exposure and disease	Relative Risk (RR)	Risk elevation in %
Passive smoke and lung cancer	1.19 (EPA) 1.25 (IARC)	19% (EPA) 25% (IARC)
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	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	from 2 to 4	from 100% to 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	12.500	Over 12,000% (twelve thousand)

(Chart from *"Science Without Sense – The Risky Business of Public Health Research",* by Steven Milloy. Cato Institution, 1997, p. 14)

Where does this end? Should we forbid, control, regulate everything? The above chart illustrates how common activities show what, for the layman, would appear intuitively to be "high risk" indicators. Incidentally, the risks above are calculated with the identical "scientific" methodology used for passive smoke. 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 is yes. Where causality is multi-factorial, 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, should somebody arbitrarily determine that 30% risk elevation from three cups of coffee a week is less dangerous than 30% risk elevation from passive smoke? Perhaps it's time to claim the middle ground of common sense.

REFERENCES OF ADDITIONAL INTEREST

ACGIH (1990): American Conference of Governmental and Industrial Hygienists: Documentation of the Threshold Limit values and Biological Exposure Indices. 5th Edition and supplements. ACGIH, 6500 Glenway Ave. Building D-7, Cincinnati, OH 45211-4438.

Adami H, et al. (1989):Risk of cancer in women receiving hormone replacement therapy. Int J Cancer, 44:833-839.

Akiba S, et al. (1986): Passive Smoking and Lung Cancer Among Japanese Women. Cancer Res, 46:4804-4807.

Alavanja M (1993): Saturated fat intake and lung cancer risk among nonsmoking women in Missouri. J Nat Cancer Inst, 85(23):1906-1916.

Albanes D (1989): Physical Activity and the Risk of Cancer in the NHANES I Population. Am J Pub Health, 79:744-750.

Albert R (1989): Carcinogen risk Assessment. Environ Health Perspect, 81:103-105.

Altshuler B (1989): Quantitative models for lung cancer induced by cigarette smoke. Environ Health Perspect, 81:107-108.

Agudo A, et al. (1997): Vegetable and Fruit Intake and the Risk of Lung Cancer in Women in Barcelona, Spain. Eur J Cancer, 33:1256-1261.

Angell M (1996): Science on trial. W.W. Northon & Co. New York.

Angerer J (1992): Internal exposure to organic substances in a municipal waste incinerator. Int Arch Occup Environ Health, 64:265-273.

Armitage A, et al. (1997): Environmental Tobacco Smoke: Is It Really a Carcinogen? Med Sci Res, 25:3-7. Axelsson G, et al. (1996): Dietary Factors and Lung Cancer Among Men in West Sweden. Int J Epidemiol, 25:32-39.

Baker RR, Proctor CJ (1990): The origins and properties of environmental tobacco smoke. Envir Intern, 16:231-245.

Benowitz NL, et al. (1991): Stable isotope studies of nicotine kinetics and bioavailability. Clin Pharmacol Ther 49:270-277
Blair A, et al. (1995): "Guidelines for Application of Meta-Analysis in Environmental Epidemiology," Regul Toxicol Pharmacol, 22:189-197.

Boffetta P, et al. (1998): Multicenter case-control study of exposure to environmental tobacco smoke and lung cancer in Europe. J Nat Cancer Inst, 90:1440-1450.

Boffetta P, Tredaniel J, Greco A (2000): Risk of childhood cancer and adult lung cancer after childhood exposure to passive smoke: a meta-analysis. Environ Health Perspect 108:73-82.

Boffetta P, Aguado A, Ahrens W, Benhamou E, Benhamou S, Darby SC, *et al.* (1998). Multicenter case-control study of exposure to environmental tobacco smoke and lung cancer in Europe. J Natl Cancer Inst 90(19):1440-50.

Brandt AM (1998): Blow some my way: passive smoking, risk and American culture. In: Ashes to ashes: the history of smoking and health. Lock S, et al., Eds., pp. 164-187. Radopi B.V., Amsterdam & Atlanta.

Breslow N, Day N (1980): Statistical Methods in Cancer Research: Volume 1 -- The Analysis of Case-Control Studies, International Agency for Research on Cancer, Scientific Publications, No. 32, Lyon, France.

Breslow N, Day N (1987): Statistical Methods in Cancer Research: Volume 2 -- The Design and Analysis of Cohort Studies. International Agency for Research on Cancer, Scientific Publications, No. 82, Lyon, France.

Bretthauer EW (1992): Assistant Administrator for Research and Development, EPA. Letter to JJ Tozzi. December 17, 1992, Washington DC.

Brown S, et al. (1995): The Association of Economic Status With the Occurrence of Lung Cancer. Cancer, 36:1903-1911.

Brownson R, et al. (1987): Risk Factors for Adenocarcinoma of the Lung. Am J Epidemiol, 125:25-34.

Brownson RC, et al. (1992): Passive smoking and lung cancer in nonsmoking women. Am J Publ Health, 82:1525-1530.

Brownson RC, et al. (1993): Reliability of passive smoke exposure histories in a case control study of lung cancer. Intern J Epidemiol, 22:804-808

Brownson R, et al. (1997): Family History of Cancer and Risk of Lung Cancer in Lifetime Non-Smokers and Long-Term Ex-Smokers. Int J Epidemiol, 26(2):256-263.

Brugnone F, et al. (1992): Reference values for blood benzene in the occupationally unexposed general population. Int Arch Occup Environ Health, 64:179-184.

Buffler P, et al. (1984): The Causes of Lung Cancer in Texas. In: Lung Cancer: Causes and Prevention (Mizzell M, Correa P, Eds.):, pp. 83-99. Verlag Chemie International New York.

Butler T (1988): The Relationship of Passive Smoking to Various Health Outcomes Among Seventh-Day Adventists in California. Ph.D. Dissertation, University of California, Los Angeles, California.

Byers T, et al. (1987): Diet and Lung Cancer Risk: Findings from the Western New York Diet Study. Am J Epidemiol, 125:351-363.

California Environmental Protection Agency. Health effects of exposure to environmental tobacco smoke. *Office of Environmental Health Hazard Assessment, Final Report 1997.* California Environmental Protection Agency. Sacramento, CA.

California Environmental Protection Agency. Proposed identification of Environmental Tobacco Smoke as a toxic air contaminant. Part B: Health effects. November 2003. California Environmental Protection Agency. Sacramento, CA. http://www.arb.ca.gov/toxics/ets/dreport/bcovertoc1.pdf

Candelora E, et al. (1992): Dietary Intake and Risk of Lung Cancer in Women Who Never Smoked. Nutr Cancer, 17(3):263-270.

Cardenas V, et al. (1997): Environmental Tobacco Smoke and Lung Cancer Mortality in the American Cancer Society's Cancer Prevention Study II. Cancer Causes Control, 8:57-64.

CEPA (1997): Health effects of exposure to environmental tobacco smoke. Final Report. California Environmental protection Agency. Office of Environmental Hazards Assessment. Sacramento, California.

Chan W, Fung S (1982): Lung Cancer in Non-Smokers in Hong Kong. In: Cancer Campaign, Volume 6, Cancer Epidemiology (Grundmann E, Ed.):, pp. 199-202, Gustav Fischer Verlag, Stuttgart, Germany.

Chen C (1990): Epidemiologic Characteristics and Multiple Risk Factors of Lung Cancer in Taiwan. Anticancer Res, 10:971-976.

Chen T (1989): A Review of Methods for Misclassified Categorical Data in Epidemiology. Statistics in Med, 8:1095-1106.

Choi SY, et al. (1989): A Case-Control Study on Risk Factors in Lung Cancer. Korean J Epidemiol, 11:66-80.

Collier AM, et al. (1992): Cotinine elimination in young children as a function of age, sex, and race following ETS exposure (Abstract). Am Rev Respir Dis 145(4pt2):532A.

Copas JB, Shi JQ (2000): Reanalysis of epidemiological evidence on lung cancer and passive smoking. Br Med J 320:417-418.

Correa P, et al. (1983): Passive Smoking and Lung Cancer. Lancet, 2:595-597.

Coultas DB, et al. (1990a): Variability of measures of exposure to environmental tobacco smoke in the home. Am Rev Respir Dis, 142:602-606.

Coultas DB, et al. (1990b): A personal monitoring study to assess the workplace exposure to environmental tobacco smoke. Am J Publ Health, 80:988-990.
Cress RD, et al. (1994): Characteristics of women nonsmokers exposed to passive smoke. Prev Med, 23:40-47.

Crouse W (1988): Results from a survey of environmental tobacco smoke in restaurants. Presented at the APCA International Conference, Niagara Falls, NY.

Dai XD, et al. (1996): The Etiology of Lung Cancer in Nonsmoking Females in Harbin, China. Lung Cancer, 14(Suppl.1):S85-91.

Daling JR, et al. (1994): Risk of breast cancer among young women: relationship to induced abortion. J Natl Cancer Inst, 86:1584-92.

Delfino RJ, et al. (1993): Questionnaire assessment of recent exposure to environmental tobacco smoke in relation to salivary cotinine. Eur Respir J, 6:1104-1108.

Dement J, et al. (1994): Follow-Up Study of Chrisotyle Asbestos Textile Workers: Cohort Mortality and Case-Control Analyses. Am J Ind Med, 26:431-447.

De Stefani E, et al. (1997): Fatty Foods and the Risk of Lung Cancer: A Case-Control Study From Uruguay. Int J Cancer, 71:760-766.

Doll R, Peto R (1976): Mortality in relation to smoking: 20 years' observations on male British doctors. Br Med J, 2:1525-1536.

Doll R, Peto R (1978): Cigarette smoking and bronchial carcinoma: dose and time relationships among regular smokers and lifelong non-smokers. J Epidemiol Commun Health, 32:303-313.

Doll R (1978): An epidemiologic perspective of the biology of cancer. Cancer Res, 38:3573-3583.

Doll R, et al. (1980): Mortality in relation to smoking: 22 years' observations on female British doctors. Br Med J, 280:967-971.

Doll R, et al. (1994): Mortality in relation to smoking: 40 years' observations in male British doctors. Br Med J, 309:901-911.

Domino EF, et al. (1993): The relevance of nicotine content of common vegetables to the identification of passive tobacco smokers. Med Sci Res 21:571-572.

Du Y, Cha Q, Chen X, Chen Y, Huang L, Feng Z, *et al.* (1996). An epidemiological study of risk factors for lung cancer in Guangzhou, China. Lung Cancer 14(Suppl 1):S9-S37.

Du Y, Cha Q, Chen Y, Lei Y, Xue S (1995). Exposure to environmental tobacco smoke and female lung cancer. Indoor Air 5(4):231-6.

Easterbrook P, et al. (1991): Publication bias in clinical research. Lancet, 337:867-872.

Edlin C, et al. (1984): Radon in Homes -- A Possible Cause of Lung Cancer. Scand J Work Environ Health, 10:25-34.

Enstrom JE, Kabat GC (2003). Environmental tobacco smoke and tobacco related mortality in a prospective study of Californians, 1960-98. Br Med J 326(7398):1057.

Emmons KM, et al. (1992): Exposure to environmental tobacco smoke in naturalistic settings. Am J Publ Health, 82:24-28

Emmons K, et al. (1995): Dietary intake and exposure to environmental tobacco smoke in a worksite population. Eu J Clin Nutr, 49:336-343.

Fontham ET, et al. (1991): Lung cancer in nonsmoking women: A multicenter case-control study. Cancer Epidemiol Biomarkers Prev, 1:35-43.

Fontham E, et al. (1992): Lung Cancer in Nonsmoking Women: Dietary Antioxidants. Cancer Epidemiol Biomarkers Prev, 1:249.

Fontham E, et al. (1994): Environmental Tobacco Smoke and Lung Cancer in Nonsmoking Women: A Multicenter Study. J Am Med Assn, 271:1752-1759.

Freund KM, et al. (1993): The health risks of smoking. The Framingham study: 34 years of follow-up. Ann Epidemiol, 3:417-424.

Gao Y, et al. (1987): Lung Cancer Among Chinese Women. Int J Cancer, 40:604-609.

Gaylor DW, et al. (1997): Health Risk assessment practices in the U.S. Food and Drug Administration. Regulatory Toxicology and Pharmacology 26:307-321, 1997.

Garfinkel L (1981): Time Trends in Lung Cancer Mortality Among Nonsmokers. J Nat Cancer Inst, 66:1061-1066.

Garfinkel L, et al. (1985): Involuntary Smoking and Lung Cancer: A Case-Control Study. J Nat Cancer Inst, 75:463-469.

Geng G, et al. (1988): On the Relationship Between Smoking and Female Lung Cancer. In: Smoking and Health 1987 (Aoki M, et al., Eds.):, pp. 483-86, Elsevier Science Publishers, Amsterdam.

Givens G, et al. (1997): Publication Bias in Meta-Analysis: A Bayesian Data-Augmentation Approach To Account For Issues Exemplified in the Passive Smoking Debate. Stat Sci, 12:221-250.

Goodman M, et al. (1988): The Effect of Dietary Cholesterol and Fat on the Risk of Lung Cancer in Hawaii. Am J Epidemiol, 128:1241-1255.

Gori GB (1976): Low risk cigarettes: a prescription. Science, 194:1243-1246.

Gori GB, Mantel N (1991): Mainstream and environmental tobacco smoke. Regul Toxicol Pharmacol, 14:88-105.

Gori GB (1998a): Epidemiology and public health: is a new paradigm needed or a new ethic? J Clin Epidemiol 51:637-641.

Gori GB (1998b): Reply to the preceding dissent. J Clin Epidemiol 51:647-649.

Gori GB, Luik JC (1999): Passive smoke: the EPA’s betrayal of science and policy. The Fraser Institute, Vancouver.

Gravelle JG, Zimmermann D (1994): Cigarette taxes to fund health care reform: An economic analysis. Congressional Research Services, The Library of Congress, Washington DC.

Greenwald AG (1975): Consequences of prejudices against the null hypothesis. Psychol. Bull, 82:1-20.

Guerin MR, et al. (1987): Measuring environmental emissions from tobacco combustion: sidestream cigarette smoke literature review. Atmos Environ, 21:291-297.

Gustavsson P, et al. (1990): Lung Cancer and Exposure to Diesel Exhaust Among Bus Garage Workers. Scand J Work Environ Health, 16:334-54.

Hackshaw A, et al. (1997): The Accumulated Evidence on Lung Cancer and Environmental Tobacco Smoke. Br Med J, 315:980-988.

Haevner DL, et al. (1996): Determination of volatile organic compounds and respirable suspended particulate matter in New Jersey and Pennsylvania homes and workplaces. Environ Int 22:159-183.

Hatziandreu EJ, et al (1989): The reliability of self-reported cigarette consumption in the United States. Am J Publ Health, 79:1020-1023.

Hayes R, et al.(1989): Lung Cancer in Motor Exhaust-Related Occupations. Am J Ind Med, 16:685-695.

Hill AB (1965): The environment and disease: Association or Causation? Proc R Soc Med, 58:295-300.

Hinds M, et al. (1982): Tuberculosis and Lung Cancer Risk in Non-Smoking Women. Am Rev Respir Dis, 125:776-778.

Hirayama T (1981): Non-Smoking wives of Heavy Smokers a Higher Risk of Lung Cancer: A Study From Japan. Br Med J, 282:183-185.

Hirayama T (1984): Lung cancer in Japan: effects of nutrition on passive smoking. In: Lung cancer: causes and prevention. Mizell M, Correa P, Eds. pp.175-195. Verlag Chemie International Inc., New York.

Hoffmann D, Hecht SS (1989): Advances in tobacco carcinogenesis. In: Chemical carcinogenesis and mutagenesis. Cooper CS, Grover PL, eds., Springer-Verlag, New York

Hole D, et al. (1989): Passive Smoking and Cardiorespiratory Health in a General Population in the West of Scotland. Br Med J, 299:423-427.

Horwitz R, et al. (1988): An Ecogenetic Hypothesis for Lung Cancer in Women. Arch Intern Med, 148:2609-2612.

Humble C, et al. (1987): Marriage to a Smoker and Lung Cancer Risk. Am J Pub Health, 77:598-602.

IARC (1986): Tobacco smoking: Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol 38. International Agency for Research on Cancer, World Health Organization, Lyon, France.

ICSH (1998): Fourth report of the Independent Committee on Smoking and Health. Her Majesty's Stationery Office, London.

Inoue R, Hirayama T (1988): Passive Smoking and Lung Cancer in Women. In: Smoking and Health 1987. Aoki M, et al., Eds. pp.283-285. Elsevier Science Publishers, Amsterdam.

Jacobsson R, et al. (1997): Increased Risk of Lung Cancer Among Professional Drivers in Urban But Not Rural Areas of Sweden. Occup Environ Med, 54:189-193.

Jain M, et al. (1990): Dietary Factors and Risk of Lung Cancer: Results from a Case-Control Study, Toronto, 1981-85. Int J Cancer, 45:287-293.

Janerich D, et al. (1990): Lung Cancer and Exposure to Tobacco Smoke in the Household. N Engl J Med, 323:632-636.

Jarvis MJ, et al. (1987): Comparison of tests used to distinguish smokers from nonsmokers. Am J Publ Health, 77:1435-1438.

Jee SH, Ohrr H, Kim IS (1999). Effects of husbands' smoking on the incidence of lung cancer in Korean women. Intl J Epidemiol 28(5):824-8.

Jockel K-H, Pohlabeln H, Ahrens W, Krauss M (1998). Environmental tobacco smoke and lung cancer. Epidemiology 9(6):672-5.

Johnson KC, Hu J, Mao Y (2000). Passive and active smoking and breast cancer risk in Canada, 1994-97. The Canadian Cancer Registries Epidemiology Research Group. Cancer Causes Cont 11(3):211-21.

Johnson KC, Hu J, Mao Y (2001). Lifetime residential and workplace exposure to environmental tobacco smoke and lung cancer in never-smoking women, Canada 1994-97. Intl J Cancer 93(6):902-6.

Jenkins RA, et al. (1996): Exposure to environmental tobacco smoke in sixteen cities in the United States as determined by personal breathing zone air sampling. J Exp Anal Environ Epidemiol 6:473-502.

Jöckel K H (1991): Passive Smoking -- Evaluation of the Epidemiological Findings. Presentation to the Association of German Engineers, Mannheim Colloquium, VDI Reports 888.

Jöckel K H, Pohlabeln H, Ahrens W, Krauss M (1998): Environmental tobacco smoke and lung cancer. Epidemiology 9:672-675.

Jöckel K -H (1997): Environmental Tobacco Smoke and Lung Cancer in Germany. Position paper on Public Hearing of Experts on the Law on Protection of Non-Smokers, Health Policy Questions/Child and Youth Protection, Bonn, Germany.

Jöckel K-H (1998) Environmental tobacco smoke and lung cancer. Epidemiology 9:672-675.

Kabat G, Wynder E (1984): Lung Cancer in Nonsmokers. Cancer, 53:1214-1221.

Kabat G (1990): Epidemiological Studies of the Relationship Between Passive Smoking and Lung Cancer. In: Toxicology Forum: 1990 Annual Winter Meeting. pp.187-189. Washington, DC.

Kabat G (1994): Aspects of the Epidemiology of Lung Cancer in Smokers and Nonsmokers in the United States. In: Proceedings of the International Symposium on Lifestyle Factors and Human Lung Cancer, Guangzhou, China.

Kabat GC, et al. (1995): Relation between exposure to environmental tobacco smoke and lung cancer in lifetime nonsmokers. Am J Epidemiol, 142:141-148

Kalandidi A, et al. (1990): Passive Smoking and Diet in the Etiology of Lung Cancer Among Nonsmokers. Cancer Causes Control, 1:15-21.

Klawansky S, Fox MS (1984): A growth rate distribution model for the age dependence of human cancer incidence: A proposed role for promotion in cancer of the lung and breast. J Theor Biol, 111:531-587.

Klesges LM, et al. (1992): Discrepancies between self-reported smoking and carboxyhemoglobin - An analysis of the Second National Health and Nutrition Survey. Am J Publ Health, 82:1026-1029.

Kluger R (1996): Ashes to Ashes: American's hundred-year cigarette war, the public health, and the unabashed triumph of Philip Morris. A. Knopf, New York.

Ko YC, et al. (1997): Risk Factors for Primary Lung Cancer Among Non-Smoking Women in Taiwan. Int J Epidemiol, 26:24-31.

Koo L, et al. (1987a): Measurements of Passive Smoking and Estimates of Lung Cancer Risk Among Non-Smoking Chinese Females. Int J Cancer, 39:162-169.

Koo L, et al. (1997b): Dietary and Lifestyle Correlates of Passive Smoking in Hong Kong, Japan, Sweden and the USA. Soc Sci Med, 45:159-169.

Koo L, (1988): Dietary Habits and Lung Cancer Risk Among Chinese Females in Hong Kong Who Never Smoked. Nutr Cancer, 11:155-172.

Knekt P, et al.(1996): Elevated Lung Cancer Risk Among Persons With Depressed Mood. Am J Epidemiol, 144:1096-1103.

Kreuzer M, Krauss M, Kreienbrock L, Jockel KH, Wichmann HE (2000): Environmental tobacco smoke and lung cancer: a case-control study in Germany. Am J Epidemiol 151:241-250.

Kulessa C, et al. (1989): Psychosocial Personality Traits and Cigarette Smoking Among Bronchial Carcinoma Patients. Stress Med, 5:37-46.

Kvale G, et al. (1986): Occupational Exposure and Lung Cancer Risk. Int J Cancer, 37:185-193.

Lalonde M (1974): A new perspective on the health of Canadians. Health and Welfare Canada. Ottawa. Canada.

Lam W (1985): A Clinical and Epidemiological Study of Carcinoma of Lung in Hong Kong. M.D. Dissertation, Hong Kong University, Hong Kong.

Lam T, et al. (1987): Smoking, Passive Smoking and Histological Types in Lung Cancer in Hong Kong Chinese Women. Br J Cancer, 6:673-678.

Lange P, et al. (1990): Ventilatory Function and Chronic Mucus Hypersecretion as Predictors of Death from Lung Cancer. Am Rev Respir Dis, 141:613-617.

Last J (1994): New pathways in an age of ecological and ethical concerns. Int J Epidemiol, 23:1-4.

Lee PN, et al. (1986): Relationship of Passive Smoking to Risk of Lung Cancer and Other Smoking-Associated Diseases. Br J Cancer, 54:97-105.
Lee PN (1992): Environmental Tobacco Smoke and Mortality. Karger, Basle, Switzerland.

Lee PN (1993a): An estimate of adult mortality in the United States from passive smoking. Envir Intern, 19:91-100.

Lee PN (1993b): An Assessment of the Epidemiological Evidence Relating Lung Cancer Risk in Never Smokers to Environmental Tobacco Smoke Exposure. In: Environmental Tobacco Smoke (Kasuga H, ed.): pp.28-70. Springer-Verlag, New York.

Lee PN, Forey B (1995): Misclassification of Smoking Habits as Determined by Cotinine or by Repeated Self-Report -- A Summary From 42 Studies. J Smoking-Related Dis, 6:109-129.

Lee PN, Forey B (1996): Misclassification of Smoking Habits as a Source of Bias in the Study of Environmental Tobacco Smoke and Lung Cancer. Statistics Med, 15:581-605.

Lee PN (1998): Difficulties in assessing the relationship between passive smoking and lung cancer. Stat Met Med Res, 7:137-163.

Lee I, Paffenbarger R (1994): Physical Activity and Its Relation to Cancer Risk. Med Sci Sport Exerc, 26:831-837.

Lee C, Ko Y, Goggins W, Huang J, Huang M, Kao E, *et al.* (2000). Lifetime environmental exposure to tobacco smoke and primary lung cancer of non-smoking Taiwan women. Intl J Epidemiol 29(2):224-31.

Lees R, et al. (1987): A Case-Control Study of Lung Cancer Relative to Domestic Radon Exposure. Int J Epidemiol, 16:7-12.

Lei YX, et al. (1996): Some Lifestyle Factors in Human Lung Cancer: A Case-Control Study of 792 Lung Cancer Cases. Lung Cancer, 14(Suppl.1):S121-S136.

Le Marchand L, et al. (1989): Vegetable consumption and lung cancer risk: a population-based case-control study in Hawaii. JNCI, 81:1158-1164.

Le Marchand L, et al. (1991): Dietary patterns of female nonsmokers with and without exposure to environmental tobacco smoke. Cancer Cause Contr, 2:11-16.

LeVois ME, Layard MW (1994): Inconsistencies between workplace and spousal studies of environmental tobacco smoke and lung cancer. Regul Toxicol Pharmacol, 19:309-316.

LeVois M, Switzer P (1998): Differential Exposure Misclassification in Case-Control Studies of Environmental Tobacco Smoke and Lung Cancer. J Clin Epidemiol, 51:37-54.

Liu Q, et al. (1993): Indoor Air Pollution and Lung Cancer in Guangzhou, People's Republic of China. Am J Epidemiol, 137:145-154.

Liu Z, et al. (1991): Smoking and Other Risk Factors for Lung Cancer in Xuanwei, China. Int J Epidemiol, 20:25-31.

Luik JC (1996): Smokescreen: passive smoking and public policy. Institute of Public Affairs, Melbourne.

Luo RX, et al. (1996): Indoor Burning Coal Air Pollution and Lung Cancer: A Case-Control Study in Fuzhou, China. Lung Cancer, 14(Suppl.1):S113-119.

Mantel N, Haenszel W (1959): Statistical Aspects of the Analysis of Data From Retrospective Studies of Disease. J Nat Cancer Inst, 22:719-748.

Margetts BM, Jackson AA (1993): Interactions between people's diet and their smoking habits: the dietary and nutritional survey of British adults. Br Med J, 307:1381-1384.

Matanoski G, et al. (1995): Characteristics of Nonsmoking Women in NHANES I and NHANES II Epidemiologic Follow-Up Study With Exposure to Spouses Who Smoke. Am J Epidemiol, 142:149-157.

Mercer RR, Crapo JD (1993): Three-dimensional analysis of lung structure and its application to pulmonary dosimetry models. In: Toxicology of the lung, Gardner DE, et al., eds. Raven Press, New York.

Mettlin C, (1989): Milk Drinking, Other Beverage Habits, and Lung Cancer Risk. Int J Cancer, 43:608-612.

Miesner EA, et al. (1989): Particulate and nicotine sampling in public facilities and offices. J Air Pollut Control Assoc, 39:1577-1582.

Mumford J, et al. (1987): Lung Cancer and Indoor Air Pollution in Xuan Wei, China. Science, 235:217-220.

NAS, (1986): National Research Council, National Academy of Sciences. Environmental Tobacco Smoke: measuring exposures and assessing health effects. National Academy Press, Washington, DC

NAS (1992): Responsible Science: Ensuring the integrity of the research process, Volume I. National Academy of Sciences, National Academy Press, Washington DC.

Nelson PR, et al. (1997): Composition of environmental tobacco smoke (ETS) from international cigarettes and determination of ETS-RSP:particulate marker ratios. Environ Int 23:47-52.

Nishino Y, Tsubono Y, Tsuji I, Komatsu S, Kanemura S, Nakatsuka H, *et al.* (2001). Passive smoking at home and cancer risk: a population-based prospective study in Japanese nonsmoking women. Cancer Causes Cont 12(9):797-802.

Nyberg F, Agrenius V, Svartengren K, Svensson C, Pershagen G (1998a). Environmental tobacco smoke and lung cancer in nonsmokers: does time since exposure play a role? Epidemiology 9(3):301-8.

Nyberg F, Agudo A, Boffetta P, Fortes C, Gonzalez CA, Pershagen G (1998b). A European validation study of smoking and environmental tobacco smoke exposure in nonsmoking lung cancer cases and controls. Cancer Causes Cont 9(2):173-82.

NTP (1998): Report on Carcinogens Subcommittee. Board of Scientific Counselors. National Toxicology Program. Session of December 2-3, 1998. National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina.

Nyberg F, et al. (1997): Environmental Tobacco Smoke and Lung Cancer -- Does Time Since Exposure Play a Role? Epidemiology, 8:S38.

Ogden MW, et al. (1997): National incidence of smoking and misclassification among U.S. married female population. J Clin Epidemiol 50:253-263.

Ohlin P, et al. (1976): Carbon monoxide blood levels and reported cessation of smoking. Psychopharmacology (Berlin), 49:263-265.

Oksa P, et al. (1997): Cancer Incidence and mortality among Finnish asbestos sprayers and in asbestosis and silicosis patients. Am J Ind Med, 31:693-698.

Oldaker GB, et al. (1990): Results from surveys of environmental tobacco smoke in offices and restaurants. In: Indoor air quality. Kasuga H, ed., pp.99-104. Springer Verlag, Berlin.

Ooi W, et al. (1986): Increased Familial Risk for Lung Cancer. J Nat Cancer Inst, 76:217-222.

Osteen WL (1998): Order and Judgment in: Flue-cured Tobacco Cooperative Stabilization Corporation et al. V. United States Environmental Protection Agency and Carol Browner, Administrator, Environmental Protection Agency. U.S. District Court, Middle District of North Carolina, Winston-Salem Division. July, 17.

Papineau D (Ed.).The philosophy of science. Oxford University Press, Oxford. 1996.

Pastorino U, et al. (1987): Vitamin A and female lung cancer:A case-control study on plasma and diet. Nutr Cancer, 10:171-179.

Perbellini L, et al. (1988): Environmental and occupational exposure to benzene by analysis of breath and blood. Br J Ind Med, 45:345-352.

Perez-Stable E, et al. (1992): Misclassification of smoking status by self-reported cigarette consumption. Am Rev Respir Dis, 145:53-57.

Pershagen G, et al. (1987): Passive Smoking and Lung Cancer in Swedish Women. Am J Epidemiol, 125:17-24.

Phillips K, et al (1994): Assessment of personal exposures to environmental tobacco smoke in British nonsmokers. Environ Int 20:693-712.

Phillips K, et al. (1996): Assessment of air quality in Stockholm by personal monitoring of nonsmokers for respirable suspended particles and environmental tobacco smoke. Scand J Work Environ Health 22(1):1-24.

Phillips K, et al (1997a): Assessment of air quality in Barcelona by personal monitoring of nonsmokers for respirable suspended particles and environmental tobacco smoke. Environ Int 23:173-196.

Phillips K, et al (1997b): Assessment of air quality in Turin by personal monitoring of nonsmokers for respirable suspended particles and environmental tobacco smoke. Environ Int 23:851-871.

Phillips K, et al. (1998): Assessment of environmental tobacco smoke and respirable suspended particle exposures for nonsmokers in Prague using personal monitoring. Int Arch Occup Environ Health 71:379-390.

Pollack E. et al. (1984): Prospective Study of Alcohol Consumption and Cancer. N Engl J Med, 310:617-621.

Potter J, et al. (1992): Alcohol, Beer and Lung Cancer in Post-menopausal Women: The Iowa Women Health Study. Ann Epidemiol, 2:587-595.

Proctor CJ, et al. (1989):Measurement of environmental tobacco smoke in an air-conditioned office building. In: Present and future of indoor air quality. Bieva CJ, et al., eds., pp.169-172. Elsevier, Amsterdam.

Proctor CJ (1990): Measurement of ETS on smoking allowed and smoking prohibited public buses. In: Indoor air quality and ventilation. Luna F, Reynolds GL, eds., pp.427-436. Selper, London.

Rachtan J (2002). Smoking, passive smoking and lung cancer cell types among women in Poland. Lung Cancer 35(2):129-36.

Radon Research Act (1986): Radon Gas and Indoor Air Quality Research Act of 1986. Pub. L. No. 99-499, 100 Stat.1758-60. Codified 1994 at U.S.C. 5-7401 note.

Raffin E, et al. (1993): Incidence of Lung Cancer by Histological Type Among Asbestos Cement Workers in Denmark. Br J Ind Med, 50:85-89.

Rapiti E, Jindal SK, Gupta D, Boffetta P (1999): Passive smoking and lung cancer in Chandigarh, India . Lung Cancer (Ireland) 23:183-189.

Reilly WE (1991): Memorandum from William E. Reilly, Administrator, US Environmental Protection Agency, to Louis W. Sullivan, Secretary, US Department of Health and Human Services, July 2, 1991. US Environmental Protection Agency. Washington DC.

Redhead C, Rowberg R (1995): Environmental Tobacco Smoke and Lung Cancer Risk, US Congressional Research Service, US Library of Congress, Washington, DC.

Rodgman A (1992): Environmental tobacco smoke. Regul Toxicol Pharmacol, 16:223-224.

Rosenberg WG (1991): Assistant EPA Administrator for Air and Radiation. Letter to Erich Bretthauer, Assistant EPA Administrator for Research and Development, October 7, 1991. Letter to Donald G. Barnes, Director, EPA Science Advisory Board, June 28, 1991.

Rosenberg L (1994): Induced abortion and breast cancer: More scientific data are needed. J Nat Cancer Inst, 86:1569-1570.

Rothman KJ (1982): Causation and causal inference. In: Cancer epidemiology and prevention. Schottenfeld D, Fraumeni JF, eds. pp. 15-22. WB Saunders Co., Philadelphia.

Rothman KJ (1986): Modern Epidemiology. Little, Brown & Co, Boston.

Rylander R, (1996): Lung Cancer, Smoking, and Diet Among Swedish Men. Lung Cancer 14(Supp.1):S75-83.

Sakurai R, et al. (1989): Prognosis of Female Patients With Pulmonary Tuberculosis. Japan J Med, 28:471-477.

Samet J, et al. (1986): Personal and Family History of Respiratory Disease and Lung Cancer Risk. Am Rev Respir Dis, 134:466-470.

Samet JM (1992): Environmental tobacco smoke. In: Environmental toxicants. Lippmann M, ed. Van Nostrand & Reinhold, New York.

Sankaranarayanan R, et al. (1994): A Case-Control Study of Diet and Lung Cancer in Kerala, South India. Int J Med, 58:644-649.

Saracci R (1995): Ethical Issues in Assessing Scientific Evidence. Medicina del Lavoro, 86:174-177.

Schwartz A, et al. (1996): Familial risk of lung cancer among nonsmokers and their Relatives. Am J Epidemiol, 144:554-562.

SCOTH (1998): Report of the Scientific Committee on Tobacco and Health. Department of Health. Department of Health and Social Services, Northern Ireland. The Scottish Office Department of Health. Welsh Office. The Stationery Office. London.

Severson R, et al. (1989): A Prospective Analysis of Physical Activity and Cancer. Am J Epidemiol, 130:522-529.

Shapiro S (1997): Is Meta-Analysis a Valid Approach to the Evaluation of Small Effects in Observational Studies? J Clin Epidemiol, 50:223-229.

Schaffner KF (1991): Causing harm: Epidemiological and physiological concepts of causation. In: Acceptable evidence: Science and values in risk management. Mayo DG, Hollander RD, eds. Oxford University Press, New York.

Shapiro S (1998): Is meta-analysis a valid approach to the evaluation of small effects? In: Epidemiological practices in research on small effects. Hoffmann H, Szklo M, Thamm M, eds. Springer Verlag, Berlin.

Sheldon LS, et al. (1989): An investigation of infiltration and indoor air quality. Final report to the New York State Energy Research and Development Authority. Albany, New York.

Shen XB, et al. (1996): Relationship of Passive Smoking and Pulmonary Adenocarcinoma in Non-Smoking Women -- A Case Control Study in Nanjing, P.R. China. Epidemiology, 7:S20.

Shimizu H, et al. (1998): A Case Control Study of Lung Cancer in Nonsmoking Women. Tohoku J Exper Med, 154:389-397.

Shy C, (1984): Air Pollution and Lung Cancer. In: Lung Cancer: Causes and Prevention (Mizell M, Correa P, eds.), pp. 65-72. Verlag Chemie International, Berlin.

Sidney S, et al. (1989): Dietary intake of carotene in nonsmokers with and without passive smoking at home. Am J Epidemiol, 129:1305-1309.

Sillett RW, et al. (1978): Deception among smokers. Br Med J, 2:1185-1186.

Sobue T, et al. (1990): Association of Indoor Air Pollution and Passive Smoking With Lung Cancer in Osaka, Japan. Japan J Cancer Clin, 36:329-333.

Spengler JD, et al. (1981): Long-term measurement of respirable sulphates and particles inside and outside homes. Atmos Environ, 15:23-30.

Spengler JD, et al. (1985): Personal exposure to respirable particulates and implications for air pollution epidemiology. Environ Sci Technol, 19:700-707.

Steenland K (1992): Passive smoking and the risk of heart disease. JAMA, 267:94-99.

Sterling TD, Sterling EM (1983): Investigations on the effect of regulating smoking on levels of indoor pollution and on the perception of health and comfort of office workers. Eu J Respir Dis, 65:(Supp.133):17-32.

Sterling EM, et al. (1996): Assessment of nonsmoker exposure to environmental tobacco smoke using personal-exposure and fixed-location monitoring. Indoor Build Environ 5:112-125.

Stockwell HG, et al. (1992): Environmental tobacco smoke and lung cancer risk in nonsmoking women. JNCI, 84:1417-1422.

Stookey GK, et al. (1987): Evaluation of biochemical validation measures in determination of smoking status. J Dent Res, 66:1597-1601.

Stolwijk JAJ (1993): Statement: Transcript of the July 15, 1993 ETS session, Toxicology Forum, Aspen, Colorado. Toxicology Forum, Washington DC.

Sullum J (1998): For your own good, The Free Press, New York.

Sun X, et al. (1996): Environmental Tobacco Smoke (ETS): and Lung Cancer Among Nonsmoking Women in Harbin, China. Lung Cancer, 14(Suppl.1):S237.

Svensson C, et al. (1989): Smoking and Passive Smoking in Relation to Lung Cancer in Women. Acta Oncologica, 28:623-629.

Teel RW, Castonguay A (1992): Antimutagenic effects of polyphenolic compounds. Cancer Lett, 66:107-113.

Teich AH, Frankel MS (1992): Good science and responsible scientists. Meeting the challenge of fraud and misconduct in science. American Association for the Advancement of Science, Washington, DC.

Tenkanen L, et al. (1987): Smoking and Cardiac Symptoms as Predictors of Lung Cancer. J Chronic Dis, 40:1121-1128.

Thornton A, et al. (1994): Differences between smokers, exsmokers, passive smokers, and nonsmokers. J Clin Epidemiol, 47:1143-1162.

Thune I, Lund E (1997): The Influence of Physical Activity on Lung Cancer Risk: A Prospective Study of 81,516 Men and Women. Int J Cancer, 70:57-62.

Trichopoulos D, et al. (1981): Lung Cancer and Passive Smoking. Int J Cancer, 27:1-4.

Trichopoulos D, et al. (1983): Lung Cancer and Passive Smoking: Conclusion of Greek Study. Lancet, 2:677-678.

Tsuda T, et al. (1995): Ingested Arsenic and Internal Cancer: A Historical Cohort Followed for 33 Years. Am J Epidemiol, 141:198-209.

van Loon A, et al. (1997): Socioeconomic Status and Lung Cancer Incidence in Men in the Netherlands: Is There a Role for Occupational Exposure? J Epidemiol Commun. Health, 51:24-29.

USEPA (1986): U.S. Environmental Protection Agency. Guidelines for carcinogen risk assessment. Fed Reg 51:185:33993-34003; September 24

USEPA (1989): Indoor air facts No.5. Environmental tobacco smoke. US Environmental Protection Agency. Office of Air and Radiation. ANR-445. June 1989. Washington DC.

USEPA (1990a): United States Environmental Protection Agency. Health Effects of Passive Smoking: Assessment of lung cancer in adults and respiratory disorders in children. Office of Research and Development, Washington DC.

USEPA (1990b): U.S. Environmental Protection Agency. Technical Support Document for the 1990 Citizens Guide to Radon. USEPA. Office of Radiation Programs, Radon Division. August 16, 1990, Washington, DC.

USEPA (1992a): U.S. Environmental Protection Agency. Science Advisory Board, Environmental Tobacco Smoke Review Panel. July 21-22, 1992. Crystal City Holiday Inn, Arlington, VA 22202. Transcript from: Barrera Associates Inc., 733 15th Street NW, Suite 1120, Washington DC.

USEPA (1992b): United States Environmental Protection Agency. Respiratory health effects of passive smoking: Lung Cancer and other Disorders. May 1992. Office of Research and Development, Washington DC.

USEPA (1992c): United States Environmental Protection Agency. Respiratory health effects of passive smoking. Lung cancer and other disorders. December 1992. Office of Research and Development, Washington, DC.

USEPA (1992d): United States Environmental Protection Agency. Reviews of the EPA-ETS report by the EPA Environmental Assessment Office, Cincinnati, OH: Memorandum of April 27, 1990 from Chris DeRosa to William Farland. Memorandum of March 24, 1992 from Terry Harvey to Linda Bailey-Becht. US Environmental Protection Agency, Washington DC.

USEPA (1992e): United States Environmental Protection Agency. Safeguarding the future: credible science, credible decisions. The report of an expert panel on the role of science at EPA. EPA/600/9-91/050. March 1992. Office of the Administrator. Washington, DC.

USEPA (1994): Setting the record straight: Second hand smoke is a preventable health risk. EPA Publication 402-F-94-005. Environmental Protection Agency, Washington,DC

USEPA (1996): Proposed guidelines for carcinogen risk assessment; Notice. Fed Reg 61, No.79:17960-18011

USOSHA (1994): US Occupational Safety and Health Administration. Indoor air quality; Proposed Rule. Fed Reg 59(65):, April 5, 1994:15969-16039.

USSG (1964): Smoking and health. Report of the advisory committee to the Surgeon General of the Public Health Service. U.S. Department of Health, Education, and Welfare. Public Health Service -Publication No.1103., Washington, DC

USSG (1972): The health consequences of smoking. A report of the Surgeon General: 1972. DHEW Publication No. HSM 72-7516. Department of Health Education and Welfare, Washington, DC.

USSG (1975): The health consequences of smoking 1975.DHEW Publication No. CDC 76-8704. US Department of Health Education and Welfare. Public Health Service. Center for Disease Control. Atlanta, GA.

USSG (1979): Smoking and health: A report of the Surgeon General. DHEW publication No. (PHS):79-50066. Department of Health Education and Welfare, Washington DC.

USSG (1982): The health consequences of smoking: Cancer. A report of the Surgeon General. US Department of Health and Human Services. Office on Smoking and Health, Rockville, MD.

USSG (1984): The health consequences of smoking: Chronic obstructive lung diseases. A report of the Surgeon General. US Department of Health and Human Services, Washington DC.

USSG (1986): The health consequences of involuntary smoking, a report of the Surgeon General. US Public Health Service, Rockville MD.

USSG (1989): Reducing the health consequences of smoking: 25 years of progress. A report of the Surgeon General. DHHS publication No. CDC 89-8411. US Department of Health and Human Services. Public Health Service. Center for Disease Control. Atlanta GA.

USSG (1993): The health consequences of smoking: Cardiovascular diseases. A report of the Surgeon general. US Department of Health and Human Services, Washington DC.

Van Loy M, et al., (1997): Dynamic behavior of semivolatile organic compounds in indoor air. Nicotine in a stainless steel chamber. Environ Sci Technol 31:2554-2561.

Van Duuren BL (1980): Carcinogens, cocarcinogens, and tumor inhibitors in cigarette smoke condensate. In: A safe cigarette? Gori GB, Bock FG, eds. pp. 105-112. Cold Spring Harbor Laboratory, Cold Spring Harbor, NY.

Wagenknecht LE, et al. (1992): Misclassification of smoking status in the CARDIA study - A comparison of self-report with serum cotinine levels. Am J Publ Health, 82:33-36.

Wald NJ, et al. (1981): Serum cotinine levels in pipe smokers: Evidence against nicotine as a cause of coronary heart disease. Lancet 10:775-777.

Wang FL, et al. (1994): Childhood and Adolescent Passive Smoking and the Risk of Female Lung Cancer. Int J Epidemiol, 23:223-230.

Wang FL, et al. (1996): A Case-Control Study of Childhood and Adolescent Exposure to Environmental Tobacco Smoke (ETS): and the Risk of Female Lung Cancer. Lung Cancer, 14(Suppl.1):S238.

Wang SY, et al. (1996): A Comparative Study of the Risk Factors for Lung Cancer in Guangdong, China. Lung Cancer, 14(Suppl.1):S99-S105.

Wang SY, Hu YL, Wu YL, Li X, Chi GB, Chen Y, *et al.* (1996a). A comparative study of the risk factors for lung cancer in Guangdong, China. Lung Cancer 14 Suppl 1:S99-105.

Wang TJ, et al. (1996): Lung Cancer in Nonsmoking Chinese Women: A Case-Control Study. Lung Cancer, 14(Suppl.1):S93-S98.

Wang TJ, Zhou BS (1997): Meta-Analysis of the Potential Relationship Between Exposure to Environmental Tobacco Smoke and Lung Cancer in Nonsmoking Chinese Women. Lung Cancer, 16:145-15O.

Wang L, Lubin JH, Zhang SR, Metayer C, Xia Y, Brenner A, *et al.* (2000). Lung cancer and environmental tobacco smoke in a non-industrial area of China. Intl J Cancer 88(1):139-45.

Weed DL, Kramer BS (1997): Breast cancer studies aren't "political". The Wall Street Journal March 26:A19

Wu A, et al. (1985): Smoking and Other Risk Factors for Lung Cancer in Women. J Nat Cancer Inst, 74:747-751.

Wu A, et al. (1988): Personal and Family History of Lung Disease as Risk Factors for Adenocarcitoma of the Lung. Cancer Res, 48:7279-7284.

Wu-Williams A, et al. (1990): Lung Cancer Among Women in NorthEast China. Br Med J, 62:982-987.

Wynder EL (1987): Workshop on guidelines to the epidemiology of weak associations. Prev Med, 16:139-141.

Wynder EL, et al. (1987): Association of Dietary Fat and Lung Cancer. J Nat Cancer Inst, 79:631-637.

Wynder EL (1990): Epidemiological Issues in Weak Associations. Int J Epidemiol, 19(Suppl.1):S5-7.

Wu A, et al. (1985): Smoking and Other Risk Factors for Lung Cancer in Women. J Nat Cancer Inst. 74:747-751.

Wu A, et al. (1988): Personal and Family History of Lung Disease as Risk Factors for Adenocarcinoma of the Lung. Cancer Res, 48:7279-84.

Xu Z, et al. (1989): Smoking, Air Pollution, and High Rates of Lung Cancer in Shenyang, China. J Nat Cancer Inst, 6:1800-1806.

Ye Z, et al. (1990): The Environmental Factors of Lung Cancer in Family Women. Tianjin Chin J Clin Oncol, 17:195-198.

Yong L, et al. (1997): Intake of Vitamins E, C, and A and Risk of Lung Cancer: The NHANES-I Epidemiologic Followup Study, First National Health and Nutrition Examination Survey II. Am J Epidemiol, 146:321-343.

Yu S, Zhao N (1996): Combined Analysis of Case-Control Studies of Smoking and Lung Cancer in China. Lung Cancer, 14(Suppl.1):S161-170.

Yu ZF, et al. (1996): Environmental Factors and Lung Cancer. Lung Cancer, 14(Suppl.1):S240-241.

Zaridze D, et al. (1994): Relation Between Exposure to Environmental Tobacco Smoke and Lung Cancer in Non-Smoking Women in Moscow. Exper Oncol, 16:441-445.

Zhu H, Wang Z (1993): Study of Occupational Lung Cancer in Asbestos Factories in China. Br J Ind Med, 50:1039-1042.

Ziegler R, et al. (1986): Carotenoid Intake, Vegetables, and the Risk of Lung Cancer Among White Men in New Jersey. Am J Epidemiol, 123:1080-1093.

Zhong L, Goldberg MS, Gao YT, Jin F (1999): A case-control study of lung cancer and environmental tobacco smoke among nonsmoking women living in Shanghai, China. Cancer Causes and Control 10:607-616.

_____

Gori GB, Luik JC. Passive smoke: EPA's betrayal of science and policy. The Fraser Institute, Vancouver, 1999

_____

BMJ. 1995 Sep 9;311(7006):649-52. - **Incidence of cancer among Finnish airline cabin attendants, 1967-92. - Pukkala E, Auvinen A, Wahlberg G.** - Finnish Cancer Registry, Helsinki.

OBJECTIVE--To assess whether occupational exposure among commercial airline cabin attendants are associated with risk of cancer. DESIGN--Record linkage study. SETTING--Finland. SUBJECTS-1577 female and 187 male cabin attendants who had worked for the Finnish airline companies. MAIN OUTCOME MEASURE--Standardised incidence ratio; expected number of cases based on national cancer incidences. RESULTS--A significant excess of breast cancer (standardised incidence ratio 1.87 (95% confidence interval 1.15 to 2.23)) and bone cancer (15.10 (1.82 to 54.40)) was found among female workers. The risk of breast cancer was most prominent 15 years after recruitment. Risks of leukaemia (3.57 (0.43 to 12.9)) and skin melanoma (2.11 (0.43 to 6.15) were not significantly raised. Among men, one lymphoma and one Kaposi's sarcoma were found (expected number of cases 1.6). CONCLUSIONS--Although the lifestyle of cabin attendants is different from that of the reference population--for example, in terms of social status and parity--concentration of the excess risks to primary sites sensitive to radiation suggests that ionising radiation during flights may add to the cancer risk of all flight personnel. Otherwise the lifestyle of cabin attendants did not seem to affect their risks of cancer. Estimates of the effect of reproductive risk factors only partly explained the increased risk of breast cancer. If present estimates of health hazards due to radiation are also valid for cosmic radiation, then the radiation doses of cabin attendants seem too small to account entirely for the observed excess risk.

Cancer Causes Control. 2001 Feb;12(2):95-101. - **Risk of breast cancer in female flight attendants: a population-based study (Iceland).** - **Rafnsson V, Tulinius H, Jonasson JG, Hrafnkelsson J. -** Department of Preventive Medicine, University of Iceland, Reykjavik. vilraf@hi.is

OBJECTIVES: To study whether increased cancer risk, particularly of cancer types previously related to radiation, was found among cabin attendants, using employment time as a surrogate of exposure to cosmic radiation. METHODS: A cohort of 1690 cabin attendants, 158 men and 1532 women from the Icelandic Cabin Crew Association and two

airline companies in Iceland, was established. Cancer sites were ascertained between 1955 and 1997 by follow-up in a cancer registry. The personal identification number of each subject was used in record linkage to population-based registers containing vital and emigration status, reproductive factors and histologically verified cancer diagnosis. Standardized incidence rates (SIR) of different cancer sites in relation to employment time and year of hiring were calculated, as well as predictive values of breast cancer risk for evaluating possible confounding due to reproductive factors. RESULTS: The total number of person-years was 27,148. Among the women, 64 cancers were observed whereas 51.63 were expected (SIR 1.2, 95% CI 1.0-1.6), and significantly increased risk for malignant melanoma (SIR 3.0, 95% CI 1.2-6.2) was found. Significantly increased risks of overall cancers (SIR 1.3, 95% CI 1.0-1.8) and breast cancer (SIR 1.6, 95% CI 1.0-2.4) were observed among the female cabin attendants when 15 years lag time was applied. Those hired in 1971 or later had the heaviest exposure to cosmic radiation at a young age and had significantly increased risk of overall cancer (SIR 2.8, 95% CI 1.4-4.9) and breast cancer (SIR 4.1, 95% CI 1.7-8.5). Predictive values calculated on the basis of reproductive factors among the cabin attendants and the population, and risk of breast cancer were 1.0 for parous vs. nulliparous, 1.0 for number of children, and 1.1 for age at birth of first child. CONCLUSION: The increased risk of breast cancer and malignant melanoma among cabin attendants seems to be occupationally related. The part played by occupational exposures, i.e. cosmic radiation, disturbance of the circadian rhythm, and electromagnetic fields or combination of these factors in the etiology of breast cancer among the cabin crew, is still a puzzle as confounding due to parity appears to be ruled out. The relationship between the sunbathing habits of the cabin crew and the increased risk of malignant melanoma needs to be clarified. There is also an urgent need to elucidate the importance of these findings for today's aviation.

Int J Epidemiol. 2001 Aug;30(4):825-30. - **Cancer incidence among Norwegian airline cabin attendants.** Haldorsen T, Reitan JB, Tveten U. - The Cancer Registry of Norway, Oslo, Norway. tor.haldorsen@kreftregisteret.no

BACKGROUND: Cabin crews are exposed to cosmic radiation at work and this may increase their incidence of radiation-induced cancers. Former studies indicate an increased risk of breast cancer. METHODS: A retrospective cohort study was performed. The cohort was established from the files of the Civil Aviation Administration and included people with a valid licence as a cabin attendant between 1950 and 1994. The cohort was linked to the Cancer Registry of Norway. Observed number of cases was compared with expected, based on national rates. Breast cancer incidence was analysed, adjusting for individual fertility variables. RESULTS: A group of 3693 cabin attendants were followed over 72 804 person-years. Among the women, 38 cases of breast cancer were observed (standardized incidence ratio (SIR) = 1.1, 95% CI : 0.8-1.5). Among men excess risks were found for cancers in the upper respiratory and gastric tract (SIR = 6.0, 95% CI : 2.7-11.4) and cancer of the liver (two cases, SIR = 10.8, 95% CI : 1.3-39.2). For both sexes elevated risks were found for malignant melanoma and non-melanoma skin cancer; for men these were SIR = 2.9 (95% CI : 1.1-6.4) and SIR = 9.9 (95% CI : 4.5-18.8) respectively, while for women these were SIR = 1.7 (95% CI : 1.0-2.7) and SIR = 2.9 (95% CI : 1.0-6.9) respectively. For no cancer site was a significant decreased risk found. CONCLUSIONS: An increased risk of radiation-induced cancers was not observed. The excess risks of some other cancers are more probably explained by factors related to lifestyle.

LUNG CANCER RISKS - LITERATURE

Adami H, et al.: Risk of Cancer in Women Receiving Hormone Replacement Therapy. Int. J. Cancer 44:833-439 (1989).

Alavanja M: Saturated Fat Intake and Lung Cancer Risk Among Nonsmoking Women in Missouri. J. Nat. Cancer Inst., 85(23):1906-16 (1993).

Albanes D: Physical Activity and the Risk of Cancer in the NHANES I Population. Am. J. Pub. Health, 79:744-50 (1989).

Agudo A, et al.: Vegetable and Fruit Intake and the Risk of Lung Cancer in Women in Barcelona, Spain. Eur. J. Cancer, 33:1256-61 (1997).

Axelsson G, et al.: Dietary Factors and Lung Cancer Among Men in West Sweden. Int. J. Epidemiol., 25:32-39 (1996).

Brown S, et al.: The Association of Economic Status With the Occurrence of Lung Cancer. Cancer, 36:1903-11 (1975).

Byers T, et al.: Diet and Lung Cancer Risk: Findings from the Western New York Diet Study. Am. J. Epidemiol., 125:351-63 (1987).

Brownson R, et al.: Family History of Cancer and Risk of Lung Cancer in Lifetime Non-Smokers and Long-Term ExSmokers. Int. J. Epidemiol., 26(2):256-63 (1997).

Candelora E, et al.: Dietary Intake and Risk of Lung Cancer in Women Who Never Smoked. Nutr. Cancer, 17(3):26370 (1992).

Dement J, et al.: Follow-Up Study of Chrisotyle Asbestos Textile Workers: Cohort Mortality and Case-Control Analyses. Am. J. -Tnd. Med., 26:431-47 (1994).

Doll R, et al. (1994): Mortality in relation to smoking: 40 years' observations in male British doctors. Br Med J, 309:901-911.

De Stefani E, et al.: Fatty Foods and the Risk of Lung Cancer: A Case-Control Study From Uruguay. Int. J. Cancer, 71:760-66 (1997).

Edlin C, et al.: Radon in Homes-A Possible Cause of Lung Cancer. Scand. J. Work Environ. Health, 10:25-34 (1984).

Gao Y, et al.: Lung Cancer Among Chinese Women. Int. J. Cancer, 40:604-09 (1987).

Geng G, et al.: On the Relationship Between Smoking and Female Lung Cancer. In: Smoking and Health 1987 (Aoki M,et al., eds.), Elsevier Science Publishers, Amsterdam, Netherlands, pp. 483-86 (1988).

Goodman M, et al.: The Effect of Dietary Cholesterol and Fat on the Risk of Lung Cancer in Hawaii. Am. J. Epidemiol., 128:1241-55 (1988).

Gori GB, Luik JC : Passive smoke: The EPA's betrayal of science and policy. The Fraser Institute, Vancouver (1999).

Gustavsson P, et al.: Lung Cancer and Exposure to Diesel Exhaust Among Bus Garage Workers. Scand. J. Work Environ. Health, 16:334-54 (1990).

Hayes R, et al.: Lung Cancer in Motor Exhaust-Related Occupations. Am. J. Ind. Med., 16:685-95 (1989).

Hinds M, et al.: Tuberculosis and Lung Cancer Risk in Non-Smoking Women. Am. Rev. Respir. Dis., 125:776-78 (1982).

Horwitz R, et al.: An Ecogenetic Hypothesis for Lung Cancer in Women. Arch. Intern. Med., 148:2609-12 (1988).

Jain M, et al.: Dietary Factors and Risk of Lung Cancer: Results from a Case-Control Study, Toronto, 1981-85. Int. J. Cancer, 45:287-93 (1990).

Knekt P, et al.: Elevated Lung Cancer Risk Among Persons With Depressed Mood. Am. J. Epidemiol., 144:1096-103 (1996).

Ko Y-C, et al.: Risk Factors for Primary Lung Cancer Among Non-Smoking Women in Taiwan. Int. J. Epidemiol., 26:24-31 (1997).

Koo L: Dietary Habits and Lung Cancer Risk Among Chinese Females in Hong Kong Who Never Smoked. Nutr. Cancer, 11:155-72 (1988).

Kvale G, et al.: Occupational Exposure and Lung Cancer Risk. Int. J. Cancer, 37:185-93 (1986).

Kulessa C, et al.: Psychosocial Personality Traits and Cigarette Smoking Among Bronchial Carcinoma Patients. Stress Med., 5:37-46 (1989).

Lange P, et al.: Ventilatory Function and Chronic Mucus Hypersecretion as Predictors of Death from Lung Cancer. Am. Rev. Respir. Dis., 141:613-17 (1990).

Lee I, Paffenbarger R: Physical Activity and Its Relation to Cancer Risk. Med. Sci. Sport Exerc., 26:831-37 (1994).

Lees R, et al.: A Case-Control Study of Lung Cancer Relative to Domestic Radon Exposure. Int. J. Epidemiol., 16:7-12 (1987).

Le Marchand L, et al.: Vegetable Consumption and Lung Cancer Risk: A Population-Based Case-Control Study in Hawaii. J. Nat. Cancer Inst., 81:1158-64 (1989).

Mettlin C: Milk Drinking, Other Beverage Habits, and Lung Cancer Risk. Int. J. Cancer, 43:608-12 (1989).

Mumford J, et al.: Lung Cancer and Indoor Air Pollution in Xuan Wei, China. Science, 235:217-20 (1987).

Oksa P, et al.: Cancer Incidence and Mortality Among Finnish Asbestos Sprayers and in Asbestosis and Silicosis Patients. Am. J. Ind. Med., 31:693-98 (1997).

Ooi W, et al.: Increased Familial Risk for Lung Cancer. J. Nat. Cancer Inst., 76:217-22 (1986).

Pollack E, et al.: Prospective Study of Alcohol Consumption and Cancer. N. Engl. J. Med., 310:617-21 (1984).

Pastorino U, et al.: Vitamin A and Female Lung Cancer: A Case-Control Study on Plasma and Diet. Nutr. Cancer, 10: 17179 (1987).

Potter J, et al. , Alcohol, Beer and Lung Cancer in Postmenopausal Women: The Iowa Women Health Study. Ann. Epidemiol., 2:587-95 (1992).

Raffin E, et al.: Incidence of Lung Cancer by Histological Type Among Asbestos Cement Workers in Denmark. Br. J. Ind. Med., 50:85-89 (1993).

Rylander R: Lung Cancer, Smoking and Diet Among Swedish Men. Lung Cancer, 14(Supp. 1):S75-83 (1996).

Sakurai R, et al.: Prognosis of Female Patients With Pulmonary Tuberculosis. Japan J. Med., 28:471-77 (1989).

Samet J, et al.: Personal and Family History of Respiratory Disease and Lung Cancer Risk. Am. Rev. Respi-r. Dis., 134:466-70 (1986).

Sankaranarayanan R, et al.: A Case-Control Study of Diet and Lung Cancer in Kerala, South India. Int. J. Med., 58:644-49 (1994).

Severson R, et al.: A Prospective Analysis of Physical Activity and Cancer. Am. J. Epidemiol., 130:522-29 (1989).

Severson R, et al.: A Prospective Analysis of Physical Activity and Cancer. Am. J. Epidemiol., 130:522-29 (1989).

Shy C: Air Pollution and Lung Cancer. In: Lung Cancer: Causes and Prevention. (Mizell M, Correa P, eds.), Verlag Chemie International, Berlin/Heidelberg, Germany, pp. 65-72 (1984).

Sobue T, et al.: Association of Indoor Air Pollution and Passive Smoking With Lung Cancer in Osaka, Japan. Japan J. Cancer Clin., 36:329-33 (1990).

Tenkanen L, et al.: Smoking and Cardiac Symptoms as Predictors of Lung Cancer. J. Chronic Dis., 40:1121-28 (1987).

Thune I, Lund E: The Influence of Physical Activity on Lung Cancer Risk: A Prospective Study of 81,516 Men and Women. Int. J. Cancer, 70:57-62 (1997).

Tsuda T, et al.: Ingested Arsenic and Internal Cancer: A Historical Cohort Followed for 33 Years. Am. J. Epidemiol., 141:198-209 (1995).

USEPA : United States Environmental Protection Agency. Respiratory health effects of passive smoking. Lung cancer and other disorders. December 1992. Office of Research and Development, Washington, DC, (1992).

USSG (1984): The health consequences of smoking: Chronic obstructive lung diseases. A report of the Surgeon General. US Department of Health and Human Services, Washington DC.

Van Loon A, et al.: Socioeconomic Status and Lung Cancer Incidence in Men in the Netherlands: Is There a Role for Occupational Exposure? J. Epidemiol. Comm. Health, 51:24-29 (1997.

Wu A, et al.: Smoking and Other Risk Factors for Lung Cancer in Women. J. Nat. Cancer Inst., 74(4):747-51 (1985).

Wu A, et al.: Personal and Family History of Lung Disease as Risk Factors for Adenocarcinoma of the Lung. Cancer Res., 48:7279-84 (1988).

Wynder E, et al.: Association of Dietary Fat and Lung Cancer. J. Nat. Cancer Inst., 79:631-37 (1987).

Yong L, et al.: Intake of Vitamins E, C, and A and Risk of Lung Cancer: The NHANES-I Epidemiologic Followup Study, First National Health and Nutrition Examination Survey. Am. J. Epidemiol., 146:321-43 (1997).

Ziegler R, et al.: Carotenoid Intake, Vegetables, and the Risk of Lung Cancer Among White Men in New Jersey. Am. J. Epidemiol., 123:1080-93 (1986).

Zhu H, Wang Z: Study of Occupational Lung Cancer in Asbestos Factories in China. J. Ind. Med. 50:1039-42 (1993).